

5TH EDITION



THE **ATLAS** OF **EMERGENCY MEDICINE**

**Mc
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KEVIN J. KNOOP • LAWRENCE B. STACK
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The Atlas of **Emergency** FIFTH EDITION Medicine

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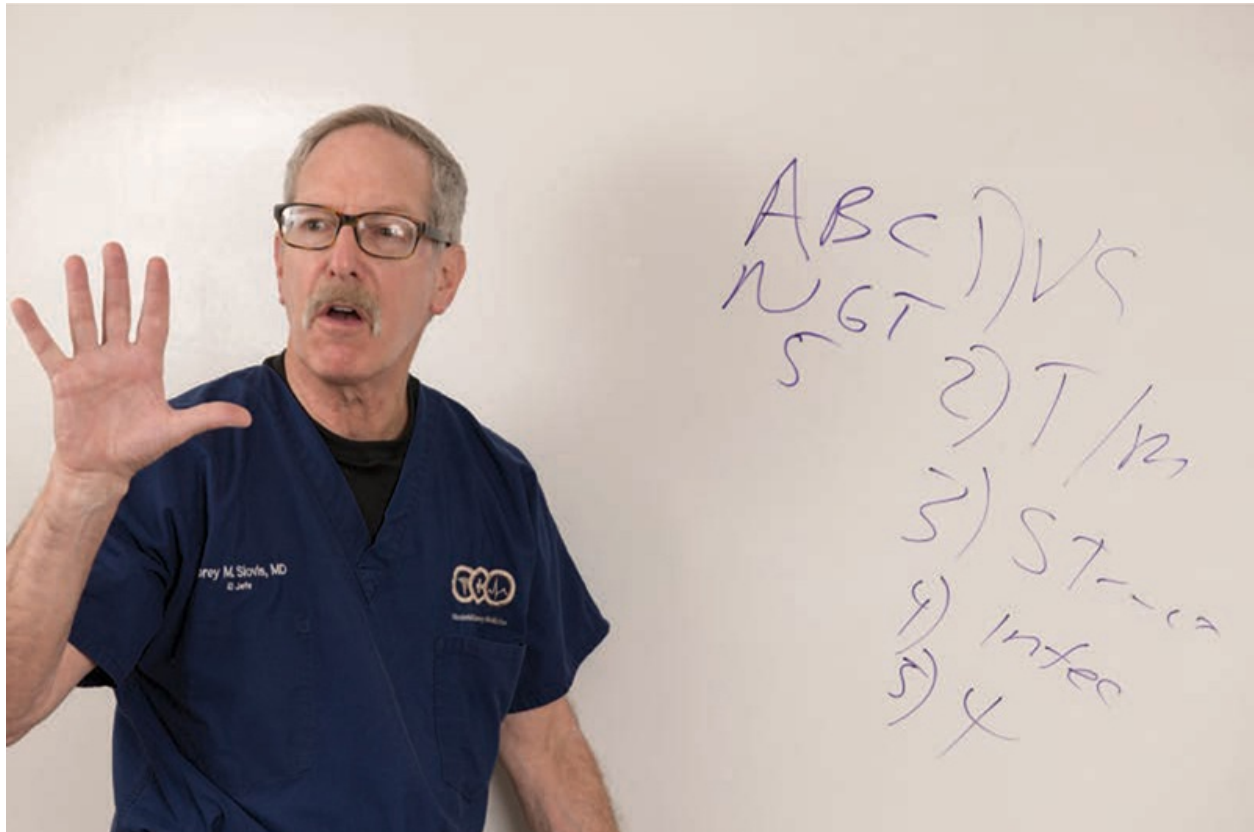
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DEDICATION



The authors dedicate this work to Dr. Corey Slovis, long-time mentor, teacher, and friend. For Emergency Medicine, he is iconic and his impact immeasurable. Dr. Slovis has led and taught thousands of emergency care providers and is one of the greatest educators we know. Through his teachings we have learned how high-quality education is essential for medical knowledge and clinical expertise. His “5 Causes, 5 Steps, 5 Reasons” for almost everything in medicine has resonated worldwide. As “El Jefe” retires, we offer our gratitude and deep appreciation for his service to our specialty and to Edition 5 of *The Atlas of Emergency Medicine*. Our patients have been and will be the ultimate beneficiaries of his magnificent career.

“Giddy Up”

KJK

LBS

ABS

RJT

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Chapter 23

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ACUTE ANTERIOR MYOCARDIAL INFARCTION
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LEFT MAIN LESION
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WOLFF-PARKINSON-WHITE SYNDROME

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“CEREBRAL” T WAVES
HYPERKALEMIA
HYPOKALEMIA
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TRICYCLIC ANTIDEPRESSANT EFFECT
LIMB LEAD REVERSAL
LOW VOLTAGE

Chapter 24

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SPUTUM
URINE
SYNOVIAL FLUID
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FOREWORD

Despite the expanding use of electronic media in current medical education, *The Atlas of Emergency Medicine* remains an essential textbook for any clinician's library. What makes *The Atlas of Emergency Medicine* so invaluable is its ability to comprehensively capture all of emergency medicine in a timeless way with high-quality figures, artwork, and videos.

Anyone who evaluates patients or who needs to see what a disease or clinical finding looks like will find the complete teacher in this atlas. Medical students, residents in training, educators, and clinicians will all greatly benefit from using this book. Although its primary audience are those of us in emergency medicine, the depth and breadth of the atlas will also be of significant value to residents and practicing physicians in other specialties, along with those in nursing, emergency medical services, and allied health. Anyone who wants to review the classic findings of a disease that might present to the emergency department or see subtle differences between two related entities will find them here.

This newest edition expands on its long-standing reputation as the definitive atlas in emergency care by adding chapters on mental health conditions and rheumatologic disorders and by expanding its presentation of microscopic findings and the analysis of body fluids, along with significantly increasing the number of high-quality videos, figures, and photographs. *The Atlas of Emergency Medicine* continues its long history of being the single most authoritative collection of clinical images, illustrations, ultrasounds, radiographs, and electrocardiograms encountered in the emergency department.

I view this book as indispensable in learning and practicing emergency medicine.

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PREFACE

“He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all.”

William Osler

We have a passion for improving patient care. Our journey with *The Atlas of Emergency Medicine* began with superb mentors who instilled in us a drive to become excellent clinician educators. We discovered imaging was a powerful tool to take the learner “to the bedside” in a fashion unlike any other didactic technique. In 1994, much by chance, collegial networking brought three, then later four, of us together to pursue an aggressive goal of producing the most comprehensive source of high-quality emergency care images available. While there were some initial detractors, our first four editions received widespread praise, have been translated into multiple foreign languages, and have been reproduced in alternative electronic media. We are humbled and honored to present our fifth iteration.

Emergency care is defined by time and space. The emergency department is by far the most diverse melting pot of acute conditions in the hospital. Diagnostic accuracy, risk stratification, and treatment rely heavily on visual clues. We desire to maximize this practitioner skill for the benefit of our patients. We also strongly believe the visual experience, while sometimes downplayed within the hectic and time-pressured environment of modern medicine, is critical to education. Images can teach faster and with greater impact than many pages of text or hours of lecture.

We continue our pursuit of these goals with a substantially updated, expanded, and improved fifth edition of *The Atlas of Emergency Medicine*. Nearly all our changes and additions come from reader suggestions and criticisms as well as superb guidance from our editors at McGraw Hill. All are received with sincere gratitude.

We have reduced text to essential information to allow for more images and increasing depth. After extensive review and critique, new and replacement

images and video have been added. While there have been radical changes in the way we access medical knowledge over the past two decades, an image in any form maintains a potent means to teach and learn. New chapters include Rheumatologic Conditions and Mental Health Conditions.

The audience for this text is all who provide emergency care, including clinicians, educators, residents, nurses, prehospital caregivers, medical technicians, and medical students. Many have also found it extremely useful as a review for written board examinations containing pictorial questions. Other healthcare workers, such as internists, family physicians, pediatricians, nurse practitioners, and physician assistants, will find the *Atlas* a useful guide in identifying and treating acute conditions, where visual clues significantly guide, improve, and expedite diagnosis as well as treatment.

We thank the many contributors, readers, and editors who have helped make this edition possible. Lastly, and most importantly, we express our deepest gratitude to our patients who were willing to be a “great case” in the *Atlas*, thus ultimately paving the way for improved emergency care.

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On a personal level, the editors would like to gratefully acknowledge:

Our patients, for being such willing teachers; Dr. Tomisaku Kawasaki, a special colleague, for his passion and generosity; MJ, Mimi and Stephen, for their unending love and inspiration.

KJK

The patients and their families who have allowed us to photograph their examination findings for the benefit of caring for others, I pray God's grace and blessings on you.

LBS

Father Steve Roberts and Jim O'Dowd. Thank you for being part of my journey.

ABS

Lauren, Kate, and Ben—the cornerstones of my life. My colleagues and friends who have been so incredibly supportive at Skyline. My lifelong friends—the incomparable CHP, especially our beloved brother Trent (“AXE”) who is sorely

missed. All the residents over the years who have allowed me to share in their journeys—it is an honor to have learned with you.

RJT

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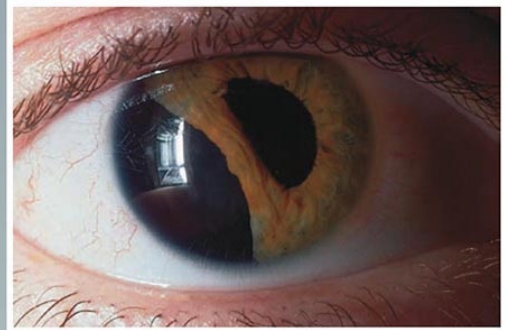
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PART 1

Regional Anatomy



Chapter 1

HEAD AND FACIAL TRAUMA

Christopher L. Stark



Burr Hole. A right temporal bone burr hole is being performed in the emergency department due to rapid decompensation from extra-axial bleeding and clinical signs of uncal herniation. (Photo contributor: Kevin J. Knoop, MS, MD.)

SCALP LACERATION

Clinical Summary

The scalp is the soft tissue that covers the cranial vault. Scalp lacerations are common in the emergency department (ED), and most are managed without complications with simple suturing or stapling of the soft tissues under local anesthetic. If admitted, it is usually due to severe injuries associated with the trauma that caused the laceration. Infrequently, bleeding from the scalp's copious blood supply requires resuscitation and transfusion from acute blood loss. Most delayed complications from scalp wounds occur secondary to infection from gross contamination, retained foreign bodies, bite wounds (including human), or retained hematoma. Infection is rare given the excellent blood supply, and antibiotics are generally not required.

The scalp consists of five layers:

S – Skin

C – Connective tissue

A – Aponeurosis and muscle (Galea)

L – Loose areolar tissue

P – Pericranium (periosteum)

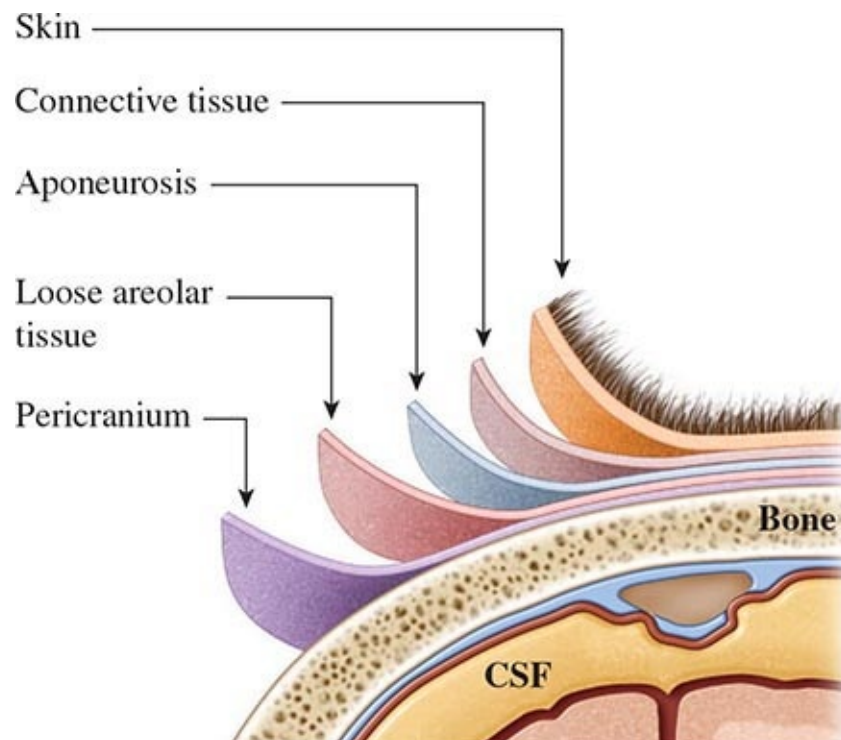


FIGURE 1.1 ■ Layers of the Scalp. Skin, connective tissue, aponeurosis, loose areolar tissue, and pericranium make up the layers of the scalp.

The 1st three layers are bound together, allowing them to move over the 4th. Thus, when lacerated, separation most commonly occurs at the loose areolar layer. Most bleeding comes from the superficial temporal artery and the occipital artery, in the aponeurosis and muscular layer.

Management and Disposition

Scalp laceration closure should not hinder trauma resuscitation unless bleeding is uncontrollable and contributing to patient instability. Primary closure is preferred in most cases, as delayed closure of the wound increases the risk of infection and scarring.

Closure options include surgical staples, hair apposition, and suturing. Staples are the preferred closure method in lacerations through the dermis in which bleeding is controlled as they are fast, inexpensive, and have few complications. They achieve similar cosmetic results compared to sutures. When choosing simple interrupted sutures (large wound, significant bleeding, short-haired patient), choose a blue proline suture or similar that stands out from the patient's hair color and leave long tails when cutting. Hair apposition is more time consuming than the other choices but is a good option for straight wounds under 10 cm in length.

Diagnostic imaging is not necessary for minor isolated scalp lacerations. Bony defect or suspicion for brain injury should prompt a head computed tomography (CT) scan. Concern for foreign body should prompt plain films if radiopaque or ultrasound if not radiopaque.

Pearls

1. Goals for primary scalp laceration closure include hemostasis, infection prevention, and cosmesis.
2. Hemostasis is typically achieved with pressure and wound closure, but may require lidocaine with epinephrine, cautery, or deep and specialty stitches.
3. Factors that contribute to undesired outcomes include delayed closure, crush injury, infection, foreign body, retained hematoma, bite wound, contaminated wound, diabetes, and peripheral vascular disease.

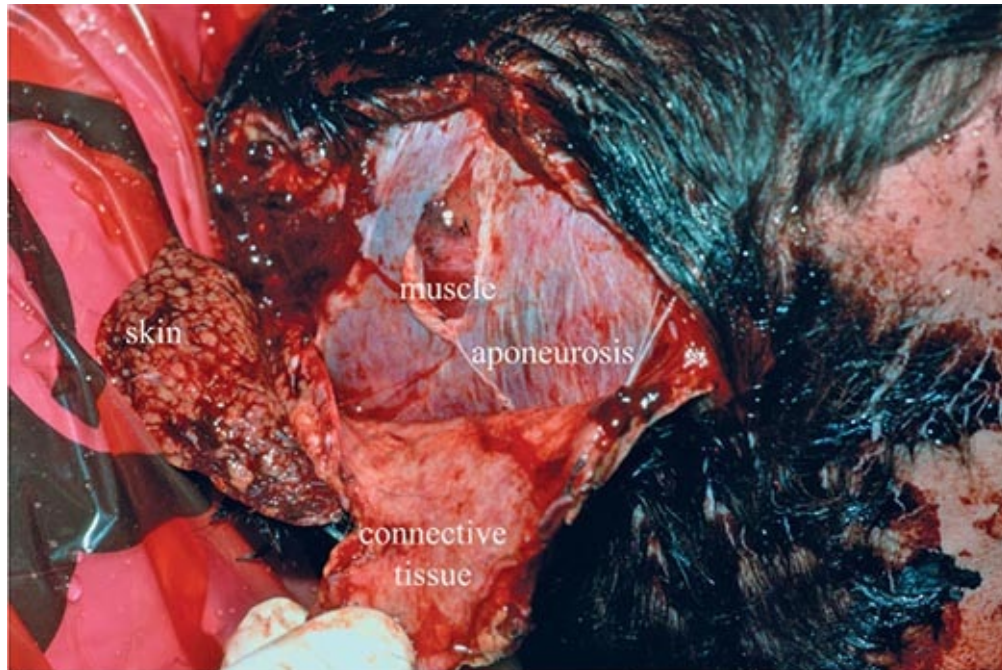


FIGURE 1.2 ■ Scalp Laceration. Laceration resulted from a motor vehicle crash. Skin, connective tissue, and aponeurosis/muscle layers are exposed. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.3 ■ Child Scalp Laceration. Laceration resulted from a motor vehicle crash. Periosteum is seen here as layer separation most commonly occurs at the loose areolar layer, which is wispy and falls away, leaving the periosteum (pericranium) exposed. (Photo contributor: Christopher L. Stark, DO.)

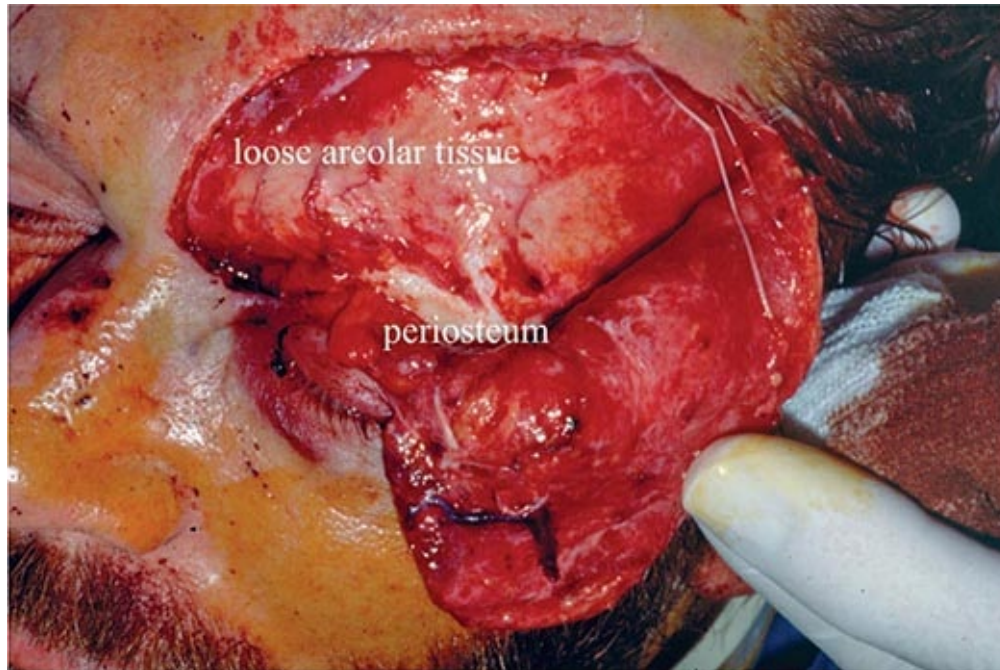


FIGURE 1.4 ■ Forehead Scalp Laceration. Laceration resulted from a motor vehicle crash. Loose areolar tissue is seen overlying the periosteum, which is clearly exposed over lateral superior orbital rim. (Photo contributor: Lawrence B. Stack, MD.)

DEPRESSED SKULL FRACTURE

Clinical Summary

Depressed skull fractures typically occur when a large force is applied over a small area. They are classified as open if the skin above them is lacerated. Abrasions, contusions, and hematomas may also be present over the fracture site. The patient's mental status is dependent upon the degree of underlying brain injury. Direct trauma can cause abrasions, contusions, hematomas, and lacerations without an underlying depressed skull fracture. Evidence of other associated injuries, such as basilar fracture, facial fractures, cervical spinal injuries, intracerebral hemorrhage, and nerve injury, may also be present.

Management and Disposition

Explore all scalp lacerations to exclude a depressed fracture by finding the base of the wound and, if skull is evident, running the tip of a closed pair of hemostats

across the skull surface to find a bone step-off. CT should be performed in all suspected depressed skull fractures to determine the extent of underlying brain injury. Depressed skull fractures require immediate neurosurgical consultation. Treat open fractures with antibiotics and tetanus prophylaxis as indicated. The decision to observe or operate immediately is made by the neurosurgeon. Children below 2 years of age with skull fractures can develop leptomenigeal cysts, which are extrusion of cerebrospinal fluid (CSF) or brain through dural defects. For this reason, children below age 2 with skull fractures require close follow-up or admission.

Pearls

1. Examine all scalp injuries including lacerations for evidence of fractures or depression. When fragments are depressed 5 mm below the inner table, penetration of the dura and injury to the cortex are more likely.
2. Children with depressed skull fractures are more likely to develop epilepsy.
3. Ping pong ball skull fractures can occur from a forceps delivery or from compression by mother's sacral promontory during delivery.
4. Patients with head injuries must be evaluated for cervical spine injuries.



FIGURE 1.5 ■ Depressed Skull Fracture. A scalp laceration overlying a depressed skull fracture. Scalp lacerations should undergo sterile exploration for skull fracture. (Photo contributor: David W. Munter, MD.)

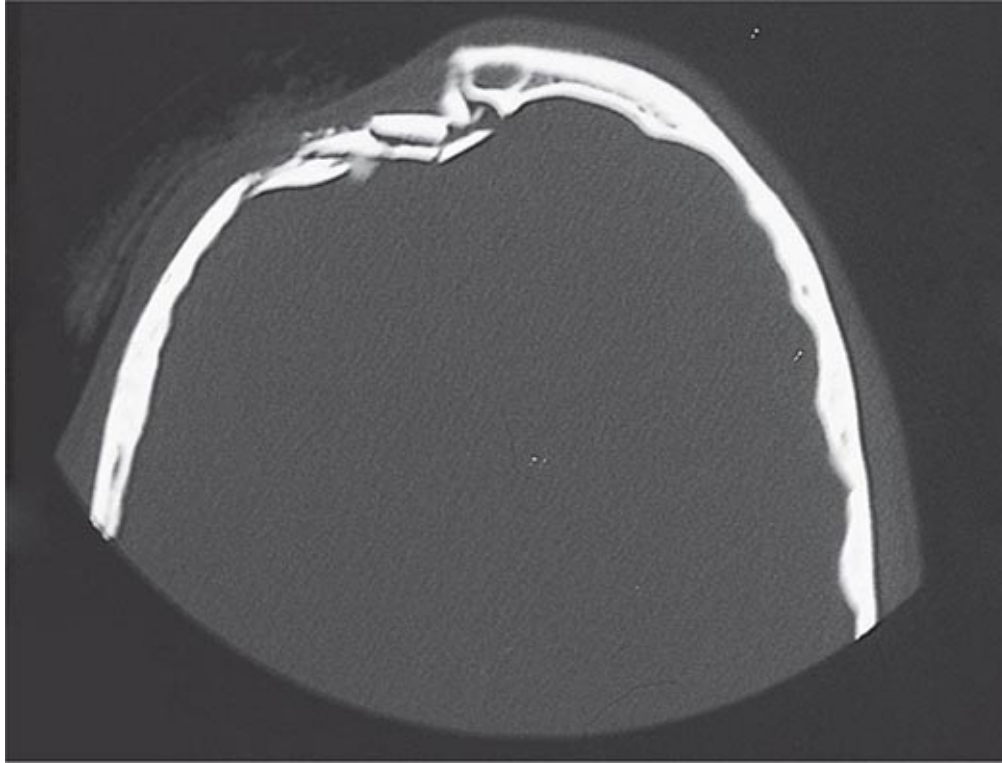


FIGURE 1.6 ■ Depressed Skull Fracture. CT demonstrating depressed skull fracture. (Photo contributor: David W. Munter, MD.)

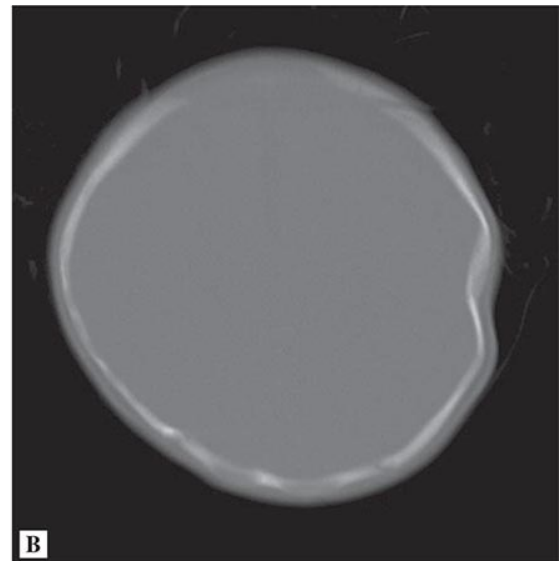


FIGURE 1.7 ■ Ping Pong Ball Skull Fracture. (A) Akin to the greenstick fracture, a ping pong ball fracture occurs when a newborn or infant's relatively soft skull is indented by the corner of a table or similar object without causing a frank break in the bone. (B) CT demonstrates the ping pong ball effect. (Photo contributor: Johnny Wei, MD.)

FRONTAL SINUS FRACTURE

Clinical Summary

Blunt trauma to the frontal bone may result in a depressed frontal sinus fracture. Often, there is an associated laceration. Isolated frontal fractures normally do not have the associated features of massive blunt facial trauma such as seen in LeFort II and III fractures. Careful nasal speculum examination may reveal blood or CSF leak high in the nasal cavity. Posterior table involvement can lead to mucopyocele or epidural empyema as late sequelae. Involvement of the posterior wall of the frontal sinus may occur and result in intracranial injury or dural tear. Frontal fractures may be part of a complex of facial fractures, as seen in frontonasoethmoid fractures, but generally more extensive facial trauma is required.



FIGURE 1.8 ■ Frontal Laceration. Any laceration over the frontal sinuses should be explored to rule out a fracture. This laceration was found to have an associated frontal fracture. (Photo contributor: David W. Munter, MD.)

Management and Disposition

Obtain head CT with bone windows on patients suspected of frontal sinus fractures. Fractures involving only the anterior table of the frontal sinus can be treated conservatively with referral to otolaryngology (ENT) or plastic surgery in 1 to 2 days. Fractures involving the posterior table require urgent neurosurgical consultation. Treat frontal sinus fractures with broad-spectrum antibiotics against both skin and sinus flora. ED management also includes control of epistaxis, application of ice packs, and analgesia.

Pearls

1. Explore every frontal laceration digitally before repair. Digital palpation is sensitive for identifying frontal fractures, although false positives from lacerations extending through the periosteum can occur.
2. Communication of irrigating solutions with the nose or mouth indicates a breach in the frontal sinus.
3. A head CT scan is needed to assess the integrity of the posterior sinus wall and intracranial injury.



FIGURE 1.9 ■ Frontal Sinus Fracture. Fracture defect seen at the base of a laceration over the frontal sinus. (Photo contributor: Jeffrey Kuhn, MD.)



FIGURE 1.10 ■ Frontal Sinus Fracture. Fracture of the outer table of the frontal sinus is seen under this forehead laceration. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.11 ■ Frontal Sinus Fracture. CT of the patient in [Fig. 1.8](#) demonstrating a fracture of the anterior table of the frontal sinus. (Photo contributor: David W. Munter, MD.)

BASILAR SKULL FRACTURE

Clinical Summary

The skull “base” comprises the frontal bone, occiput, occipital condyles, clivus, carotid canals, petrous portion of the temporal bones, and the posterior sphenoid wall. A basilar skull fracture is basically a linear fracture of the skull base. Trauma resulting in fractures to this area typically does not have localizing symptoms. Indirect signs of the injury may include visible evidence of bleeding from the fracture into the surrounding soft tissues of the base of the head, such as a Battle sign or “raccoon eyes.” Bleeding into other structures, blood in the middle ear causing hemotympanum, or blood in the sphenoid sinus seen as an air-fluid level on CT may also be seen. CSF leaks may also be evident and noted

as clear or pink rhinorrhea. If CSF is present, a dextrose stick test may be positive. The fluid can be placed on filter paper, and a “halo” or double ring may be seen. Bedsheets may reveal the halo sign.



FIGURE 1.12 ■ Battle Sign. Ecchymosis in the postauricular area develops when the fracture line communicates with the mastoid air cells, resulting in blood accumulating in the cutaneous tissue. This patient had sustained injuries several days prior to presentation. (Photo contributor: Frank Birinyi, MD.)

Management and Disposition

Identify underlying brain injury, which is best accomplished by head CT. CT is also the best diagnostic tool for identifying the fracture site, but fractures may not always be evident. Evidence of open communication, such as a CSF leak, requires neurosurgical consultation and admission. Otherwise, the decision for admission is based on the patient's clinical condition, other associated injuries, and evidence of underlying brain injury as seen on CT. The use of antibiotics in the presence of a CSF leak is controversial because of the possibility of selecting resistant organisms.

Pearls

1. Clinical manifestations of basilar skull fracture may take several hours to fully develop.
2. Have a low threshold for head CT in any patient with head trauma, loss of consciousness, change in mental status, severe headache, visual changes, or nausea or vomiting.
3. The use of filter paper or a dextrose stick test to determine if CSF is present in rhinorrhea is not 100% reliable.



FIGURE 1.13 ■ Battle Sign. A striking Battle sign is seen in this patient with head trauma. This finding may take hours to days to develop. (Photo contributor: David Effron, MD.)



FIGURE 1.14 ■ Raccoon Eyes. Acute periorbital ecchymosis seen in this patient with a basilar skull fracture. These findings may also be caused by facial fractures. (Photo contributor: Shannon Koh, MD.)



FIGURE 1.15 ■ Early Raccoon Eyes. Subtle periorbital ecchymosis manifests 1 hour after a blast injury. (Photo contributor: Kevin J. Knoop, MD, MS.)

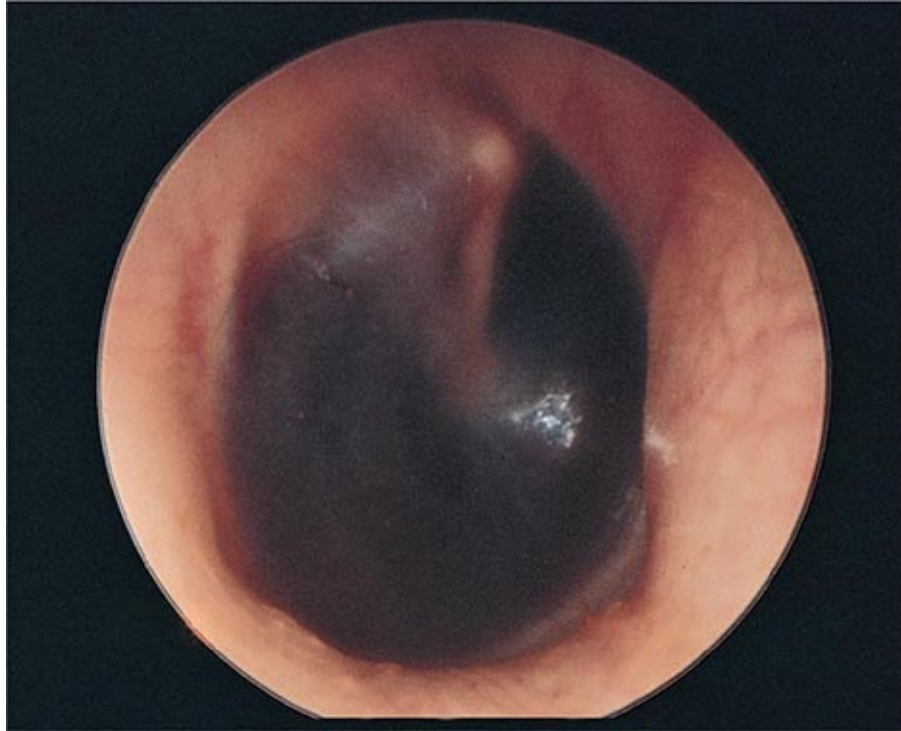


FIGURE 1.16 ■ Hemotympanum. Seen in a basilar skull fracture when the fracture line communicates with the auditory canal, resulting in bleeding into the middle ear. Blood can be seen behind the tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)



FIGURE 1.17 ■ Bloody Otorrhea. Tympanic membrane rupture in this pediatric trauma patient with basilar skull fracture. No canal or external ear bleeding was found. (Photo contributor: Christopher L. Stark, DO.)



FIGURE 1.18 ■ CT of Basilar Skull Fracture. CT bone window demonstrates a fracture of the posterior wall left sphenoid sinus (arrow) and an air-fluid level. (Photo contributor: Jared McKinney, MD.)



FIGURE 1.19 ■ Halo Sign—Cerebrospinal Fluid Leak. Otorrhea on bedsheet demonstrating a halo sign from a patient with severe head trauma. The distinctive double-ring sign, seen here, comprises blood (*inner ring*) and CSF (*outer ring*). The reliability of this test has been questioned. (Photo contributor: Suzanne Bryce, MD.)

HERNIATION SYNDROMES

Clinical Summary

Severe head injury can result in extra-axial hematoma, cerebral contusion, or diffuse cerebral edema, which, in turn, may cause one of five brain herniation syndromes: uncal, central transtentorial, cerebellotonsillar, subfalcine, and

external. Uncal herniation occurs when the uncus of the temporal lobe is displaced inferiorly through the medial edge of the tentorium. Compression of cranial nerve III can cause an ipsilateral dilated pupil. Typically, patients with uncal herniation are unconscious and require intubation. A contusion to the eye may also result in a dilated, nonresponsive pupil and arouse suspicion for uncal herniation, but typically these patients are alert.

Management and Disposition

Intubate unconscious head trauma patients with a unilateral dilated pupil and transfer them immediately to a facility capable of caring for traumatic brain injury. A head CT scan without contrast can identify a subdural or epidural hematoma, diffuse edema, or temporal lobe contusion. These conditions often cause midline shift of cerebral structures and compression of the quadrigeminal cistern. Unilateral effacement of the quadrigeminal cistern confirms uncal herniation. Initial management focuses on maintaining cerebral perfusion pressure and normal tissue oxygenation as hypotension and hypoxia significantly contribute to secondary brain injury. Mannitol, hypertonic saline, burr holes, and hyperventilation should be considered in ED patients with uncal herniation. Definitive care requires neurosurgical consultation.

Pearls

1. Uncal herniation is the most common of the five herniation syndromes.
2. If a patient has a unilateral dilated pupil after head and face trauma but is awake and talking, be suspicious for isolated traumatic anisocoria rather than herniation.
3. Extra-axial blood refers to bleeding outside the brain parenchyma and includes subdural, epidural, and subarachnoid bleeding. Extra-axial blood is most likely a neurosurgical rather than a neurological condition.
4. A temporal lobe contusion in an initially neurologically intact patient may continue to expand and cause uncal herniation.
5. Excessive hyperventilation ($\text{PaCO}_2 < 25 \text{ mm Hg}$) in patients with severe traumatic brain injury is associated with cerebral ischemia.
6. Effacement of the quadrigeminal cistern is the hallmark CT finding of uncal herniation.

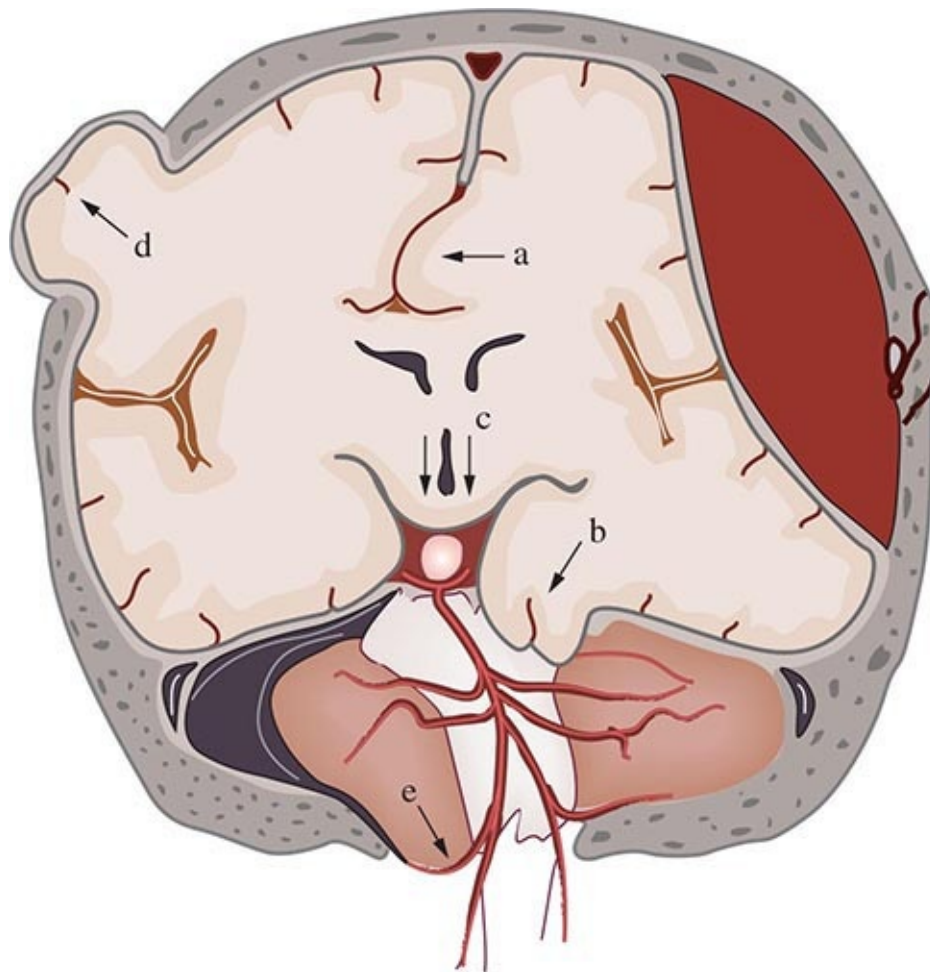


FIGURE 1.20 ■ Herniation Syndromes. a. Subfalcine; b. uncal; c. central transtentorial; d. external; e. cerebellotonsillar.

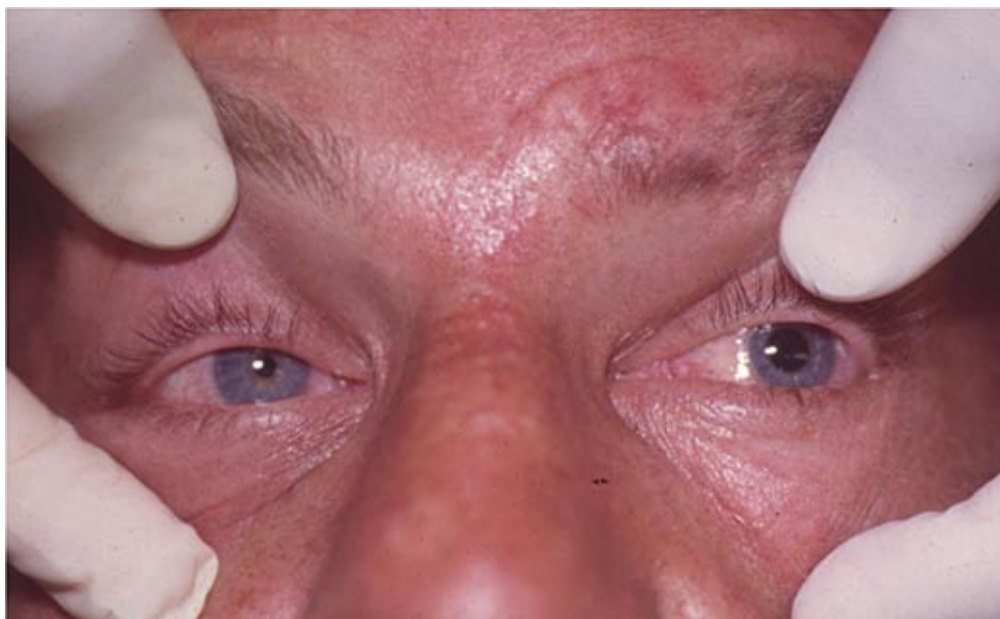


FIGURE 1.21 ■ Ipsilateral Dilated Pupil due to Uncal Herniation. CT revealed a right epidural hematoma and unilateral effacement of the quadrigeminal cistern. (Photo contributor: Lawrence B. Stack, MD.)

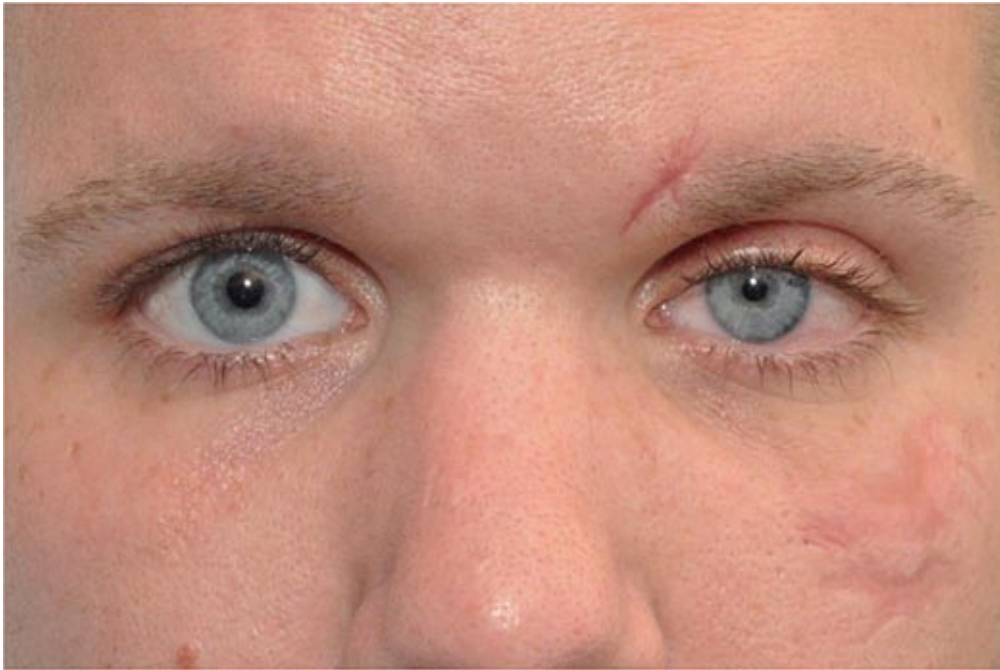


FIGURE 1.22 ■ Isolated Traumatic Anisocoria. Anisocoria due to remote traumatic Horner syndrome (ptosis, miosis, and anhidrosis). Direct injury to the globe can cause traumatic miosis and most commonly traumatic mydriasis due to stunning of the ocular constrictor or dilator muscles. (Photo contributor: Christopher L. Stark, DO.)



FIGURE 1.23 ■ Normal Quadrigeminal Cistern. The normal appearance of this CSF space is shaped like a baby's bottom (see arrow). It is located within two cuts superiorly of the dorsum sellae. (Photo contributor: Lawrence B. Stack, MD.)

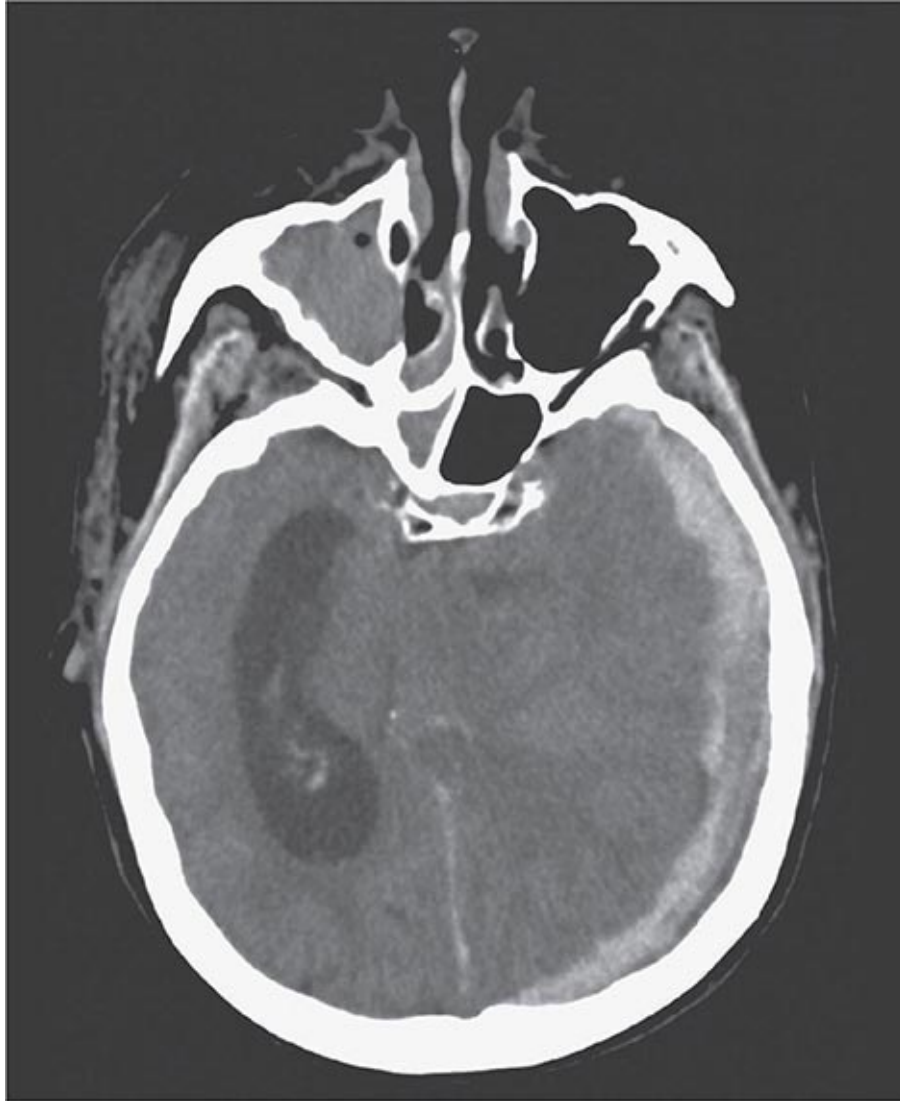


FIGURE 1.24 ■ Left Subdural Hematoma. A crescent-shaped subdural hematoma is seen on the left. The quadrigeminal cistern should be seen on this slice and is completely effaced, suggesting herniation. (Photo contributor: Lawrence B. Stack, MD.)

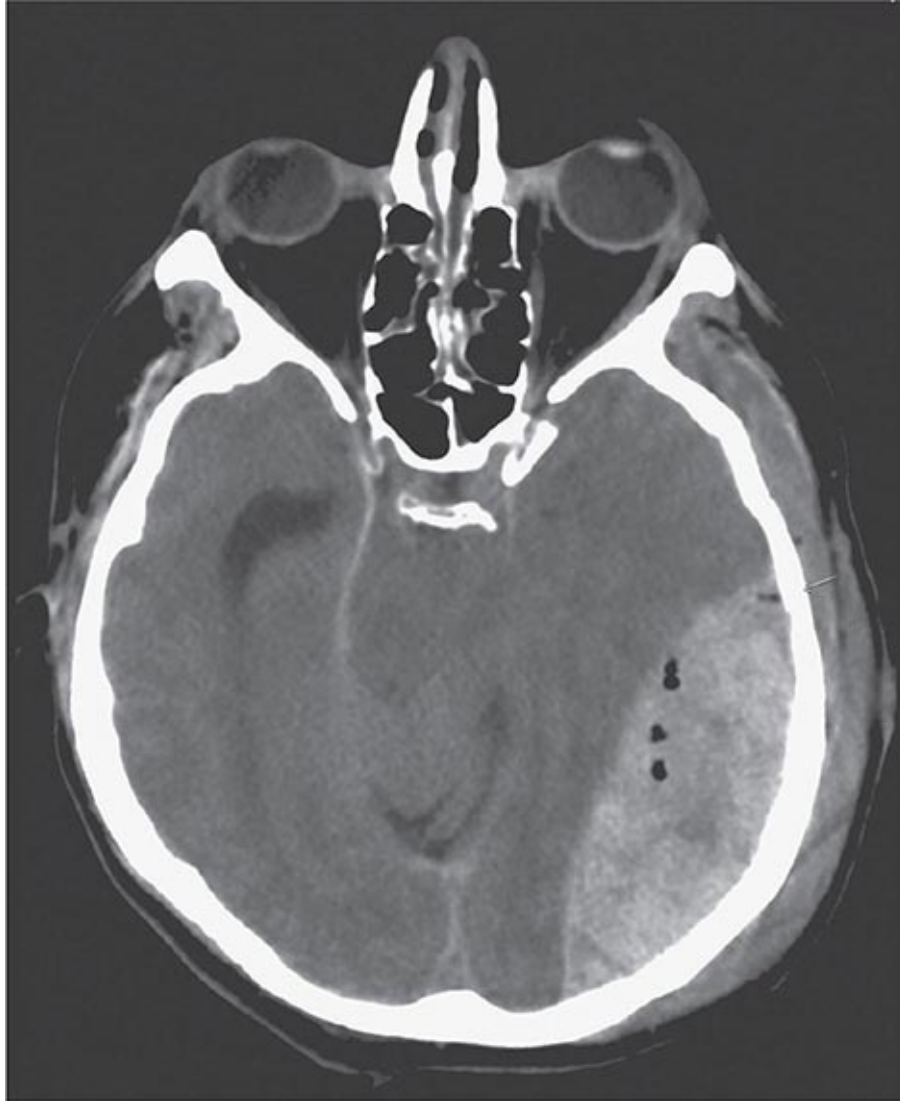


FIGURE 1.25 ■ Left Epidural Hematoma. A lens-shaped epidural hematoma is seen on the left. The quadrigeminal cistern should be seen on this slice and is completely effaced, suggesting herniation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.26 ■ Right Temporal Lobe Contusion. A temporal lobe contusion is seen on the right. The quadrigeminal cistern is partially effaced suggesting early herniation. A small area of pneumocephalus is seen against the right skull. (Photo contributor: Lawrence B. Stack, MD.)

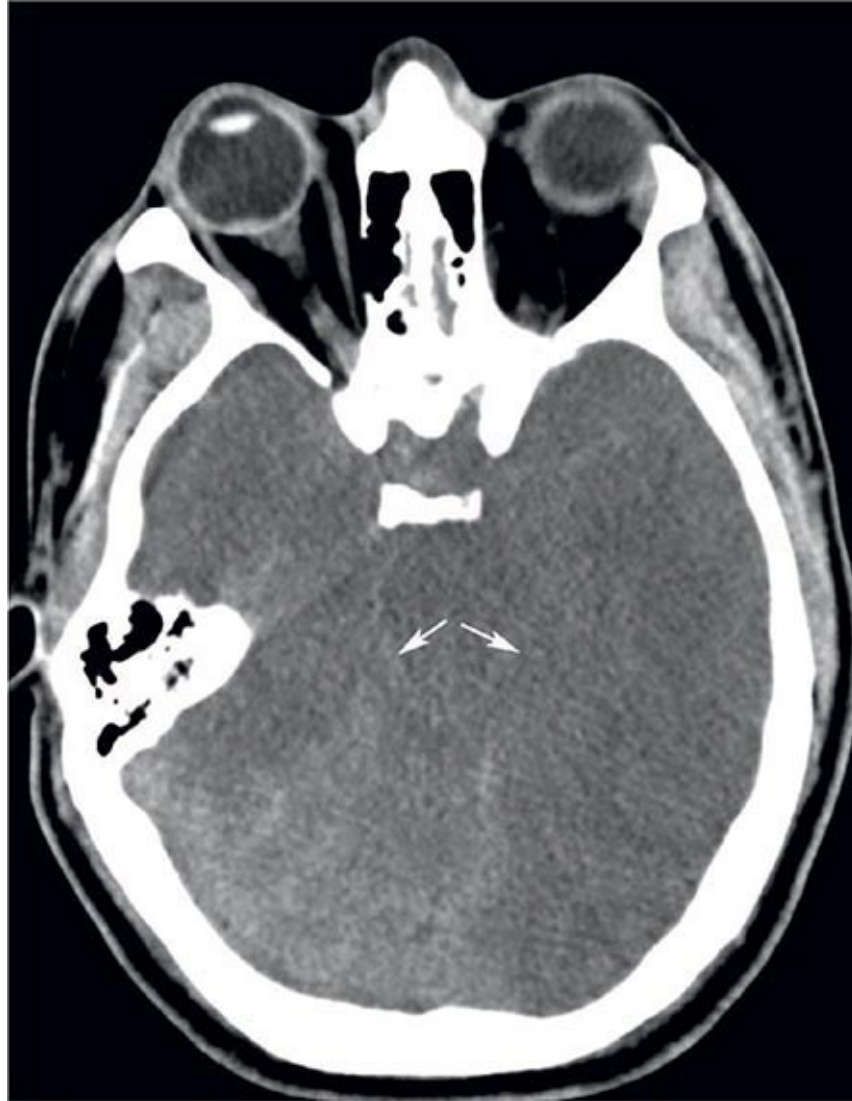


FIGURE 1.27 ■ Diffuse Edema. Diffuse edema causing uncal herniation is seen by effacement of the bilateral quadrigeminal cistern, which should be seen in the location of the arrows. (Photo contributor: Lawrence B. Stack, MD.)

NASAL INJURIES

Clinical Summary

Nasal fractures that require intervention are almost always evident with deformity, swelling, laceration, and ecchymosis. Fractures to adjacent bony structures, including the orbit, frontal sinus, or cribriform plate, often occur. Epistaxis may be due to a septal or turbinate laceration but can also be seen with

fractures of adjacent bones, including the cribriform plate. Septal hematoma is a rare but important complication that, if untreated, may result in necrosis of the septal cartilage and a resultant “saddle nose” deformity. A frontonasalethmoid fracture has nasal or frontal crepitus and may have telecanthus or obstruction of the nasolacrimal duct.

Management and Disposition

Identify and treat life-threatening injuries first. Patients with associated facial bone deformity or tenderness require CT to rule out facial fractures. Isolated nasal fractures rarely require radiographs. Refer obvious deformities within 2 to 5 days for reduction, after the swelling has subsided. Nasal fractures with mild angulation and without displacement may be reduced in the ED. Nasal injuries without deformity need only conservative therapy with an analgesic and a nasal decongestant. Immediately drain septal hematomas, with packing placed to prevent reaccumulation. Uncontrolled epistaxis may require nasal packing. Vigorously irrigate and suture lacerations overlying a simple nasal fracture and place the patient on antibiotic coverage. Complex nasal lacerations with underlying fractures should be repaired by a facial surgeon.

Pearls

1. Control epistaxis to perform a good intranasal examination. If obvious deformity is present, including a new septal deviation or deformity, treat with ice and analgesics and provide ENT referral in 2 to 5 days for reduction.
2. Bilateral nasal bleeding requires evaluation of each nostril individually, as bilateral nasal packing requires inpatient observation for oxygenation and apnea monitoring.
3. For patients who are anticoagulated, consider local application of liquid thrombin or tranexamic acid in conjunction with nasal packing. Check prothrombin time and international normalized ratio if on warfarin or if patient has unexplained bleeding.
4. Although the effectiveness of prophylactic antibiotics to prevent toxic shock syndrome is unproven, providers continue to often prescribe antistaphylococcal antibiotics to patients discharged with nasal packing.
5. Consider cribriform plate fractures in patients with clear rhinorrhea after nasal injury, as this finding may be delayed.
6. Patients with facial trauma should be examined for a septal hematoma.



FIGURE 1.28 ■ Nasal Fracture. Deformity is evident on examination. Note periocular ecchymosis indicating the possibility of other facial fractures (or injuries). The decision to obtain radiographs is based on clinical findings. (Photo contributor: David W. Munter, MD.)



FIGURE 1.29 ■ Septal Hematoma. A bluish, grapelike mass on the nasal septum. If untreated, this can result in septal necrosis and a saddle nose deformity. An incision, drainage, and packing are indicated. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.30 ■ Saddle Nose Deformity. Nasal septal necrosis resulting in saddle nose deformity. (Photo contributor: David Effron, MD.)



FIGURE 1.31 ■ Open Nasal Fracture. Lacerations that require a simple repair but are associated with an open nasal fracture may be primarily closed by the ED physician under consultation with ENT for close follow-up. (Photo contributor: Christopher L. Stark, DO.)



FIGURE 1.32 ■ Open Nasal Fracture. Lacerations with underlying fracture that require multilayer closure should be repaired by a facial surgeon and require antibiotics. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.33 ■ Minimally Displaced Nasal Fracture. Plain radiograph of a fracture of the nasal spine.
(Photo contributor: Lorenz F. Lassen, MD.)



FIGURE 1.34 ■ Comminuted Nasal Fracture. CT demonstrates a comminuted nasal bone fracture. (Photo contributor: Lawrence B. Stack, MD.)

ZYGOMA FRACTURES

Clinical Summary

The zygoma bone has two major components, the zygomatic arch and the body. The arch forms the inferior and lateral orbit, and the body forms the malar eminence of the face. Direct blows to the arch can result in isolated arch fractures. These present clinically with pain on opening the mouth secondary to the insertion of the temporalis muscle at the arch or impingement on the coronoid process. More extensive trauma can result in the “tripod fracture,” which consists of fractures through three structures: 1) the frontozygomatic

suture; 2) the maxillary process of the zygoma including the inferior orbital floor, inferior orbital rim, and lateral wall of the maxillary sinus; and 3) the zygomatic arch. Clinically, patients present with a flattened malar eminence and edema and ecchymosis to the area, with a palpable step-off on examination. Injury to the infraorbital nerve may result in infraorbital paresthesia, and gaze disturbances may result from injury to orbital contents. Subcutaneous emphysema may be present.

Management and Disposition

Maxillofacial CT best identifies zygoma fractures. Treat simple zygomatic arch or tripod fractures without eye injury with ice and analgesics and refer for delayed operative consideration in 5 to 7 days. Refer extensive tripod fractures or those with eye injuries urgently. Decongestants and broad-spectrum antibiotics are generally recommended for tripod fractures, since the fracture crosses into the maxillary sinus. Fractures with blood in the sinus should also be treated with antibiotics.

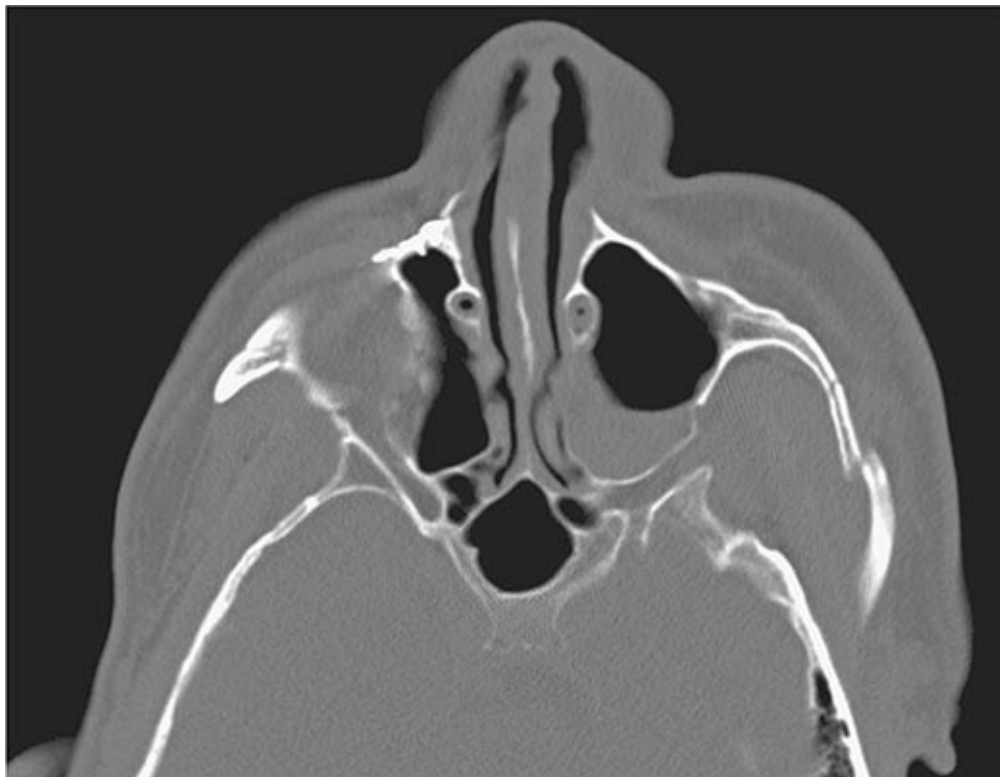


FIGURE 1.35 ■ Zygomatic Arch Fracture. Axial cut of a facial CT that reveals a minimally depressed zygomatic arch fracture. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Tripod fractures are often associated with orbital and ocular trauma. Palpate the zygomatic arch and orbital rims carefully for a step-off deformity.
2. Examine for eye findings such as diplopia, hyphema, or retinal detachment. Check for infraorbital paresthesia indicating injury or impingement of the 2nd division of cranial nerve V.
3. Visual inspection of the malar eminence from several angles (especially by viewing the area from over the head of the patient in the coronal plane) allows detection of a subtle abnormality.

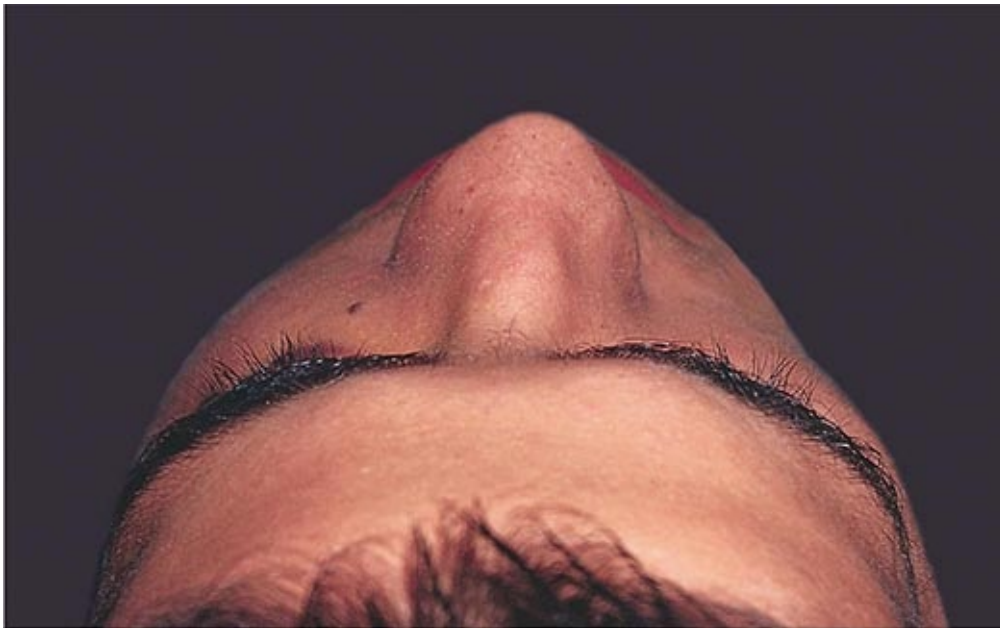


FIGURE 1.36 ■ Zygomatic Arch Fracture. Patient with blunt trauma to the zygoma. Flattening of the right malar eminence is evident. (Photo contributor: Edward S. Amrhein, DDS.)

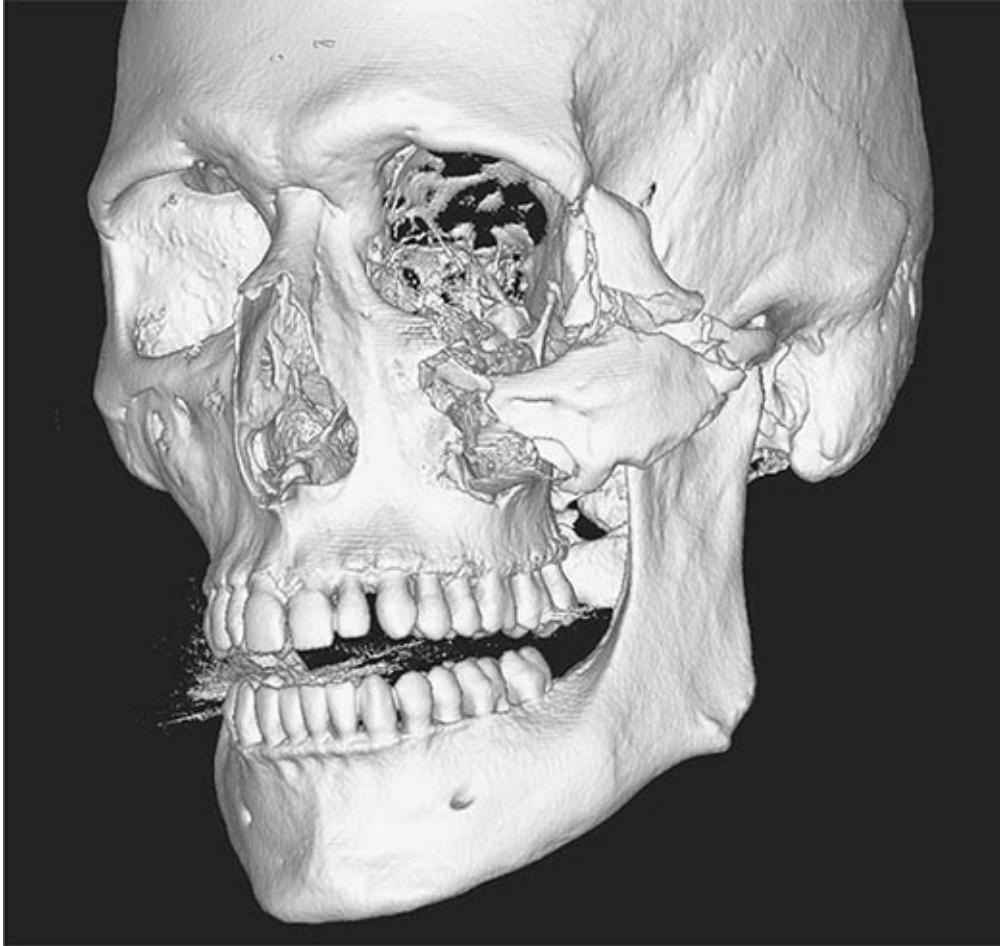


FIGURE 1.37 ■ Tripod Fracture. The fracture lines involved in a tripod fracture are demonstrated in this three-dimensional CT reconstruction. (Photo contributor: Patrick W. Lappert, MD.)

LEFORT FACIAL FRACTURES

Clinical Summary

All LeFort facial fractures involve the maxilla. Clinically, the patient has facial injuries, swelling, and ecchymosis. LeFort I fractures are those involving an area under the nasal fossa. LeFort II fractures involve a pyramidal area including the maxilla, nasal bones, and medial orbits. LeFort III fractures, sometimes described as craniofacial dissociation, involve the maxilla, zygoma, nasal and ethmoid bones, and the bones of the base of the skull. LeFort IV fractures have been described as a LeFort III fracture that also involves the frontal bone. Patients may have different LeFort fractures on each side of their face. Airway

compromise may be associated with LeFort II and III fractures. Physical examination is sometimes helpful in distinguishing the four. The examiner places fingers on the bridge of the nose and tries to move the central maxillary incisors with the other hand. If only the maxilla moves, a LeFort I is present; movement of the upper jaw and nose indicates a LeFort II; and movement of the entire midface and zygoma indicates a LeFort III. Because of the extent of LeFort II and III fractures, they may be associated with cribriform plate fractures and CSF rhinorrhea. The force required to sustain a LeFort II or III fracture is considerable, and associated brain or cervical spine injuries or other facial fractures are common.

Management and Disposition

Attend to the airway and life-threatening injuries first. Maxillo-facial CT best identifies LeFort injuries. Management of LeFort I fractures may involve only dental splinting and oral surgery referral. Consult on all LeFort II and III fractures for admission because of the need for operative repair. Epistaxis may be difficult to control in LeFort II and III fractures, in rare cases requiring intraoperative arterial ligation.

Pearls

1. Determine the need for immediate airway management, since the massive edema associated with LeFort II and III fractures may quickly lead to airway compromise.
2. Avoid nasotracheal intubation because of the possibility of intracranial passage.

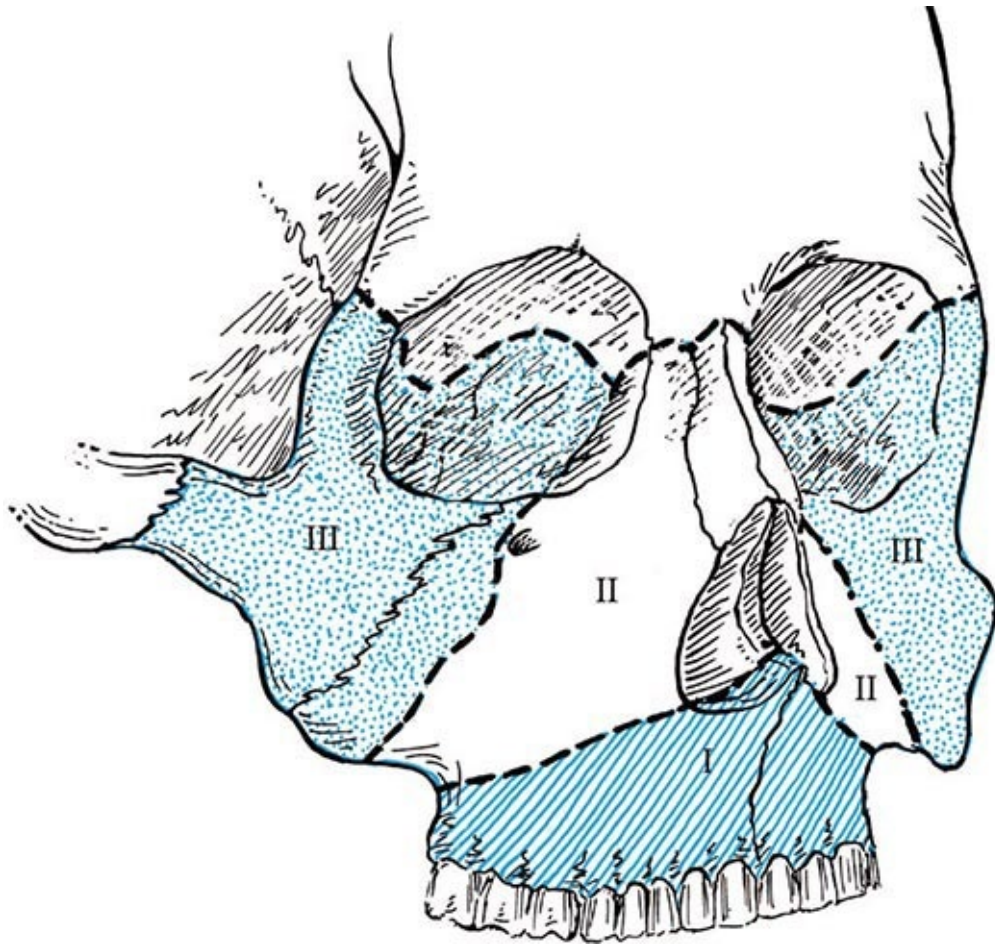


FIGURE 1.38 ■ LeFort Fractures. Illustration of the fracture lines of LeFort I (alveolar), LeFort II (zygomatic maxillary complex), and LeFort III (cranial facial dysostosis) fractures.

3. Serious facial trauma, like head injury, is often associated with cervical spine injuries; therefore, obtain a cervical spine CT.
4. Associated cranial injuries are common with severe facial trauma; therefore, obtain head CT.
5. If not recognized, an occult CSF leak may result in significant morbidity. Suspected CSF leaks require neurosurgical consultation.



FIGURE 1.39 ■ LeFort Facial Fractures. Patient with blunt facial trauma. Note the ecchymosis and edema. This patient sustained a left LeFort II fracture and a right LeFort III and intracranial hemorrhages. (Photo contributor: Stephen Corbett, MD.)



FIGURE 1.40 ■ LeFort Facial Fractures. Patient with blunt facial trauma who demonstrates the classic “dish face” deformity (depressed midface) associated with bilateral LeFort III fractures. (Photo contributor: Robert Schnarrs, MD.)

ORBITAL WALL FRACTURES

Clinical Summary

Orbital floor fractures are produced by two distinct mechanisms. The 1st is a true “blowout” fracture where all the energy is transmitted from a blunt object to the globe, causing increased orbital pressure blowing out either the orbital floor (most frequently) or medial wall. Fists and balls are the most common causative agents. This mechanism of injury is more likely to cause entrapment and globe injury. The 2nd mechanism of injury occurs when the energy from the blow is transmitted to the infraorbital rim, causing a buckling of the orbital floor. Patients with blowout fractures have periorbital ecchymosis and lid edema but may also sustain globe injuries, including chemosis, subconjunctival hemorrhage, or infraorbital numbness from injury to the infraorbital nerve. Other globe injuries seen with orbital wall fractures include corneal abrasion, hyphema, enophthalmos, proptosis, iridoplegia, dislocated lens, retinal tear, retinal detachment, and ruptured globe. Diplopia with upward gaze suggests entrapment of the inferior rectus or its supporting structures. Diplopia with lateral gaze suggests entrapment of the medial rectus muscle. Periorbital subcutaneous emphysema is frequently seen with orbital wall fractures because of the proximity to the sinuses.

Management and Disposition

Maxillofacial CT best identifies orbital wall fractures. Treat patients without eye injury or entrapment conservatively with ice and analgesics, and refer for follow-up in 2 to 3 days. Treat patients with blood in the maxillary sinus with antibiotics. Consult ophthalmology in patients with a true blowout fracture, as 30% of these patients sustain a significant globe injury. Immediately consult a facial trauma surgeon for patients with entrapment, as muscle necrosis may occur if muscle blood supply is compromised by the entrapment.

Pearls

1. Enophthalmos, limited upward gaze, diplopia with upward gaze, or infraorbital anesthesia from entrapment or injury to the infraorbital nerve should heighten suspicion of an orbital floor fracture.
2. Compare the pupillary level on the affected side with the unaffected side, since it may be lower from prolapse of the orbital contents into the maxillary sinus. Subtle abnormalities may be appreciated as an asymmetric corneal light

reflex (Hirschberg reflex).

3. Subcutaneous emphysema, a soft-tissue teardrop along the roof of the maxillary sinus on plain film, or an air-fluid level in the maxillary sinus on plain film should also be interpreted as evidence of an orbital floor fracture.
4. Patients with orbital wall fracture may present with subcutaneous emphysema after blowing their nose or air bubbles emanating from the tear duct.
5. Carefully examine the eye for visual acuity, hyphema, or retinal detachment, and the nose for septal hematoma.



FIGURE 1.41 ■ Orbital Floor Fracture. Sustained from blunt trauma to infraorbital rim causing buckling of the orbital floor. Maxillofacial CT is similar to that in [Fig. 1.43](#). Infraorbital hypesthesias and lack of entrapment suggest the buckling mechanism of injury. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.42 ■ Inferior Rectus Entrapment. The right inferior rectus muscle is entrapped within this orbital floor fracture limiting upward gaze. (Photo contributor: Lawrence B. Stack, MD.)

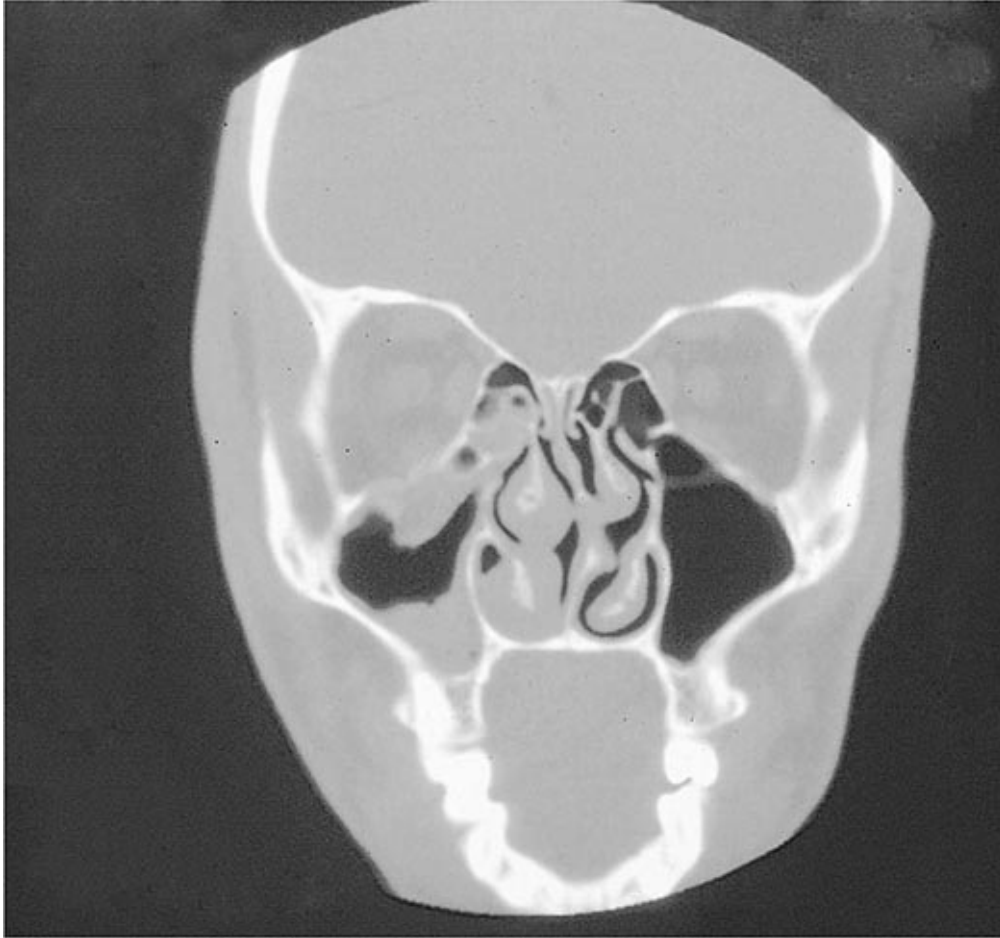


FIGURE 1.43 ■ Orbital Floor Fracture with Entrapment. Coronal CT of the patient in [Fig. 1.42](#) demonstrating the entrapped muscle extruding into the maxillary sinus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.44 ■ Medial Wall Orbital Fracture. Periorbital ecchymosis and swelling are seen in this patient with a medial wall orbital fracture. The patient blew her nose after the injury, and the swelling became more prominent. (Photo contributor: Lawrence B. Stack, MD.)

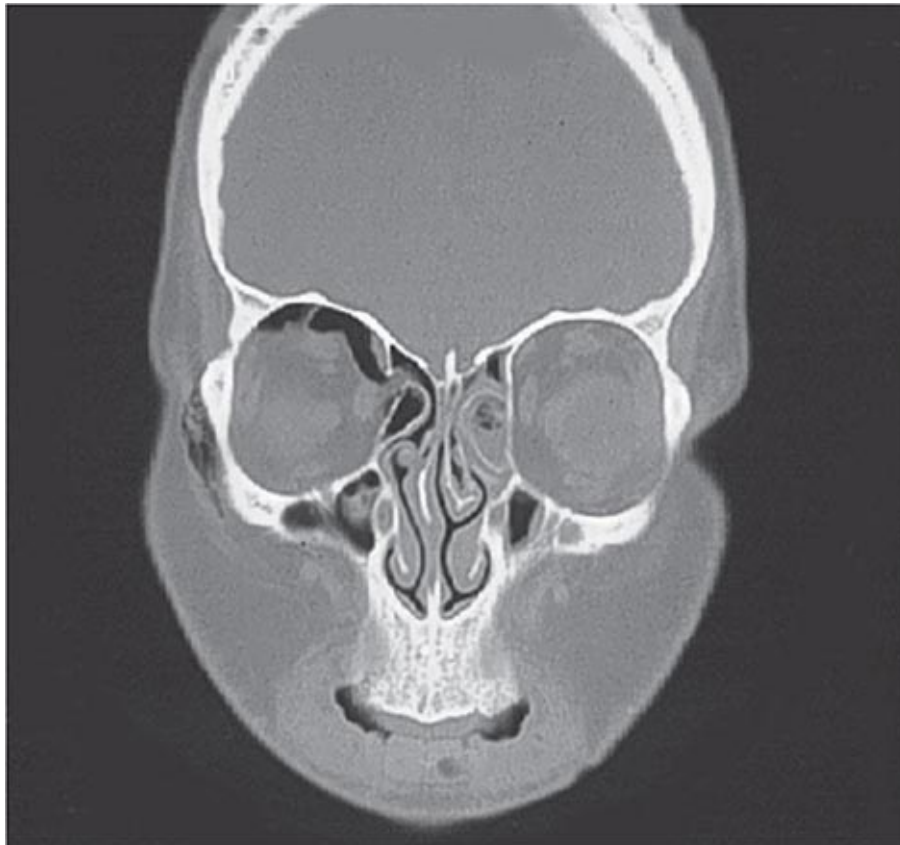


FIGURE 1.45 ■ CT of Medial Wall Orbital Fracture. Coronal CT of the patient in Fig. 1.44. Subcutaneous emphysema and orbital air are seen. An opening between the orbit and ethmoid air cells can be seen. (Photo contributor: Lawrence B. Stack, MD.)

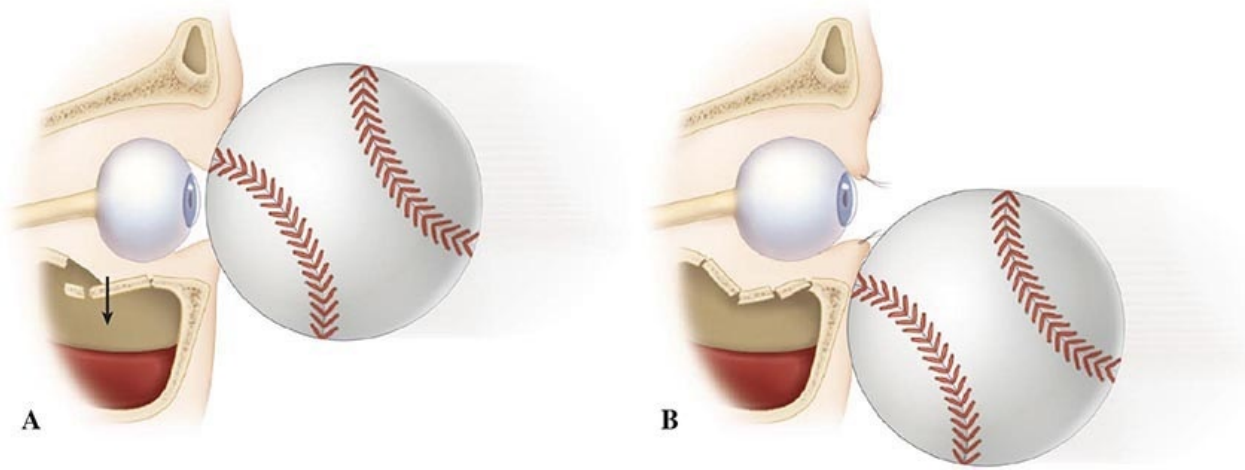


FIGURE 1.46 ■ Mechanisms of Orbital Wall Injury. (A) True “blowout” mechanism where all energy is transmitted to globe. (B) Buckle injury where energy is transmitted to inferior orbital rim, causing a buckling of the orbital floor.



FIGURE 1.47 ■ Medial Wall and Orbital Floor Fracture. Blowout fracture secondary to high velocity impact from a baseball. Notice the thread marking abrasions to the face. (Photo contributor: Christopher L. Stark, DO.)

TRAUMATIC EXOPHTHALMOS

Clinical Summary

Traumatic exophthalmos typically occurs from blunt orbital trauma causing an intraorbital hematoma that pushes the globe anteriorly. Patients present with periorbital edema, ecchymosis, a marked decrease in visual acuity, and an afferent pupillary defect in the involved eye. The exophthalmos, which may be obscured by periorbital edema, can be better appreciated from a superior view. Visual acuity may be affected by the direct trauma to the eye (retinal detachment, hyphema, globe rupture), compression of the retinal artery, or neuropraxia of the optic nerve.



FIGURE 1.48 ■ Traumatic Exophthalmos. (A) Blunt trauma resulting in periorbital edema and ecchymosis, which obscures the exophthalmos in this patient. The exophthalmos is not obvious in the anteroposterior view and can therefore be initially unappreciated. (B) The same patient viewed in the coronal plane from over the forehead demonstrating right eye exophthalmos. (Photo contributor: Frank Birinyi, MD.)

Management and Disposition

Maxillofacial or orbital CT best delineates the presence and extent of a

retrobulbar hematoma and associated facial or orbital fractures. Emergent ENT and ophthalmology consultation is indicated. Emergent lateral canthotomy and cantholysis to decompress the orbit can be sight saving.



FIGURE 1.49 ■ Retrobulbar Hematoma. CT of the patient in [Fig. 1.48](#) with right retrobulbar hematoma and traumatic exophthalmos. (Photo contributor: Frank Birinyi, MD.)



FIGURE 1.50 ■ Traumatic Exophthalmos. Proptosis, hyphema, periorbital ecchymosis, and marked swelling in the patient with a retrobulbar hematoma from severe head and face trauma. Examination findings are more obvious than in [Fig. 1.48](#). (Photo contributor: David Effron, MD.)

Pearls

1. The retrobulbar hematoma and exophthalmos may not develop for hours after the injury.
2. A subtle exophthalmos may be detected by looking down over the head of the patient and viewing the eye from the coronal plane.
3. Elevated intraocular pressure, relative afferent pupillary defect, and diminished visual acuity in patients with traumatic exophthalmos should have an emergent lateral canthotomy and cantholysis.

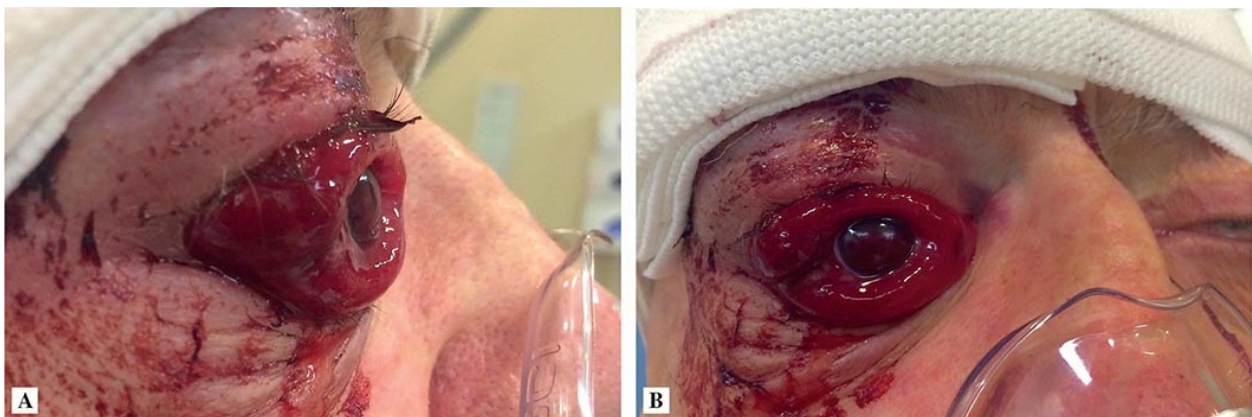


FIGURE 1.51 ■ Traumatic Exophthalmos. Exophthalmos with an initial intraocular pressure that was too high to read in this patient on anticoagulants suggests orbital compartment syndrome. Lateral canthotomy/cantholysis brought the pressure to normal. (Photo contributor: Pamela Loveland, MD.)

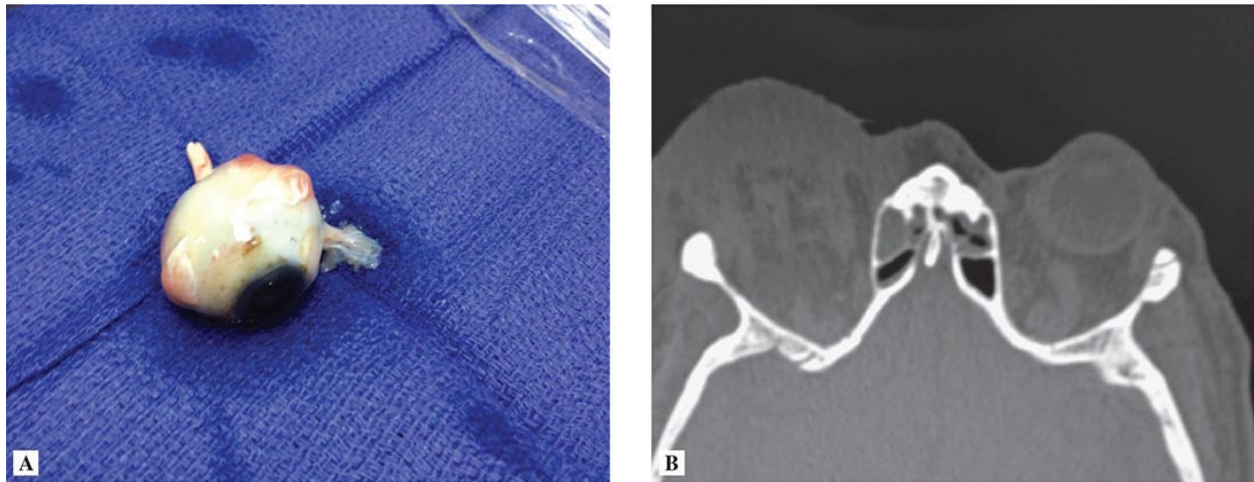


FIGURE 1.52 ■ Traumatic Enucleation. Complete enucleation of the right eye after a mechanical fall and hitting their face on the corner of a table. Family came with the patient with the eye in a plastic bag. No other injury is seen on orbit CT scan. (Photo contributor: Kevin S. Barlotta, MD.)

MANDIBULAR FRACTURES

Clinical Summary

Blunt trauma, mandibular pain, and malocclusion are typically seen in patients with mandibular fractures. A step-off in the dental line or ecchymosis or hematoma to the floor of the mouth is often present. Mandibular fractures may be open to the oral cavity, as manifested by gum lacerations. Dental trauma is frequently seen. Other clinical features include inferior alveolar or mental nerve paresthesia, loose or missing teeth, dysphagia, trismus, or ecchymosis of the floor of the mouth (considered pathognomonic). Multiple mandibular fractures are present in more than 50% of cases because of the ringlike structure of the mandible. Mandibular fractures are often classified as “favorable” or “unfavorable.” Fractures displaced by masseter muscle contraction are unfavorable and inevitably require fixation, whereas fractures that are not displaced by masseter contraction are favorable and, in most cases, will not require fixation. Injuries creating unstable mandibular fractures may create airway obstruction because the support for the tongue is lost. Mandibular fractures are also classified based on the anatomic location of the fracture.

Dislocation of the mandibular condyles may also result from blunt trauma and will always have associated malocclusion, typified by an inability to close the mouth.

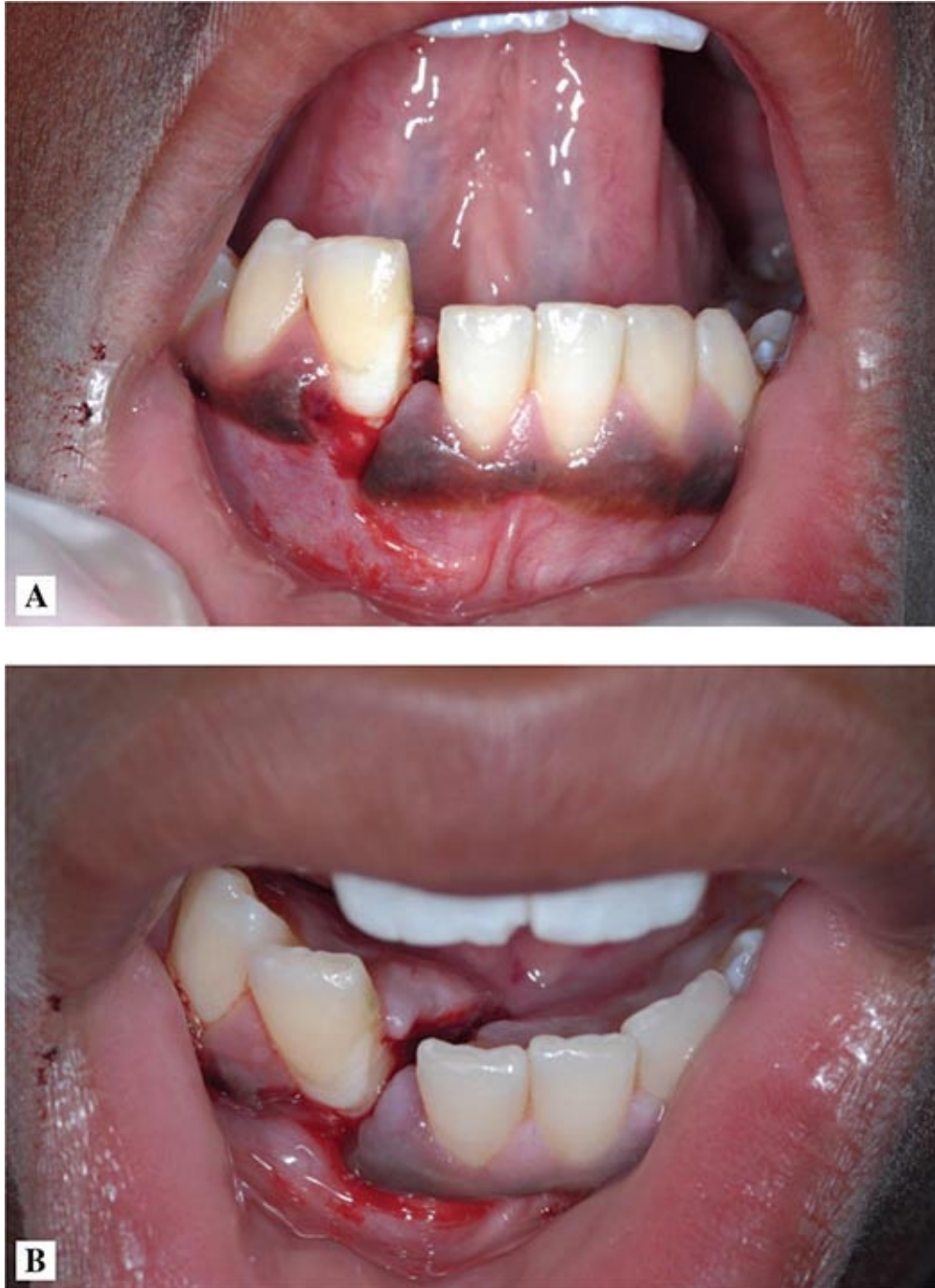


FIGURE 1.53 ■ Open Mandibular Fracture. An open fracture is suggested by the misaligned teeth and gingival disruption. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

A dental panoramic film is the best diagnostic image for evaluating mandibular trauma. If only plain films are available, obtain anteroposterior, bilateral oblique, and Towne views to evaluate the condyles. Treat nondisplaced fractures with analgesics, soft diet, and referral to oral surgery in 1 to 2 days. Displaced fractures, open fractures, and fractures with associated dental trauma need urgent consult. Treat all mandibular fractures with antibiotics effective against anaerobic oral flora (clindamycin, amoxicillin/clavulanate) and give tetanus prophylaxis if needed. The Barton bandage has been suggested to immobilize the jaw in the ED.

Pearls

1. The most sensitive sign of a mandibular fracture is malocclusion. The jaw will deviate toward the side of a unilateral condylar fracture on maximal opening of the mouth.
2. A nonfractured mandible should be able to hold a tongue blade between the molars tightly enough to break it off. There should be no pain in attempting to rotate the tongue blade between the molars.
3. Bilateral parasymphyseal fractures may cause acute airway obstruction in the supine patient. This is relieved by pulling the subluxed mandible and soft tissue forward and, in patients in whom the cervical spine has been cleared, by elevating the patient to a sitting position.

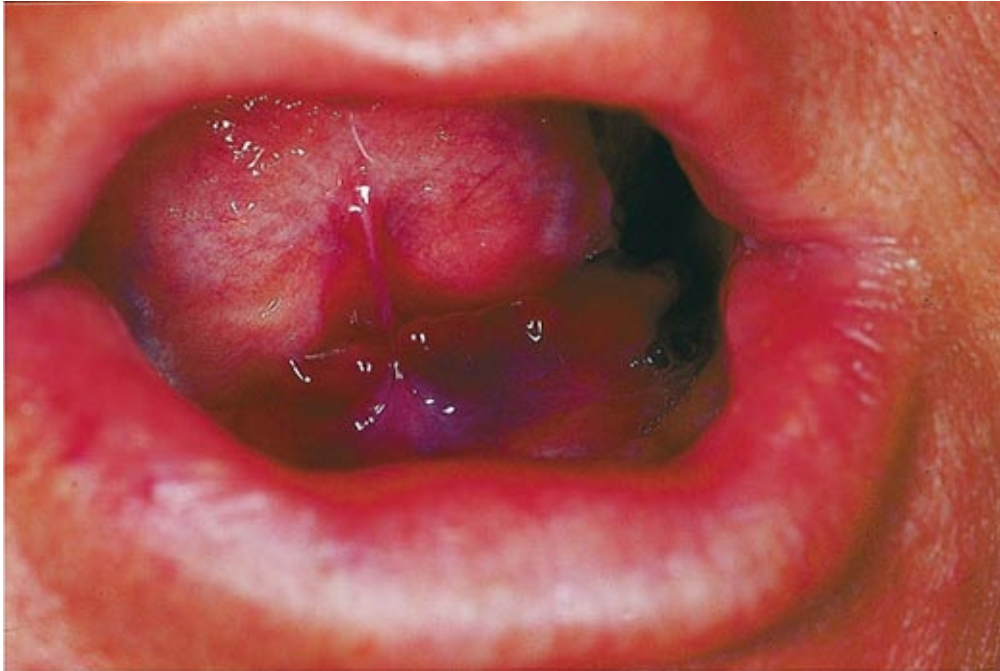


FIGURE 1.54 ■ Sublingual Hemorrhage. Hemorrhage or ecchymosis in the sublingual area is pathognomonic for mandibular fracture. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.55 ■ Bilateral Mandibular Fracture. The diagnosis is suggested by the bilateral ecchymosis

seen in this patient. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.56 ■ Favorable Mandibular Fracture. Dental panoramic view showing two nondisplaced mandibular fractures that are amenable to conservative therapy. (Photo contributor: David W. Munter, MD.)

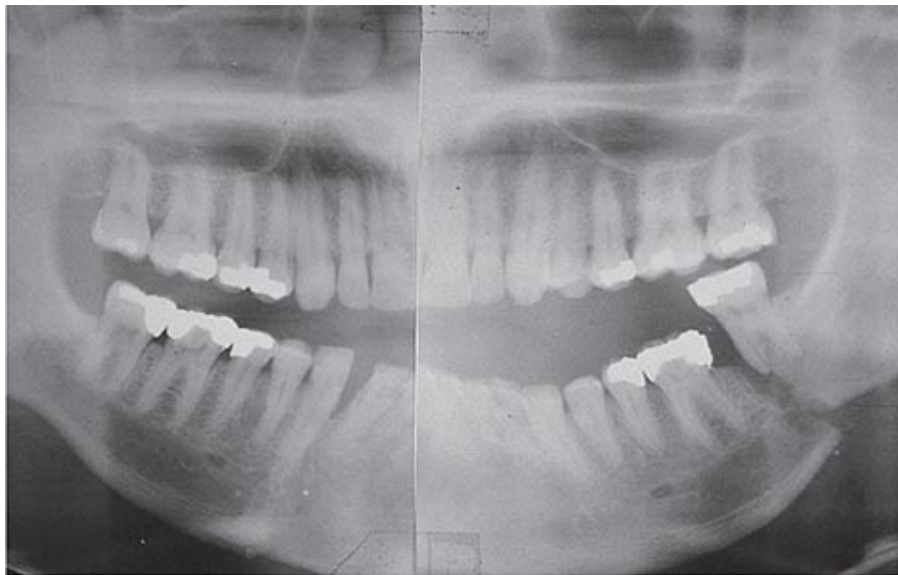


FIGURE 1.57 ■ Unfavorable Mandibular Fracture. Dental panoramic view demonstrating a mandibular fracture with obvious misalignment due to the distracting forces of the masseter muscle. (Photo contributor: Edward S. Amrhein, DDS.)

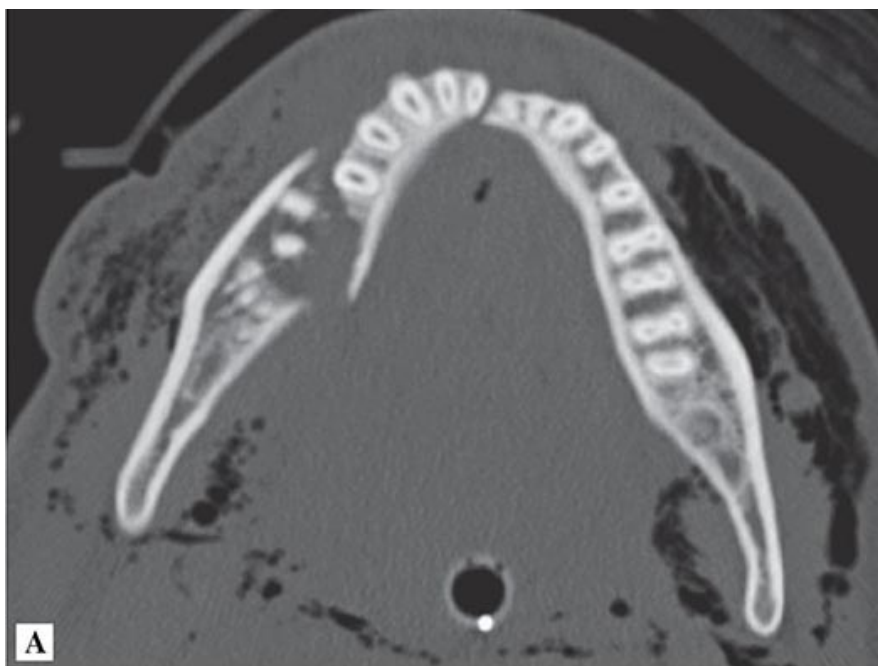


FIGURE 1.58 ■ Mandibular Fractures. Axial (A) and coronal (B) views of a maxillofacial CT reveal a right mandibular body and a parasymphyseal fracture. (Reproduced with permission from Block J, Jordanov MI, Stack LB, Thurman RJ, eds. *The Atlas of Emergency Radiology*. New York, NY: McGraw Hill; 2013; Fig. 1-99A and B.)

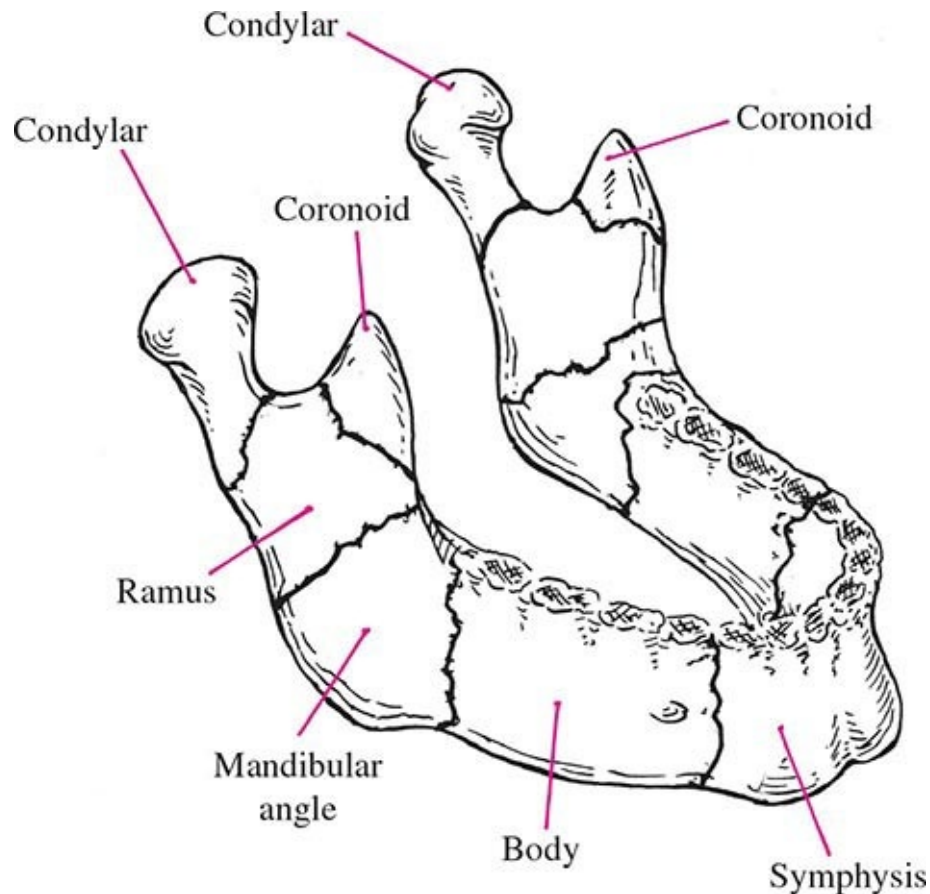


FIGURE 1.59 ■ Classification of Mandibular Fractures. Classification based on anatomic location of the fracture.

EXTERNAL EAR INJURIES

Clinical Summary

Injuries to the external ear may be open or closed. Blunt external ear trauma may cause a hematoma (otohematoma) of the pinna, which, if untreated, may result in cartilage necrosis and chronic scarring or further cartilage formation and permanent deformity (“cauliflower ear”). Open injuries include lacerations (with and without cartilage exposure) and avulsions.

Management and Disposition

Pinna hematomas must undergo incision and drainage or large-needle aspiration using sterile technique, followed by a pressure dressing to prevent reaccumulation of the hematoma. This procedure may need to be repeated several times; hence, after ED drainage, the patient is treated with antistaphylococcal antibiotics and referred to ENT or plastic surgery for follow-up in 24 hours. Lacerations must be carefully examined for cartilage involvement; if this is present, copious irrigation, closure, and postrepair oral antibiotics covering skin flora are indicated. Simple skin lacerations may be repaired primarily with nonabsorbable 6-0 sutures or surgical glue as appropriate. The dressing after laceration repair is just as important as the primary repair. If a compression dressing is not placed, hematoma formation can occur. Complex lacerations or avulsions normally require ENT or plastic surgery consultation.



FIGURE 1.60 ■ Pinna Contusion. Contusion without hematoma is present. Reevaluation in 24 hours is recommended to ensure a drainable hematoma has not formed. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.61 ■ Pinna Hematoma. A hematoma has developed, characterized by swelling, discoloration, ecchymosis, and flocculence. Immediate incision and drainage or aspiration is indicated, followed by an ear compression dressing. (Photo contributor: C. Bruce MacDonald, MD.)

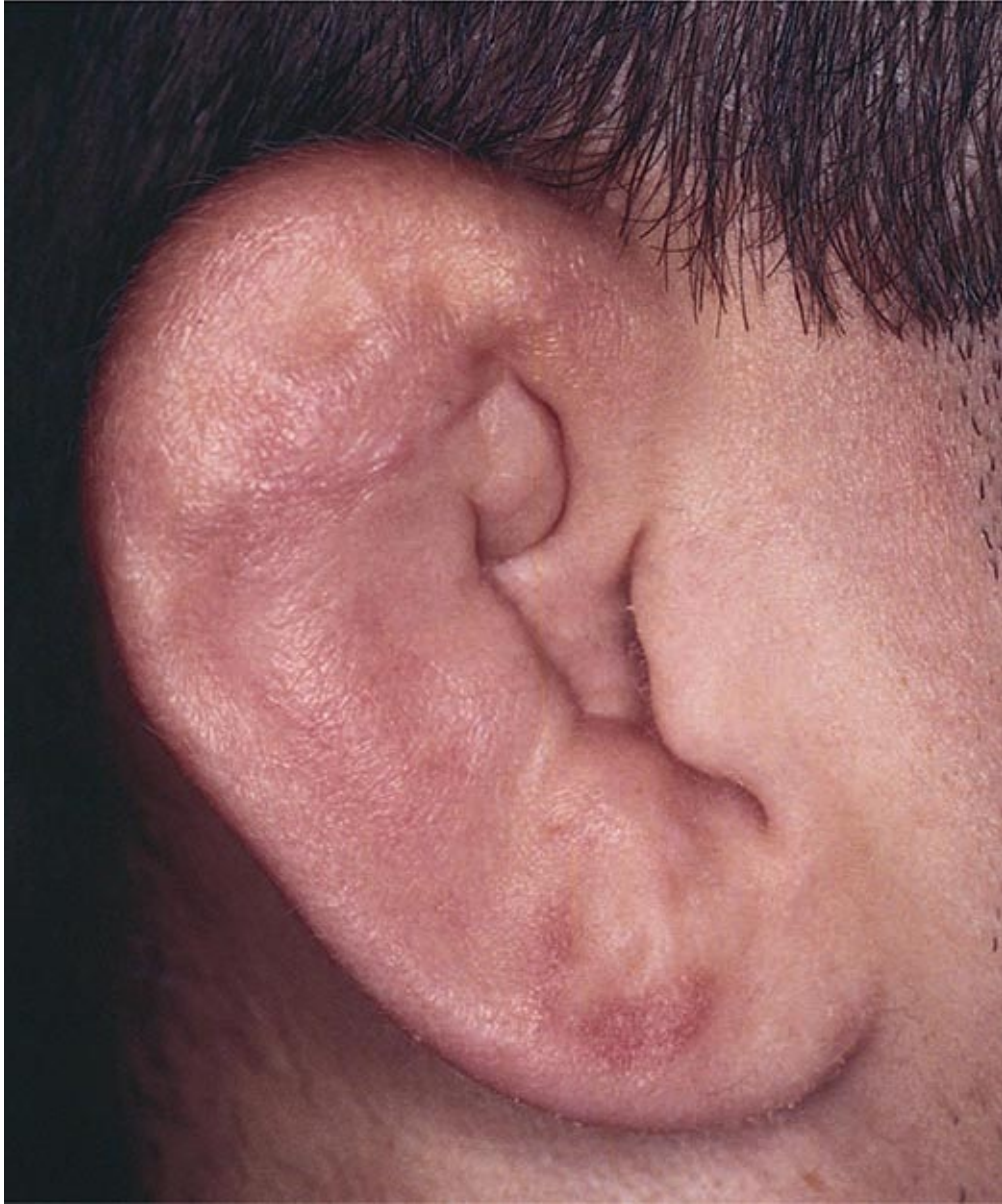


FIGURE 1.62 ■ Cauliflower Ear. Repeated trauma to the pinna or undrained hematomas can result in cartilage necrosis and subsequent deforming scar formation. (Photo contributor: Timothy D. McGuirk, DO.)



FIGURE 1.63 ■ Complete Avulsion of Partial Pinna. This ear injury, sustained in a fight, resulted when the pinna was bitten off. Plastic repair is needed. The avulsed part was wrapped in sterile gauze soaked with saline and placed in a sterile container on ice. (Photo contributor: David W. Munter, MD.)



FIGURE 1.64 ■ Partial Avulsion of Pinna. Partial avulsion of the pinna seen at its superior junction with the scalp. Cartilage exposure and injury prompts ENT consultation for repair. (Photo contributor: Eric Wu, MD.)

Pearls

1. Pinna hematomas may take hours to develop, so give patients with blunt ear trauma careful discharge instructions, with a follow-up in 12 to 24 hours to check for hematoma development.
2. Failure to adequately drain a hematoma, reaccumulation of the hematoma owing to a faulty pressure dressing, or inadequate follow-up increases the risk of infection of the pinna (perichondritis) or of a disfiguring cauliflower ear.
3. Copiously irrigate injuries with lacerated cartilage, which can usually be managed by primary closure of the overlying skin. Direct closure of the cartilage is rarely necessary and is indicated only for proper alignment, which helps lessen later distortion. Use a minimal number of absorbable 5-0 or 6-0 sutures through the perichondrium.
4. Lacerations to the lateral aspect of the pinna should be minimally debrided because of the lack of tissue at this site to cover the exposed cartilage.
5. In the case of an avulsion injury, the avulsed part should be cleansed, wrapped

in saline-moistened gauze, placed in a sterile container, and then placed on ice to await reimplantation by ENT.



FIGURE 1.65 ■ Complete Avulsion of Entire Pinna. This injury occurred as a result of a motor vehicle crash. The pinna was not found. (Photo contributor: Kevin S. Barlotta, MD.)

FACIAL NERVE INJURY

Clinical Summary

Facial paralysis can be a devastating consequence of blunt or penetrating trauma. The facial nerve is injured in 7% to 10% of temporal bone fractures. After facial nerve injury, the ensuing paralysis is immediate in 27% of cases and has a delayed presentation in 73% of patients. Penetrating trauma can occur anywhere along the nerve tract but is especially vulnerable after it exits the skull, just posterior to the styloid process of the temporal bone, traveling anterior to the

external ear to the parotid gland. Within the gland, the nerve terminates splitting into five branches. Injury to the nerve extracranially causes purely motor deficits to the muscle of facial expression. Intracranial injury, proximal to the stylomastoid foramen, produces mixed motor and sensory deficits. The signs and symptoms differ depending on the distance from the origin of the nerve and any branches from the nerve before and after the area of injury. Possible deficits include ipsilateral hyperacusis (sound hyper-sensitivity); ipsilateral loss of taste to the anterior two-thirds of the tongue; reduced parasympathetic innervation to the mucus glands of the oral cavity, nose, and pharynx (decreasing salivation); ipsilateral reduced lacrimal fluid production; and motor paresis as seen with extracranial injury.

Management and Disposition

Immediate complete traumatic paralysis warrants surgical exploration; delayed paralysis or incomplete paresis should be treated medically with high-dose steroids. Operating on these patients in a delayed fashion is reasonable as most patients require other acute management. This delay does not necessarily worsen their prognosis, and surgery can still be of benefit even 3 months following injury.

Pearls

1. Manifestations of traumatic CN7 injury are delayed in 73% of patients.
2. Temporal bone fractures are often associated with facial nerve injury.
3. Injury to CN7 motor nerve fibers affects muscles of facial expression and the stapedius muscle.
4. Injury to CN7 parasympathetic nerve fibers affects glands of the oral cavity and the lacrimal gland.
5. Injury to CN7 sensory nerve fibers affects external auditory meatus, tympanic membrane, the pinna, and taste sensation from the anterior two-thirds of the tongue.



FIGURE 1.66 ■ Traumatic Cranial Nerve 7 (CN7) Palsy—Laceration. Patient was involved in a bar fight and was cut with a broken beer bottle lacerating CN7 along with the tissues adjacent to the external ear. (Photo contributor: Christopher L. Stark, DO.)



FIGURE 1.67 ■ Traumatic CN7 Palsy—Motor Findings. Laceration of the nerve occurred close to the exit from the skull posterior to the styloid process. Injury to the nerve extracranially causes purely motor deficits to the muscle of facial expression. (Photo contributor: Christopher L. Stark, DO.)

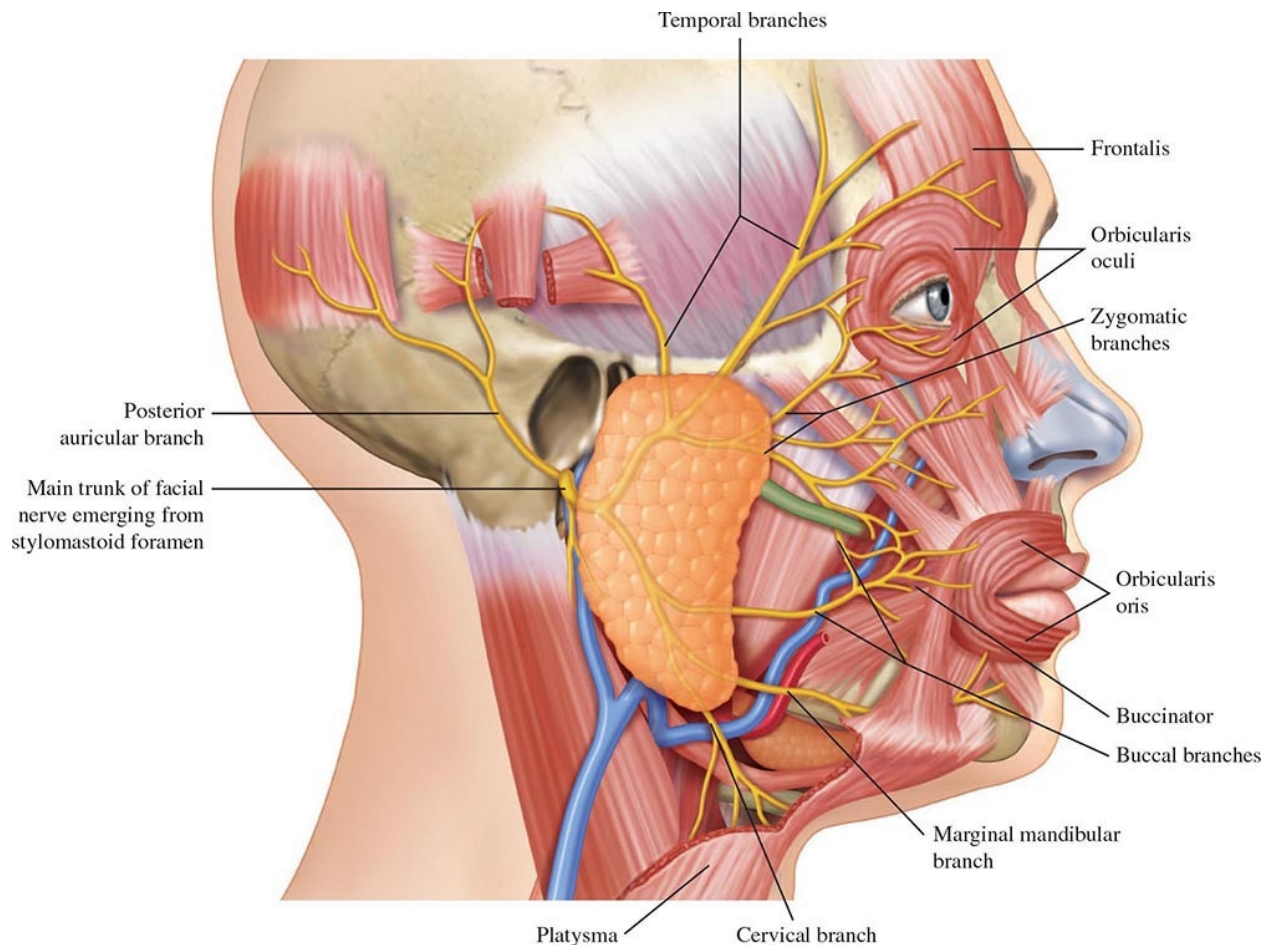


FIGURE 1.68 ■ CN7 Pathway. Main trunk of CN7 exits the skull, through the stylomastoid foramen, just posterior to the styloid process of the temporal bone. CN7 travels extracranially inferior and anterior to the external ear to the parotid gland. Within the gland, the nerve terminates splitting into five branches (cervical, marginal mandibular, buccal, zygomatic, and temporal branches).

PENETRATING FACIAL TRAUMA

Clinical Summary

Injury patterns of penetrating facial trauma can be predicted based on projectile type, entry location, and path. Midface injuries extend from the supraorbital rim superiorly to the oral commissure inferiorly and to the external auditory meatus posteriorly. Mandibular injuries extend from the oral commissure superiorly and to the lower border of the mandible inferiorly. Shotgun wounds typically involve both facial zones and will involve one or both eyes in 50% of patients. Fifty percent of patients with gunshot wounds (GSWs) to the mandible will require an

emergency airway. Stab wounds are less likely to require emergency airway than GSWs. Additional structures that require consideration during the ED evaluation include brain, blood vessels, and esophagus.

Management and Disposition

After the primary survey, strongly consider intubation in patients with any gunshot injury to the mandible, blood or swelling in the oropharynx, or any close range (< 7 m) shotgun injury. If evidence suggests a projectile remains in the face or cranium, obtain plain films in two planes to help guide subsequent evaluation such as CT angiogram or fine cuts of the orbits. CT will provide more detailed information in the injured structures to guide therapy. Removal of any projectile should only be performed after significant structure injury has been excluded and preparation for the consequences of removal is complete.

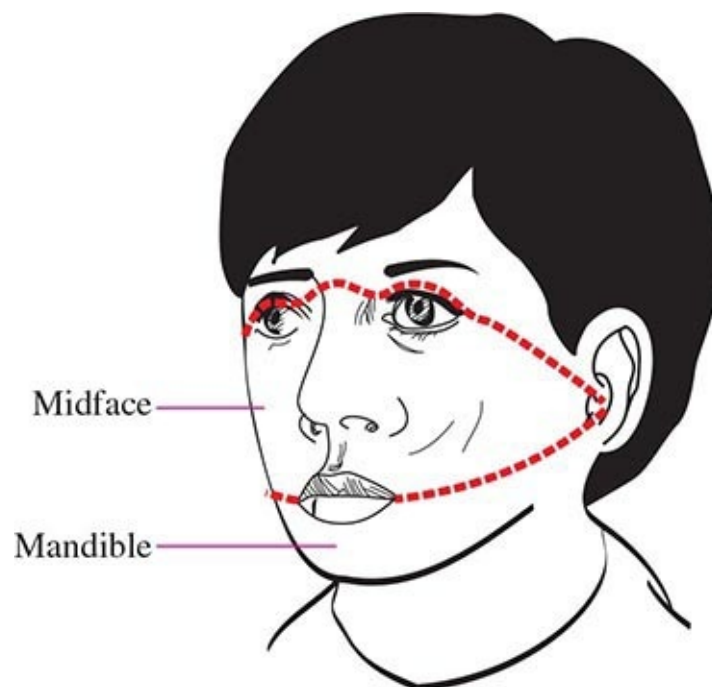


FIGURE 1.69 ■ Facial Zones of Injury. (Reproduced with permission from Chen AY, Stewart MG, Raup G. Penetrating injuries of the face. *Otolaryngol Head Neck Surg.* 1996;115:464-470. Copyright © 1996, © SAGE Publications.)

Pearls

1. Due to the likelihood of multisystem injury (vascular, ocular, cranial, face) with penetrating facial trauma, the patient should be transferred to a facility

with comprehensive sub-specialty trauma care.

2. Intubation should be strongly considered in all patients with GSWs to the mandible or with GSWs to the midface if there is any blood or swelling in the oral cavity.
3. Removal of any projectile is best done in the operating room.



FIGURE 1.70 ■ Midface Injury. A jackhammer bit is lodged into the right maxillary sinus. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 1.71 ■ Midface Injury Radiograph. Plain film of patient in [Fig. 1.70](#). CT confirmed no other injury. Projectile was removed in the operating room. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 1.72 ■ Penetrating Facial Trauma. Facial wound due to a flare gun injury. Surgical closure is delayed until the viable tissue has declared itself, which usually requires approximately 2 weeks. (Photo contributor: Michael Bezzerides, MS4.)



FIGURE 1.73 ■ Penetrating Facial Trauma. Intraoral view of the flare gun injury. Note necrotic the wound margins. (Photo contributor: Michael Bezzerides, MS4.)



FIGURE 1.74 ■ Penetrating Facial Trauma. Intra oral view of a stick which entered the maxillary vestibule after a mechanical fall. Note the trajectory of the stick seen on the accompanying CT scan. (Photo contributor: Lawrence B. Stack, MD.)

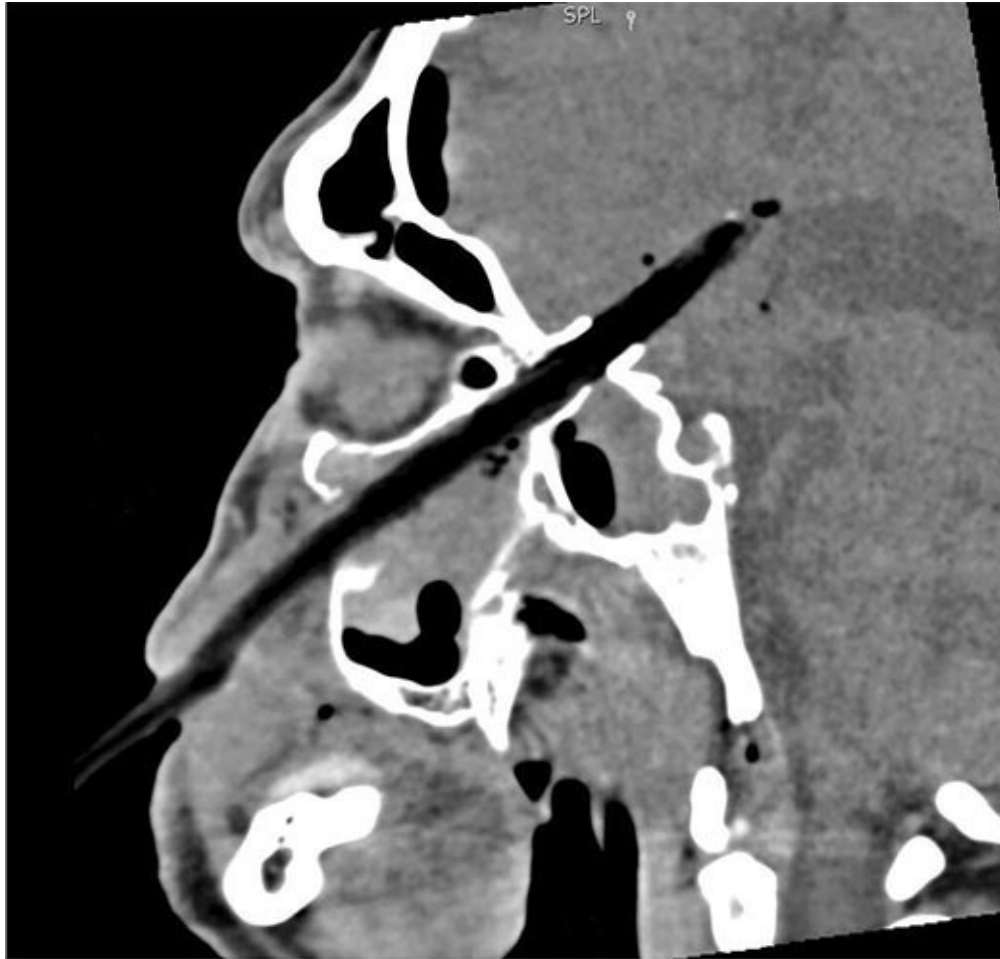


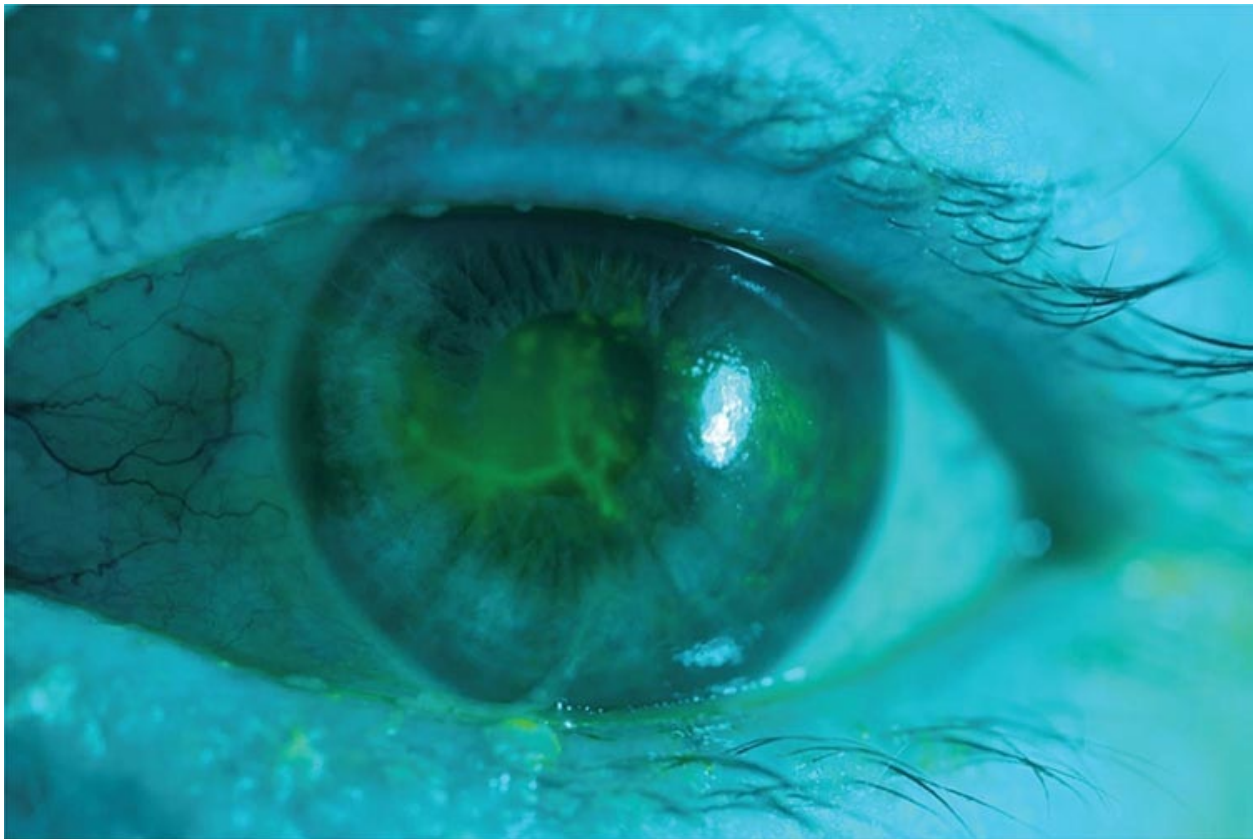
FIGURE 1.75 ■ Penetrating Facial Trauma. CT scan. Note the trajectory of the stick from the oral cavity into the cranial vault. (Photo contributor: Steven Barron Frazier, MD.)

The author wishes to thank David Munter for his contributions to prior editions.

Chapter 2

OPHTHALMIC CONDITIONS¹

Manpreet Singh
Denise Whitfield



Herpes Simplex Keratitis. Branched dendritic lesion seen on the cornea in a patient with herpes simplex virus. (Photo contributor: Lawrence B. Stack, MD.)

PALLOR/ANEMIA

Clinical Summary

The ocular conjunctiva can be a window to determining systemic disease. An unhealthy pale appearance of the palpebral conjunctiva is a sign of anemia. Anemia is defined as a reduced count of circulating red blood cells and can occur through blood loss or pathways that hinder red blood cell production. Patients with chronic blood loss may have long-standing anemia with compensatory mechanisms. Macrocytic anemias can result from any process that causes reticulocytosis, folate or vitamin B₁₂ deficiencies, alcohol abuse, liver disease, or hypothyroidism. Microcytic anemia is commonly due to iron deficiency, disorders of heme synthesis, thalassemias, or chronic disease. In the emergency department (ED), clinical priorities for an anemic patient include determining hemodynamic stability, chronicity of the anemia, and the suspected underlying cause.

Management and Disposition

In the acute setting, one should rule out causes of acute blood loss and initiate resuscitation as indicated. Patients may require blood transfusion if symptomatic or if they have comorbid risk factors for poor outcome. A complete blood count with differential to delineate the type of anemia and examine for other cell line deficiencies should always be formed. Patient disposition is determined by the underlying cause of the anemia, symptoms associated with the anemia, and the patient's hemodynamic stability.

Pearls

1. Conjunctival pallor is a simple physical exam finding that can identify anemic patients.
2. The decision to transfuse red blood cells in an anemic patient is multifactorial. In hemodynamically stable patients without symptoms, most guidelines suggest a transfusion threshold between 7 and 8 g/dL with consideration of individual patient characteristics.
3. An anemic patient with acute coronary syndrome requires transfusion, not aspirin.



FIGURE 2.1 ■ Conjunctival Pallor. Conjunctival pallor is noted in this patient with anemia (**A**) that is improved after blood transfusion (**B**). (Photo contributor: Kevin J. Knoop, MD, MS.)

OSTEOGENESIS IMPERFECTA

Clinical Summary

Osteogenesis imperfecta (OI) is a hereditary connective tissue disorder. The disease manifests on a spectrum with patients with more severe disease often suffering from fractures with minimal or no trauma. Very severe forms of disease can result in perinatal death. OI manifests most commonly as excess or atypical fractures, but other findings include short statures, hearing loss, easy bruising, and a scleral tint with the sclerae commonly appearing blue.

For a patient presenting to an ED with atypical fractures, child abuse should be considered. A full history and physical examination may lead to a suspected diagnosis of OI in a patient where one is not already established. There is no definitive laboratory tests for OI, but specific cDNA analysis can be done to detect type I collagen mutations and diagnose the disease.

Management and Disposition

The goals of management for OI patients is reducing the risk and rate of fractures, minimizing pain, and maximizing functional capabilities. Bisphosphates can be used as fracture prevention therapy. Physical therapy can also be instituted to enhance mobility and prevent fractures. Prognosis for OI

patients depends on type, with shortened life spans related to forms with more severe thoracic vertebral deformities and immobility. Most individuals with OI lead long and productive lives.

Pearls

1. OI patients with scleral tint may have sclerae that appear blue, gray, or purple. The coloration results from the projection of choroidal vein color through defective collagen layers.
2. Consider OI in patients with recurrent, atypical fractures with a thorough history and physical exam once nonaccidental trauma is excluded.



FIGURE 2.2 ■ Blue Scleral Tint. Patient with osteogenesis imperfecta with typical blue scleral tint. (Photo contributor: Alan B. Storrow, MD.)

CONJUNCTIVAL ICTERUS

Clinical Summary

Conjunctival icterus is an indicator of elevated bilirubin levels (hyperbilirubinemia) due to an underlying disease that affects the metabolism or excretion of bilirubin. The eye conjunctiva is a thin layer that overlies the sclera. It is one of the very first tissues to change in color due to increasing bilirubin levels. In general, it is seen when bilirubin levels reach at least 2 to 4 mg/dL. The term *scleral icterus* is an incorrect term to describe this condition (misnomer) because this tissue has the least amount of bilirubin staining it.

Management and Disposition

Jaundice itself is not a disease, but rather a sign of many possible underlying diseases. Patients presenting to the ED with jaundice present a diagnostic opportunity to identify the etiology. The pathophysiologic mechanism can be categorized as prehepatic/hemolytic, hepatic/hepatocellular, and posthepatic/cholestatic based on physiologic mechanism. A typical liver panel or liver function tests, including aminotransferases (alanine aminotransferase, aspartate aminotransferase), alkaline phosphatase, bilirubin (which causes the jaundice), and protein levels (total and albumin), can aid in the diagnosis. In general, elevated levels are not harmful to adults. However, once at a certain level, bilirubin-induced brain dysfunction (kernicterus) can occur and is typically seen in newborn patients.

Pearls

1. Conjunctival sclera is a sign, *not* a disease. Consider a range of etiologies when patients present to the ED.
2. Refer to this finding as conjunctival icterus, rather than scleral icterus (misnomer).



FIGURE 2.3 ■ Conjunctival Icterus. Patient with yellow-tinged conjunctiva. (Photo contributor: Kevin J. Knoop, MD, MS.)

NEONATAL CONJUNCTIVITIS (OPHTHALMIA NEONATORUM)

Clinical Summary

Neonatal conjunctivitis is acquired either during birth with passage through the mother's cervix and vagina or from cross-infection in the neonatal period. Presenting symptoms for *Neisseria gonorrhoeae* infection include a hyperacute bilateral conjunctivitis with copious purulent discharge, lid swelling, chemosis, and preauricular adenopathy.

More common etiologies include *Chlamydia trachomatis*, viruses (herpes simplex), and bacteria (*Staphylococcus aureus*, *Streptococcus pneumoniae*, *Haemophilus* species). For chlamydial conjunctivitis, the clinical features range from mild swelling with a watery discharge to marked lid swelling with a red and thickened conjunctiva with a blood-stained discharge. Fluorescein staining of herpes simplex conjunctivitis demonstrates epithelial dendrites.



FIGURE 2.4 ■ Neonatal Conjunctivitis (Ophthalmia Neonatorum). Copious purulent drainage in a newborn with neonatal gonococcal conjunctivitis. (Reprinted with permission of the American Academy of Ophthalmology. *Eye Trauma and Emergencies: A Slide-Script Program*. San Francisco, 1985. © American

Management and Disposition

With any form of neonatal conjunctivitis, Gram stain and culture are indicated. Begin treatment in the ED, and admit newborns with suspected gonococcal conjunctivitis. Evaluate concurrently for *C trachomatis*, since coinfection is common. Nucleic acid amplification testing (NAAT) is highly sensitive in confirming gonococcal or chlamydial infection.

Treatment for chlamydial conjunctivitis is based on a positive diagnostic test. While culture is the gold standard, NAATs, despite lacking US Food and Drug Administration approval, are reported to perform similarly. Untreated disease can result in corneal and conjunctival scarring. Bacterial neonatal conjunctivitis that is neither gonococcal nor chlamydial may be treated with erythromycin antibiotic ointment and should be reevaluated in 24 hours.



FIGURE 2.5 ■ Neonatal Conjunctivitis. Thick purulent drainage in a newborn diagnosed with neonatal gonococcal conjunctivitis. (Photo contributor: David Effron, MD.)



FIGURE 2.6 ■ Neonatal Conjunctivitis. A purulent discharge is seen in this newborn. Management includes excluding *Neisseria* and *Chlamydia*. (Photo contributor: Kevin J. Knoop, MD, MS.)

Herpes simplex conjunctivitis is treated with intravenous (IV) acyclovir and topical trifluridine. Despite the appearance of a localized herpes infection, there is high risk for central nervous system (CNS) or disseminated infection.

Evaluation of the newborn's parents should be undertaken in neonatal conjunctivitis due to gonococcus, *Chlamydia*, or herpes simplex virus (HSV).

Pearls

1. The “rule of fives” may help predict the most likely bacterial cause.

0-5 days	<i>Gonococcus</i>
5 days-5 weeks	<i>Chlamydia</i>
5 weeks-5 years	<i>Staphylococcus</i> , <i>Streptococcus</i> , <i>Haemophilus</i>
2. Blindness can result from gonococcal eye infection in the neonate because the organism can invade the cornea. It is one of the few emergency conjunctival infections.

3. Nasolacrimal duct obstruction is common (up to 20%) in newborns and may present with findings suggestive of conjunctivitis. It is a diagnosis of exclusion in the neonate.
4. Advise parents that infants treated with macrolides are at risk for developing hypertrophic pyloric stenosis.



FIGURE 2.7 ■ Neonatal Conjunctivitis. A scant crusty discharge is seen in this newborn who was diagnosed in follow-up with nasolacrimal duct obstruction. (Photo contributor: Kevin J. Knoop, MD, MS.)

TABLE 2.1 ■ SUMMARY OF NEONATAL CONJUNCTIVITIS

Etiologic Agent	Time of Onset	Clinical Features	Treatment
Chemical	Day 1	Common with silver nitrate; erythromycin more commonly used now for this reason	Self-limited
<i>N gonorrhoeae</i>	Day 2-7	May cause hyperacute disease with profuse discharge	Ceftriaxone IV. Caution in hyperbilirubinemia. Topicals alone inadequate
<i>C trachomatis</i>	Day 3-14	Clinical severity varies. Common cause of blindness worldwide	Erythromycin PO Azithromycin (alternate)
HSV 1, 2	Day 2-16	Should be suspected if child has any vesicular lesions on body	Vidarabine or trifluridine topically; consider systemic acyclovir
Other bacterial organisms	Day 2 and up	Empiric treatment based on Gram stain	Erythromycin ointment for gram positive; gentamicin or tobramycin ointment for gram negative

BACTERIAL CONJUNCTIVITIS

Clinical Summary

Bacterial conjunctivitis is characterized by the acute onset of conjunctival injection and a thick yellow, white, or green mucopurulent drainage. Lid edema, erythema, and chemosis may also be seen. *S aureus* is the most common causative bacteria. *S pneumoniae* and *Haemophilus influenzae* occur more frequently in children.

Hyperacute bacterial conjunctivitis, the most severe form of acute purulent conjunctivitis, is associated with *N gonorrhoeae*. Symptoms are hyperacute in onset, and findings include a purulent, thick, copious discharge; eyelid swelling and tenderness; marked conjunctival hyperemia; chemosis; and preauricular adenopathy. The condition is serious and threatens sight because *Neisseria* species are capable of invading an intact corneal epithelium. Corneal findings include epithelial defects, marginal infiltrates, and an ulcerative keratitis that can progress to perforation. A Gram stain can be performed, but NAAT or polymerase chain reaction (PCR) testing is highly sensitive in confirming gonococcal or *Chlamydia* infection.



FIGURE 2.8 ■ Bacterial Conjunctivitis. Mucopurulent discharge, conjunctival injection, and lid swelling in a 10-year-old with *H influenzae* conjunctivitis. (Photo contributor: Frank Birinyi, MD.)

Management and Disposition

Encourage frequent hand washing and warm moist compresses. Empiric

antibiotic choices include polymyxin/trimethoprim drops and erythromycin ophthalmic ointment. Other options include bacitracin, sulfacetamide, or polymyxinbacitracin ointments or fluoroquinolone or azithromycin drops. Improvement should be noted in 1 to 2 days. Patients who do not improve should be referred to an ophthalmologist.



FIGURE 2.9 ■ Bacterial Conjunctivitis. Mucopurulent discharge and conjunctival injection in an adult with conjunctivitis. (Photo contributor: Lawrence E. Heiskell, MD.)

Fluoroquinolones should be prescribed for contact lens wearers (and keratitis ruled out) due to concern for *Pseudo-monas* infection. Hyperacute bacterial conjunctivitis requires immediate ophthalmologic consultation and hospitalization.

Pearls

1. Conjunctivitis is a common presentation of the red eye and should be considered after more serious causes, such as acute angle-closure glaucoma (ACG), iritis, and infectious keratitis, have been ruled out.
2. Red flags include a significantly decreased visual acuity, ciliary flush, corneal opacity, and rapid progression.
3. Worsening symptoms during topical treatment with Neosporin or a sulfonamide suggest a contact allergic reaction.
4. *N gonorrhoeae* conjunctivitis must be considered in the sexually active adult with a prominent, thick, copious discharge. Urethritis is usually present.
5. Fluoroquinolones should be prescribed for contact lens wearers because of

concern for *Pseudomonas* infection in these patients.

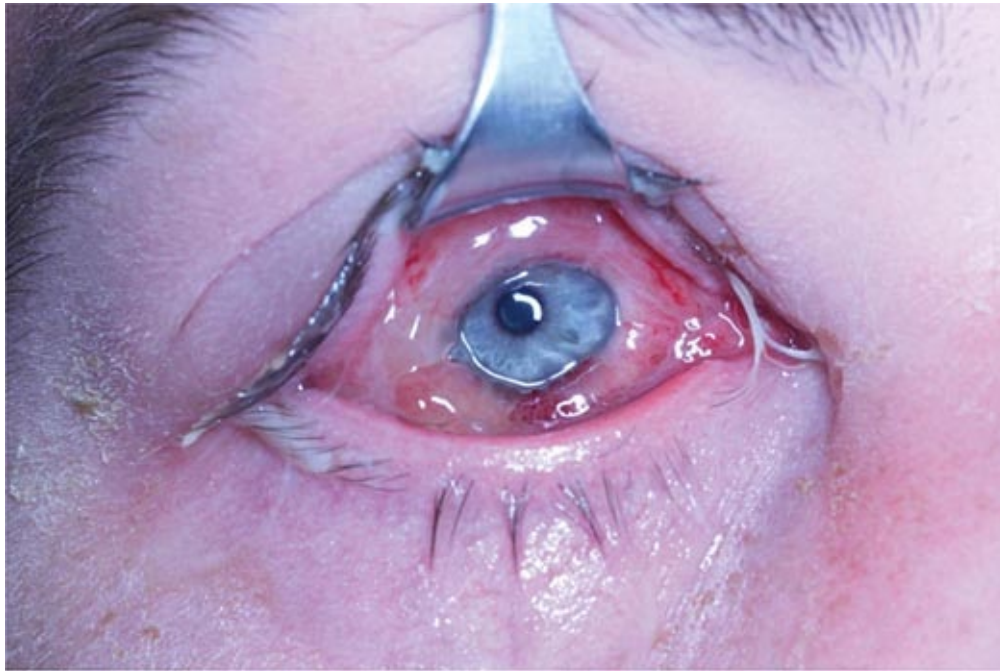


FIGURE 2.10 ■ Gonococcal Conjunctivitis. Right eye chemosis, purulent discharge, and pain in young adult male with urethritis. (Photo contributor: Catherine Burger, MD.)

VIRAL CONJUNCTIVITIS

Clinical Summary

Viral conjunctivitis is a common presentation of the red eye. Findings are mild and include a thin watery discharge, crusting in the morning, burning or irritation, conjunctival injection (typically diffuse), and lid edema. The tarsal conjunctiva may appear bumpy secondary to hyperplastic lymphoid tissue (follicles). Preauricular adenopathy may be present. The visual acuity is normal. The infection usually begins in one eye, but both eyes usually become involved due to autoinoculation. There are few to no systemic complaints. Adenovirus is the most common virus. A point-of-care test now available may help clinicians to avoid empiric antibiotic therapy.

Epidemic keratoconjunctivitis (EKC) is a severe and highly contagious adenovirus infection that also involves the cornea. Additional features may include foreign-body sensation, photo-phobia, and pseudomembranes overlying the palpebral conjunctiva. By the 8th day, a painful punctate keratitis that stains

with fluorescein may develop; by the end of the 2nd week, these are replaced by white macular subepithelial infiltrates located in the central cornea (that no longer stain). These may cause a decrease in visual acuity, but eventually resolve spontaneously.

Pharyngoconjunctival fever, usually caused by adenovirus type 3, is highly contagious and should be considered if there is associated upper respiratory tract infection and fever.

Management and Disposition

Viral conjunctivitis is self-limited and usually mild. Warm or cool compresses may be helpful. Careful hand washing by patient and staff is important. Over-the-counter topical antihistamines (pheniramine/naphazoline ophthalmic) may provide symptomatic relief. The eye irritation and discharge should improve after 5 to 7 days, but may take 2 to 3 weeks for complete resolution of all symptoms. Antivirals and antibiotics are ineffective. Patients with keratitis should follow up with an ophthalmologist.

Pearls

1. A nonspecific adenovirus conjunctivitis will improve after 5 to 7 days and resolve in 10 to 14 days, but a virulent adeno-virus causing EKC will peak in 5 to 7 days and may last 3 to 4 weeks.
2. Consider and rule out serious causes of red eye (acute ACG, iritis).
3. Frequent hand washing and the use of separate linens are advised for patients and family members.
4. Fastidious hand and equipment hygiene is necessary to prevent nosocomial transmission, as adenovirus can be recovered for extended periods of time from these surfaces.



FIGURE 2.11 ■ Viral Conjunctivitis. Note the characteristic asymmetric conjunctival injection. Symptoms first developed in the left eye, with symptoms spreading to the other eye a few days later. A thin watery discharge is also seen. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 2.12 ■ Epidemic Keratoconjunctivitis (EKC). Diffuse injection of the bulbar and palpebral conjunctiva is seen in addition to a follicular reaction of the palpebral conjunctiva. (Photo contributor: Kevin J. Knoop, MD, MS.)

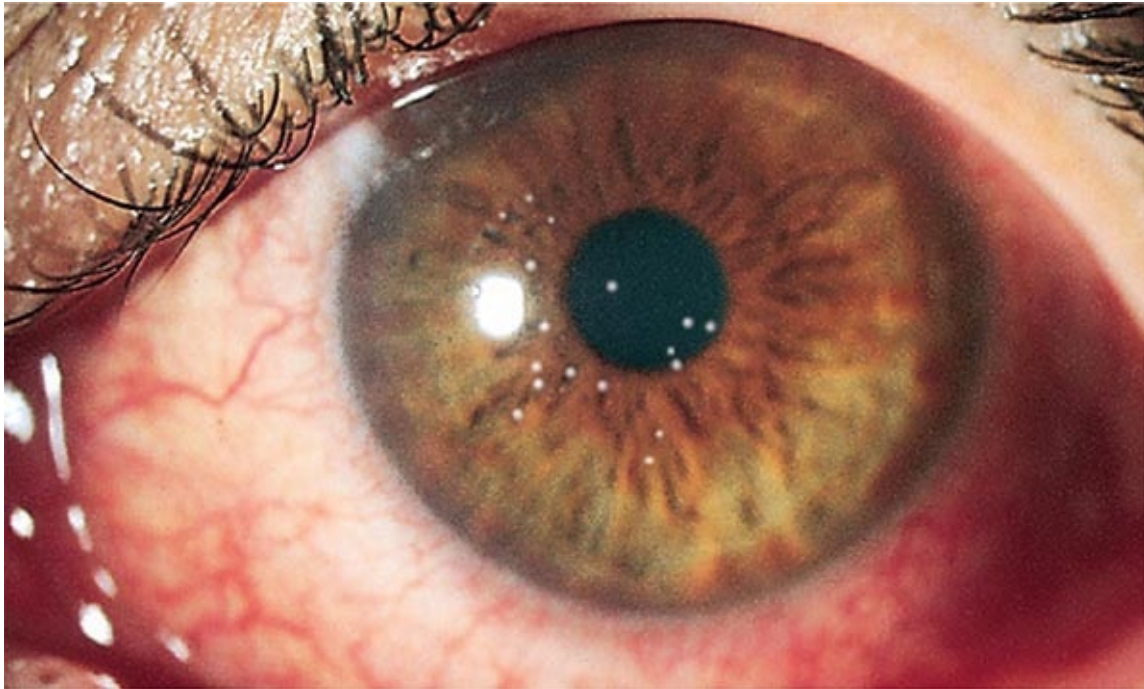


FIGURE 2.13 ■ Subepithelial Infiltrates (EKC). These usually develop in the second week. (Photo contributor: Katrina C. Santos Vongsy.)

ALLERGIC CONJUNCTIVITIS

Clinical Summary

Allergic conjunctivitis is a condition whereby airborne allergens precipitate type 1 IgE-mediated hypersensitivity reactions in the conjunctiva. Allergens include pollens, animal dander, mites, mold, and dust. Approximately 50% of patients have a personal or family history of allergic conditions such as atopy, eczema, asthma, and allergic rhinitis.

Itching is the hallmark symptom. Associated clinical features include conjunctival injection and edema, burning, discharge (clear, white, or mucopurulent), chemosis, and eyelid redness and swelling. Small papillae may be seen on the tarsal conjunctiva.

Vernal conjunctivitis is a rare but serious form of allergic conjunctivitis as it may lead to corneal scarring and subsequent vision loss if not treated. The

highest incidence is seen in the arid areas of the Middle East and North Africa secondary to wind and dust storms. Symptoms are similar to allergic conjunctivitis, but are more intense. Presentation is typically during childhood. Itching is severe and a vigorous knuckle rubbing is a typical observation. Giant, raised, pleomorphic papillae (“cobblestones”) seen over the upper tarsal plate are pathognomonic.

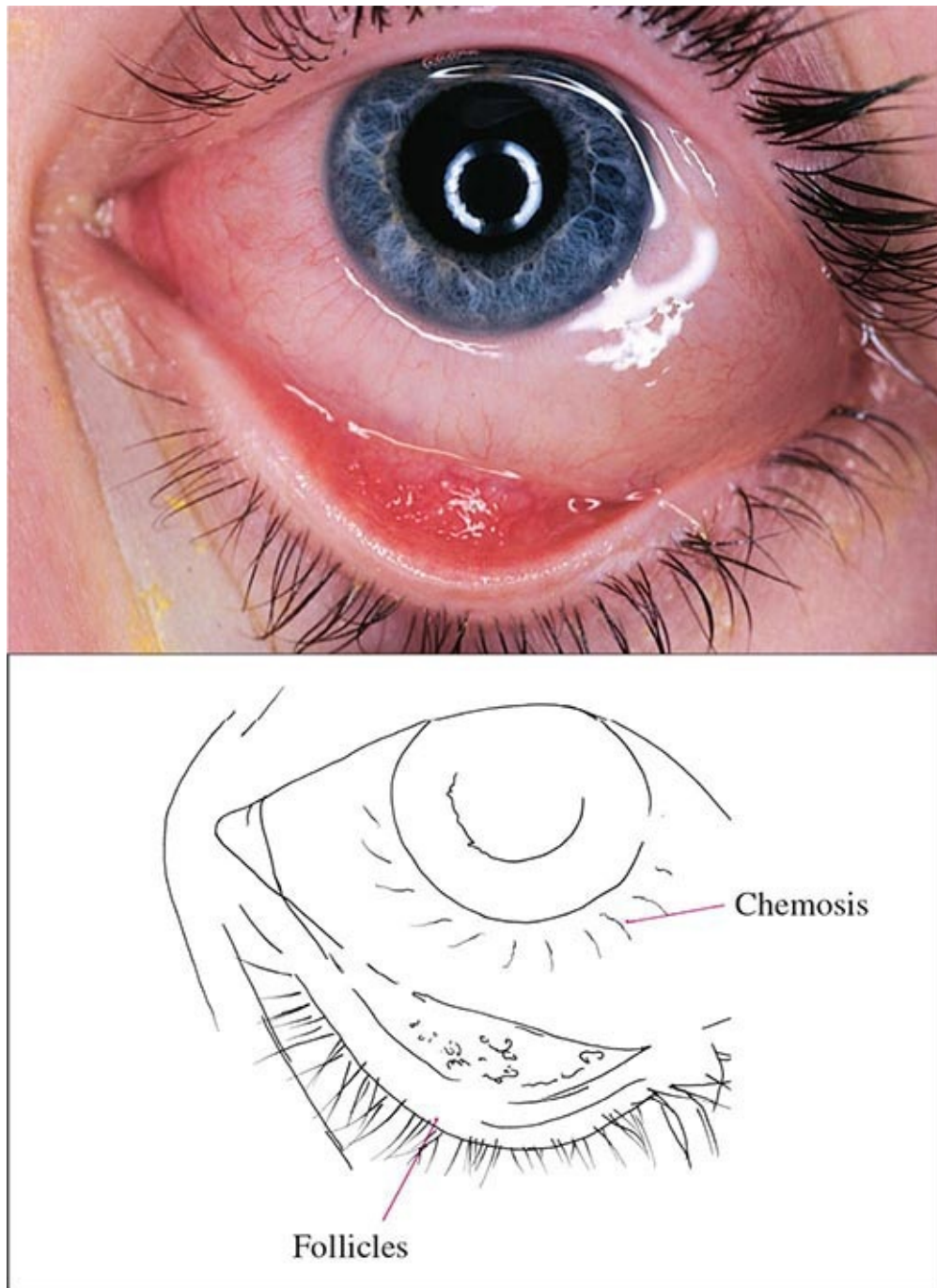


FIGURE 2.14 ■ Allergic Conjunctivitis. Conjunctival injection, chemosis, and a follicular response in the

inferior palpebral conjunctiva are seen in this patient with allergic conjunctivitis secondary to cat fur. (Photo contributor: Timothy D. McGuirk, DO.)

Management and Disposition

The initial approach is to eliminate the allergen. Avoiding animal dander, using air conditioners with appropriate filters, and limiting time outdoors will improve the condition. Topical tear substitutes are effective to dilute or wash away the allergen. H1 antihistamine–vasoconstrictor combinations such as over-the-counter pheniramine/naphazoline ophthalmic are recommended to relieve mild itching and redness. For more severe or frequent attacks, olopatadine, an antihistamine with mast cell–stabilizing properties, is more effective. Contact lens wearers should avoid use during allergic conjunctivitis flares. Mild topical steroids are an option only after consultation with an ophthalmologist.

Options for vernal conjunctivitis include olopatadine, mast cell stabilizers such as cromolyn, antihistamines, and cold compresses. Topical cyclosporine and allergen immunotherapy may be useful in resistant cases.

Pearls

1. Itching is the hallmark symptom of allergic conjunctivitis.
2. Itching is not common in nonallergic conjunctivitis.
3. Symptoms are usually bilateral.
4. Pheniramine/naphazoline ophthalmic and olopatadine are effective in treating allergic conjunctivitis.
5. Topical steroids may be considered for refractory symptoms and prescribed under the supervision of an ophthalmologist since complications of use include glaucoma, cataract formation, secondary infection, and corneal perforation.

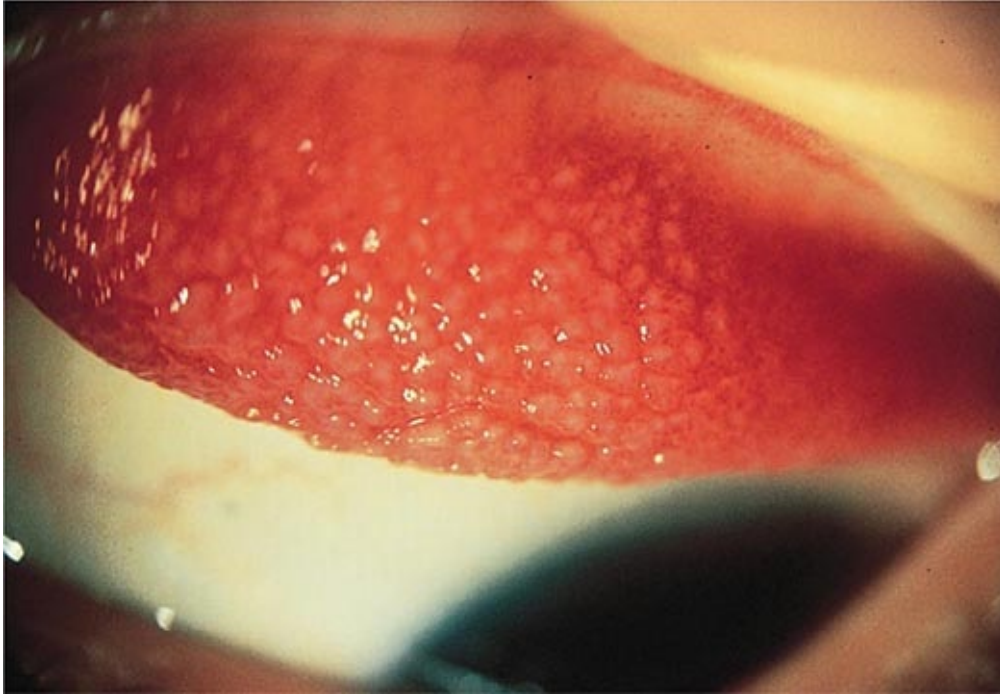


FIGURE 2.15 ■ Vernal Conjunctivitis. The tarsal conjunctiva demonstrates giant papillae and a cobblestone appearance pathognomonic for vernal conjunctivitis. (Photo contributor: William Beck, CRA.)

IRRITANT/CHEMICAL CONJUNCTIVITIS

Clinical Summary

Irritant/chemical conjunctivitis results from exposure of the conjunctiva to toxic agents that results in chemosis, edema, and hyperemia. These findings can be very similar to allergic conjunctivitis; therefore, history of exposure to an offending agent is helpful in making the diagnosis. Common exposures are smoke from fires, swimming pool chlorine, household chemicals, and cosmetics.

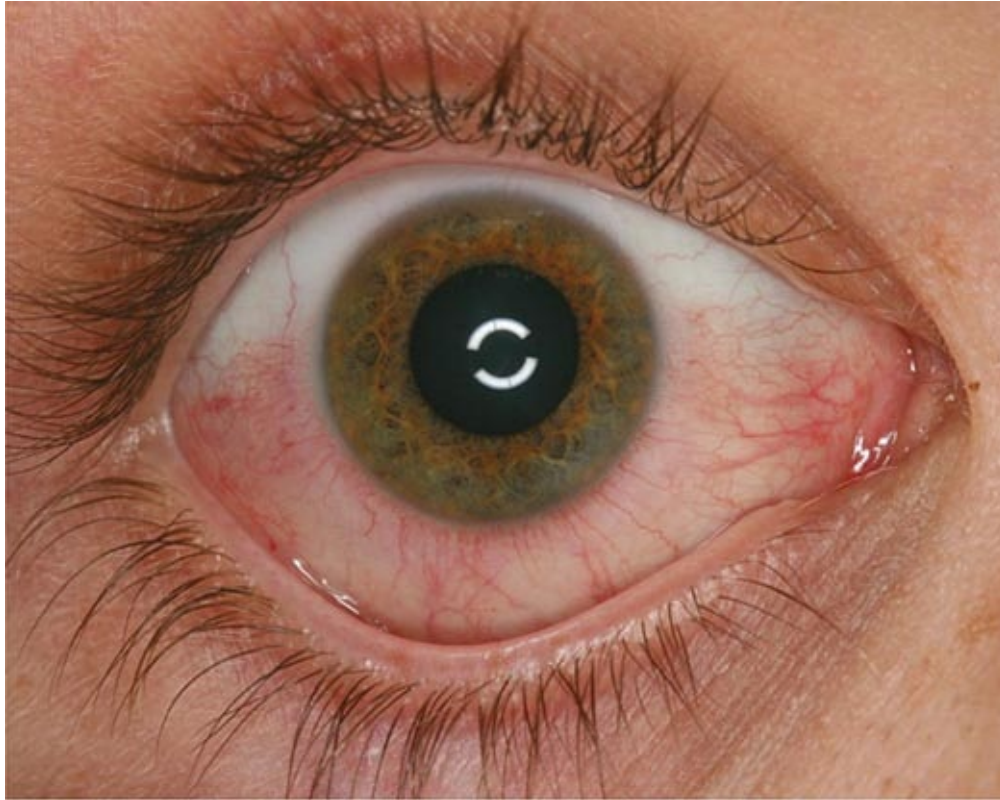


FIGURE 2.16 ■ Chemical Conjunctivitis. Typical exposure pattern conjunctivitis (upper conjunctiva spared) after laundry detergent exposure. (Photo contributor: Kevin J. Knoop, MD, MS.)

Patients will typically present with burning in the eyes. A mucous discharge may also be present with a papillary reaction of the palpebral conjunctiva. Eyelid findings may also be present with swelling, thickening, or excoriation marks due to resultant itching. Typical cases will demonstrate punctate epithelial staining with fluorescein application.

Management and Disposition

Immediately after an irritant exposure, initiate eye irrigation to remove any remaining chemical. Patients should immediately stop use of any cosmetics or topical medications that may be the source of irritation. Most cases can be treated with cold compresses, artificial tears, or lubricating eye drops or ointments to soothe ocular irritation. Most cases resolve with expectant management.

Pearls

1. Nasal cannula tubing can be connected to a bag of normal saline to irrigate chemical ocular exposures when a formal eye wash is not available. The nasal cannula prongs will direct flow directly into each eye.
2. A thorough history considering offending agents that are used with chronicity may lead to the diagnosis in patients in whom no obvious chemical irritant is identified.

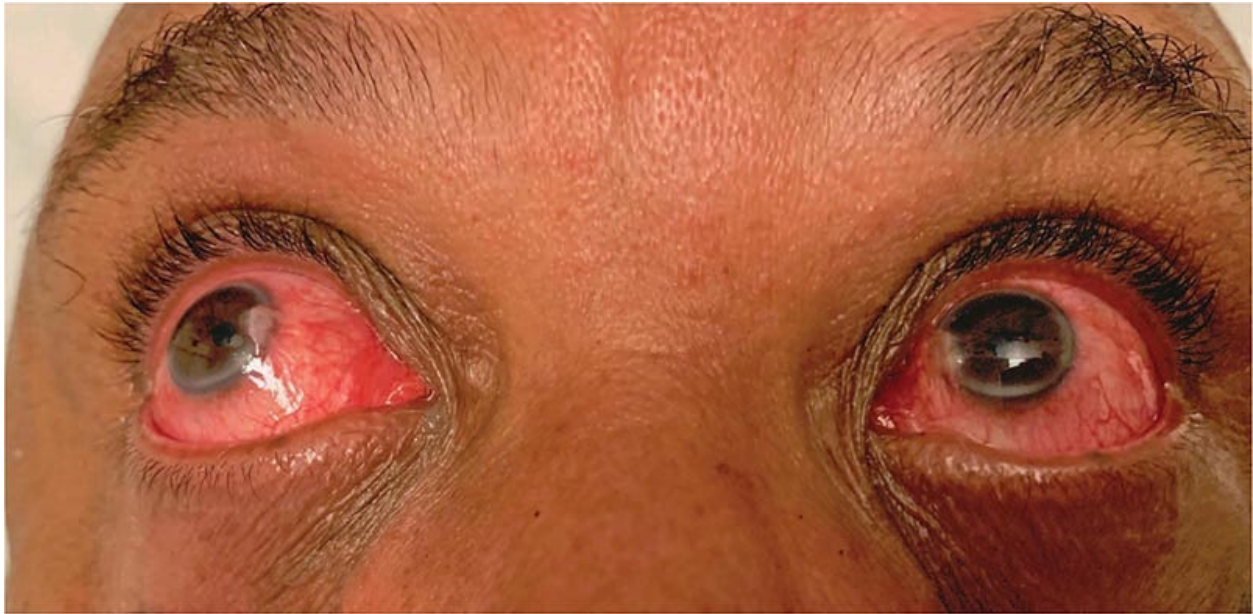


FIGURE 2.17 ■ Chemical Conjunctivitis. Patient with bilateral chemical conjunctivitis after tree sap exposure to both eyes. Bilateral pterygia are also seen. (Photo contributor: Denise Whitfield, MD, MBA.)

DACRYOCYSTITIS

Clinical Summary

Dacryocystitis is an inflammation of the lacrimal sac, positioned immediately distal to the canaliculi and proximal to the nasolacrimal duct. Inflammation is usually secondary to obstruction of the nasolacrimal duct. Clinical findings include swelling over the lacrimal sac, redness, tearing, eyelash matting and crusting, and conjunctival redness. Tears and mucopurulence may be expressed from the punctum when pressure is applied over the lacrimal sac. Complications include conjunctivitis and orbital or preseptal cellulitis.

Up to 20% of normal newborns have a closed nasolacrimal passage, and 90% spontaneously open within the 1st 6 months.

Management and Disposition

Ophthalmologic consultation is recommended. The most common organisms isolated in children are *S aureus*, *Staphylococcus epidermidis*, and α -hemolytic streptococci. Oral clindamycin for 7 to 10 days is recommended for outpatient management. Febrile and acutely ill patients require IV vancomycin in combination with a 3rd-generation cephalosporin.

Treatment of nasolacrimal duct obstruction is managed initially with downward lacrimal sac massage (“Crigler” massage) two to three times a day. Unresolved nasolacrimal duct obstruction requires lacrimal duct probing by the ophthalmologist.

Pearls

1. Nasolacrimal duct obstruction is the most common cause of persistent tearing and ocular discharge in children.
2. Swelling is localized to the extreme nasal aspect of the lower lid and may be confused with a hordeolum. Occasionally, conjunctival redness may be present.
3. Dacryocystitis may be confirmed by pressure on the lacrimal sac and the reflux of tears and purulent material from the punctum. The lacrimal sac and lacrimal fossa lie in the inferior medial aspect of the orbit, not on the side of the nose.
4. Urgent referral should be made for any signs of orbital cellulitis. These include proptosis, limitation of extraocular movements, and loss of vision.



FIGURE 2.18 ■ Dacryocystitis. Swelling and erythema over the medial lower lid and lacrimal sac developed in this 10-year-old patient with streptococcal pharyngitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

DACRYOADENITIS

Clinical Summary

Acute dacryoadenitis typically involves children and young adults with associated systemic infections such as gonorrhea, mumps, Epstein-Barr virus, and *Staphylococcus* species. Findings are localized to the outer one-third of the upper eyelid and include fullness or swelling, erythema, and tenderness. A characteristic “S”-shaped deformity with ptosis of the lid may be seen. In more advanced cases, proptosis, inferonasal globe displacement, ophthalmoparesis,

and diplopia may be present.

Chronic dacryoadenitis is more common, is seen in older patients, and is usually due to tumor or associated inflammatory disorders such as sarcoidosis, Sjögren syndrome, or IgG4-related diseases.

Management and Disposition

For acute dacryoadenitis, amoxicillin-clavulanate or IV ampicillinsulbactam is used, depending on the severity and patient's toxicity. In cases of dacryoadenitis due to mumps or Epstein-Barr virus, warm compresses are recommended.

Resolution occurs spontaneously. Patients should return to the ED for symptoms suggestive of orbital cellulitis such as decreased ocular motility or proptosis.

Treatment of chronic dacryoadenitis involves treatment of the underlying disorder.

Nonemergent ophthalmology follow-up is appropriate.

Pearls

1. Swelling is localized over the lateral one-third of the upper lid and imparts an "S"-shaped curve to the lid margin.
2. Acute dacryoadenitis is typically seen as a complication of mumps, with (bilateral) parotid swelling.
3. Chronic dacryoadenitis is more common and is seen in older patients. Malignancy should be considered.
4. IgG4-related disease should be considered in patients (particularly middle-aged and older men) with bilateral disease and either salivary gland enlargement or pancreatitis of unknown origin.
5. Urgent referral is recommended for patients with diplopia, limitation of the extraocular muscles, or reduction of vision.



FIGURE 2.19 ■ Dacryoadenitis. Unilateral localized swelling and chemosis are present laterally secondary to inflammation of the lacrimal gland. (Used with permission from the American Academy of Ophthalmology. *External Disease and Cornea: A Multimedia Collection*. San Francisco, 1994. © American Academy of Ophthalmology.)

PTERYGIUM/PINGUECULA

Clinical Summary

Pterygium (Greek, *pterygion*, meaning wing-like) is a benign proliferation of fibrovascular tissue. It usually originates in the nasal conjunctiva on the horizontal meridian of the limbus. It progressively encroaches onto the cornea and visual axis. Its appearance ranges from flat and white to thick, pink or red, and fibrovascular. Typically, it assumes a triangular appearance, with the apex directed toward the pupil. Predisposing factors include exposure to ultraviolet light, wind, and dust. Older individuals living in warmer areas with high levels of sunlight are more likely to develop pterygia.

Pterygia may be asymptomatic or become inflamed, causing mild symptoms of irritation and foreign-body sensation. Decreased visual acuity may result as the pterygium encroaches on the visual axis or if the lesion induces astigmatism.

Pinguecula (Latin, *pingueculus*, meaning fatty) is a common degenerative lesion of the bulbar conjunctiva, also arising in the horizontal meridian. It appears as a light brown or yellow-white amorphous, slightly raised conjunctival tissue adjacent to the limbus. It too may be asymptomatic or become episodically inflamed; however, it may enlarge and become a pterygium.

Management and Disposition

Mild disease can be treated with artificial tears or a topical vasoconstrictor (pheniramine/naphazoline ophthalmic). In more severe cases, consultation with an ophthalmologist is appropriate regarding topical steroids and topical nonsteroidal anti-inflammatory drugs (NSAIDs). Surgical excision by the ophthalmologist is indicated if the pterygium interferes with contact lens wear, encroaches significantly on the visual axis resulting in induced astigmatism or opacity, or restricts eye movement.

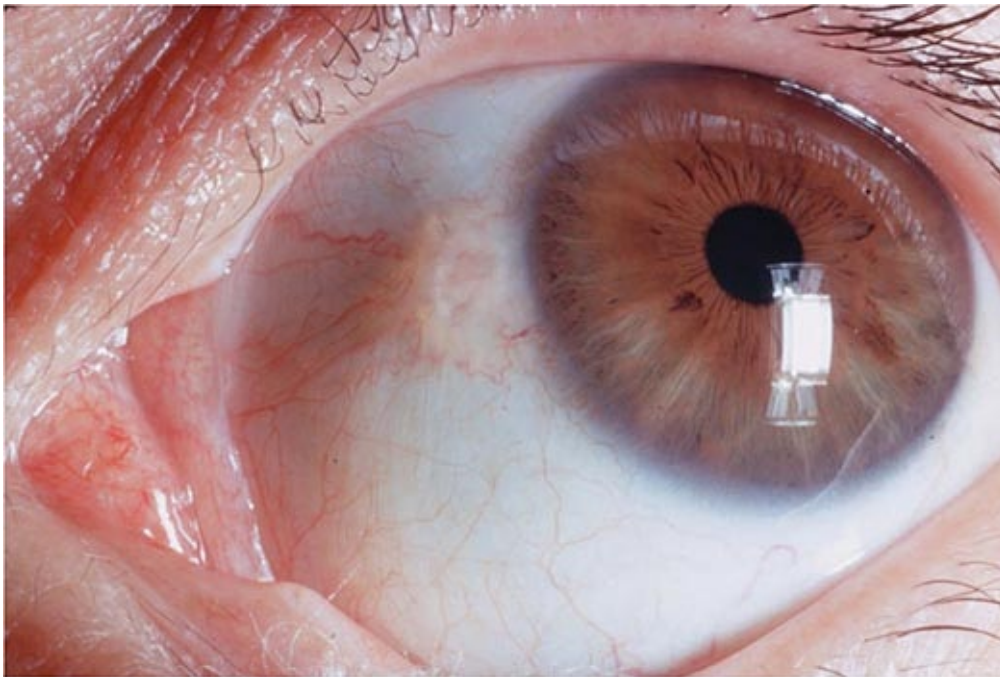


FIGURE 2.20 ■ Pinguecula. A small area of yellowish “heaped up” conjunctival tissue is seen adjacent to the nasal limbus. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

Pearls

1. Pterygia are more likely than pinguecula to be found on the nasal conjunctiva and bilateral.
2. Pterygia and pingueculae are found on the horizontal meridian. Conjunctival neoplasms often occur in axes other than the horizontal axis.
3. Following surgical excision, recurrence of pterygia is common.
4. Pterygium mimics include localized conjunctival neoplasia, conjunctivitis, and episcleritis.



FIGURE 2.21 ■ Pterygium. Pterygium appears as a raised vascular triangular area of bulbar conjunctiva that encroaches onto the cornea. (Photo contributors: Top: Rebecca Kasl, MD; Bottom: Andrew J. Hendershot, MD.)

CORNEAL HYDROPS

Clinical Summary

Corneal hydrops is the acute onset of corneal edema that results from a break in Descemet's membrane (basement membrane that lies between the corneal endothelium and the corneal substance). It is seen in patients with progressive worsening of keratoconus, a noninflammatory disorder of the cornea characterized by progressive thinning and cone-shaped protrusion, as well as patients with ocular disease processes with corneal distention. In corneal hydrops, severe corneal edema develops. Patients typically experience sudden photophobia and decreased visual acuity. Hydrops can resolve without treatment after several weeks to months, but patients may experience eye pain and decreased vision until the corneal edema improves. Hydrops can also lead to permanent corneal scarring or, rarely, perforation.

Management and Disposition

Evidence for effective treatment is minimal, but treatments include pressure patching, bandage contact lens use, topical steroids, and cycloplegics. Placement of gas or air in the anterior chamber can decrease the time to resolution of hydrops but does not change the incidence of long-term corneal scarring, vision deficits, or the need for corneal transplant.



FIGURE 2.22 ■ Corneal Hydrops. Patient with marked corneal edema consistent with acute corneal hydrops. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Patients with corneal hydrops typically present with acute-onset photophobia and decreased visual acuity. Corneal edema can be appreciated on slit-lamp examination.
2. Corneal hydrops is typically self-limiting but requires ophthalmologic consultation for follow-up as an outpatient.
3. Treatments have not been shown to change the clinical sequelae of corneal hydrops but may shorten or relieve symptoms.

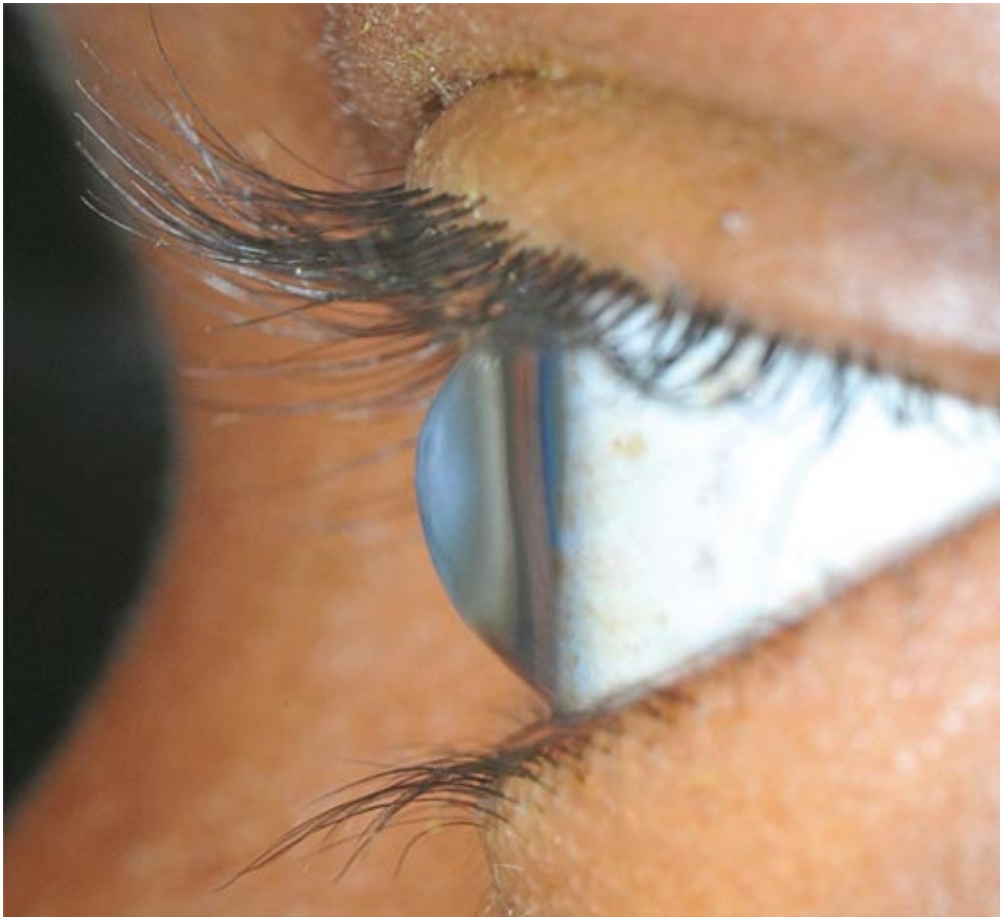


FIGURE 2.23 ■ Corneal Hydrops. Patient presenting with conical protrusion and edema seen in the lateral view. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 2.24 ■ Corneal Hydrops. Inferior conical protrusion and edema in left eye. Note the indentation (“V”-shaped) on the lower lid due to keratoconus (Munson sign). (Photo contributor: Lawrence B. Stack, MD.)

LEUKOCORIA/CATARACT

Clinical Summary

Leukocoria is an abnormal white pupillary reflex and one of the primary signs of retinoblastoma in a child, usually less than 3 years old. The “white pupil” is often noticed in photographs using a flash. Strabismus may also be a presenting sign. Meta-static disease usually occurs within 12 months of presentation, so prompt treatment is important. Survival with timely diagnosis and treatment is greater than 95%. A more common cause of leukocoria are cataracts, which form as a slow clouding of the lens due to various etiologies, leading to decrease in vision. They account for much of blindness worldwide, as well as one-third of visual impairment. Symptoms can vary and include blurry vision, faded colors, double vision, halos around lights, and difficulty with bright lights or seeing at night. The poor vision leads to increased risk of falling and depression. Etiologies of cataracts include aging (most common), trauma, radiation exposure, neonatal, genetics, iatrogenic, or following eye surgery. Most cataracts are diagnosed based on comprehensive eye exam, where slit lamp plays a key role.

Management and Disposition



FIGURE 2.25 ■ Leukocoria. Leukocoria (white pupil) is seen in this child with retinoblastoma. (Photo contributor: Lawrence B. Stack, MD.)

Urgent referral to an ophthalmologist is recommended for a child with leukocoria to rule out retinoblastoma and to assess for other causes. Incidental diagnosis of cataract requires no action in the ED. Decreasing exposure to ultraviolet B and quitting smoking are key for prevention or limiting worsening of cataracts, as well as possible oral supplementations. Once a cataract affects quality of life as a result of poor vision, outpatient surgery is the mainstay of treatment.



FIGURE 2.26 ■ Cataract. Bilateral congenital cataracts are seen in this child. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. A white pupillary reflex in a child is suggestive of retinoblastoma, whereas a blue-gray pupil is seen in congenital cataract.
2. Once a cataract is discovered, attempt to identify the etiology to slow progression and implement preventative interventions if possible.
3. In addition to performing a comprehensive examination in your ED (eg, visual acuity, slit lamp, pressures), assess how the cataract impacts quality of life, including risk of fall and depression. Available social support for the patient is important to consider.

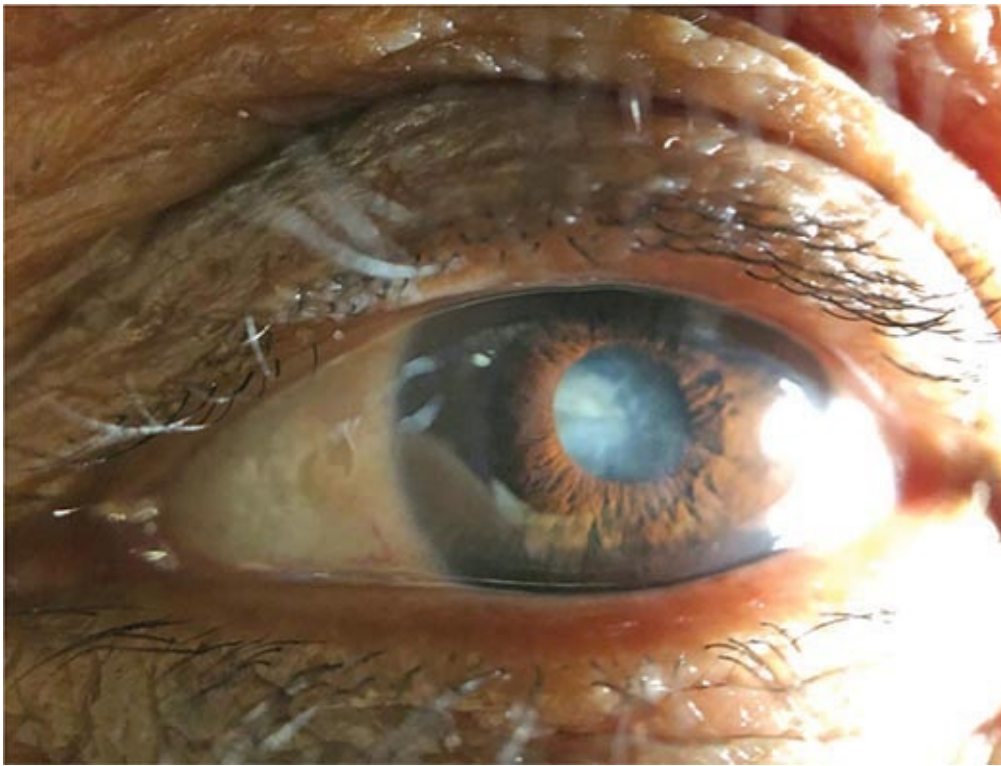


FIGURE 2.27 ■ Cataract. A white corneal opacity consistent with a cataract is demonstrated. (Photo contributor: Kevin J. Knoop, MD, MS.)

HORDEOLUM/CHALAZION

Clinical Summary

A hordeolum is a localized abscess involving the glands of the eyelid. An

external hordeolum (stye) involves an eyelash (cilia) follicle or the tear glands of the lid margin (glands of Zeis, Moll). An internal hordeolum involves the meibomian glands. *S aureus* is the most frequent isolate, although hordeola may be sterile. Clinical findings include focal swelling, edema, erythema, and tenderness.

A chalazion is a chronic localized inflammation that results from the obstruction of the meibomian glands. It may arise from a hordeolum. The lesion may point anteriorly (toward the skin of the eyelid) or posteriorly (toward the tarsal conjunctiva). It may become sufficiently large as to press on the globe and cause astigmatism. Pain and redness may be seen early in the course, but these may dissipate over time.

Management and Disposition

Hordeola can be treated with warm compresses. The clinical course of a hordeolum is usually self-limited, with spontaneous drainage and resolution in 5 to 7 days. Topical antibiotics are not helpful. Systemic antibiotics are unnecessary unless there is concurrent preseptal cellulitis. If the mass persists or is large (distorting vision), ophthalmology referral is recommended for incision and curettage or intralesional corticosteroid injection.

Frequent warm compresses applied to chalazia expedite spontaneous drainage. Most chalazia spontaneously clear after several weeks. Antibiotics are not indicated since chalazia result from a granulomatous process. Recalcitrant lesions should be referred to the ophthalmologist for incision and curettage or glucocorticoid injection.

Pearls

1. Uncomplicated hordeola and chalazia can be treated with warm compresses and generally resolve spontaneously.
2. Patients with rosacea or marginal blepharitis (a chronic low-grade inflammation of the lid margins with crusts around the lashes) have frequent hordeola.
3. Older patients with recurrent chalazia should be referred to the ophthalmologist to rule out malignancy.

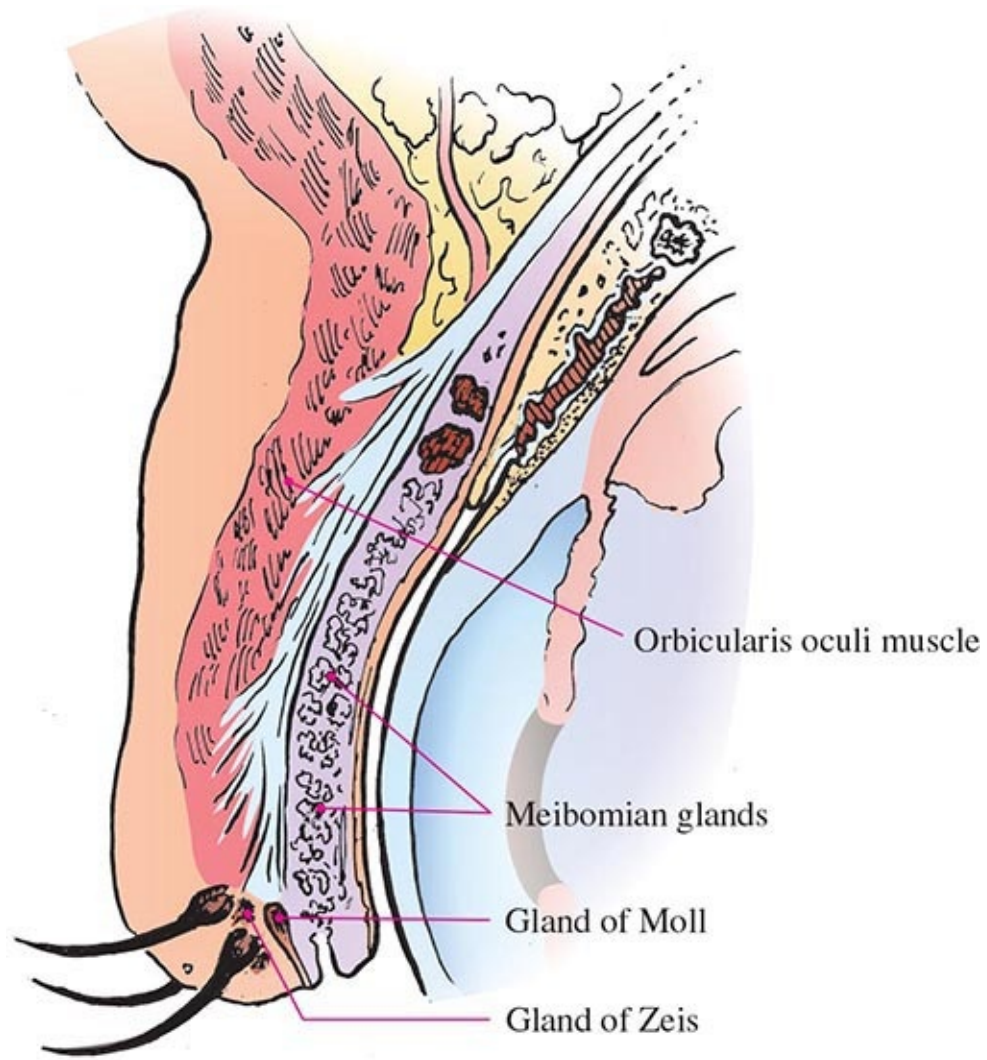


FIGURE 2.28 ■ Eyelid Anatomy. Anatomic structures related to eyelid pathology.

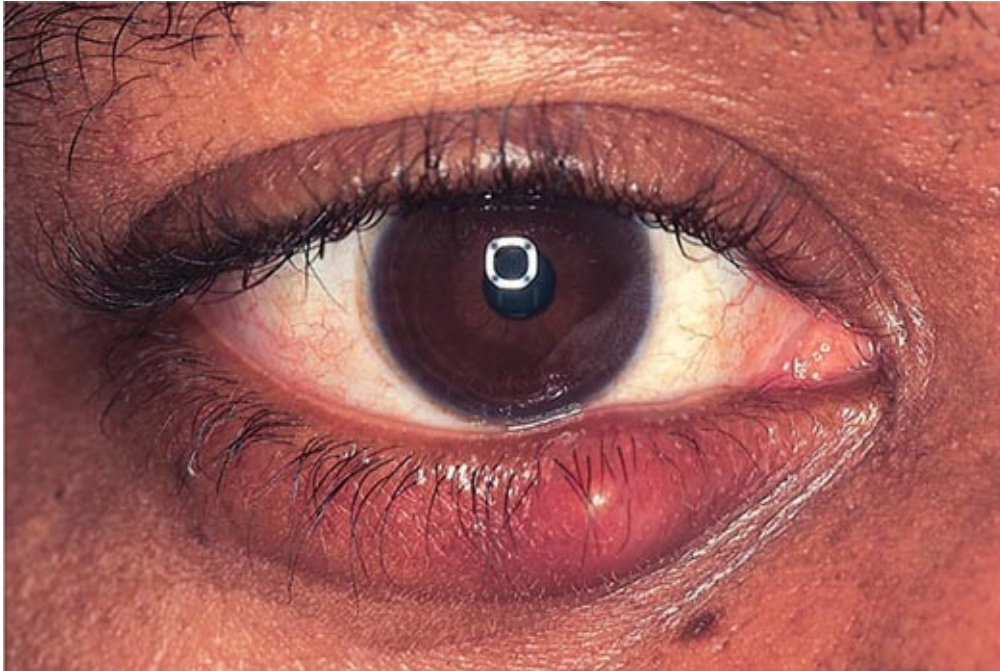


FIGURE 2.29 ■ Hordeolum. Focal swelling and erythema at the lid margin are seen in this hordeolum. (Photo contributor: Frank Birinyi, MD.)



FIGURE 2.30 ■ Chalazion. This chalazion shows painful nodular focal swelling and erythema from meibomian cyst formation. (Photo contributor: Frank Birinyi, MD.)



FIGURE 2.31 ■ Chalazion. Two months of focal pain and swelling of the lateral upper lid. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 2.32 ■ Chalazion. A nontender bipartite chalazion is shown. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 2.33 ■ Blepharitis. The eyelid margins are inflamed and erythematous. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 2.34 ■ Chalazion. This lower lid chalazion was seen only with lid eversion. (Photo contributor: James Dahle, MD.)

SCLERITIS

Clinical Summary

Scleritis is painful, destructive, and potentially blinding. The pain is constant and boring and may radiate to the face and peri-orbital region. Associated features include tearing, photophobia, globe tenderness to palpation, and painful ocular movement.

The conjunctival vessels are injected. The eye itself may be intensely red with a violaceous or purple hue secondary to engorgement of the deep vessels of the episclera and scleral thinning. These deep vessels do not move when the overlying conjunctiva is moved with a cotton-tipped applicator, nor do they blanch with topical phenylephrine. On slit-lamp microscopy, the episcleral vessels are displaced outward by scleral edema. Corneal involvement, iritis (with cells and flare in the anterior chamber), and decreased visual acuity may accompany scleritis. An associated scleritis may occur with severe infectious keratitis. Primary infectious scleritis is rare. In either instance, topical and systemic antibiotics are indicated after appropriate cultures are obtained.

In up to 50% of cases, scleritis is associated with underlying autoimmune or infectious systemic disease, with rheumatoid arthritis being the most common. It occurs more frequently in women and in the 4th to 6th decades of life.

Management and Disposition

Ophthalmology consultation is required. Treatment varies according to underlying disease (if present) and can involve NSAID therapy, glucocorticoids, and immunosuppressive medications. Rheumatology consultation by the ophthalmologist is often required for optimal management in patients with underlying autoimmune disorders.

Pearls

1. Scleritis is associated with a systemic disease in approximately 50% of cases, most commonly rheumatoid arthritis.
2. Most cases of scleritis involve the anterior portion (anterior to the insertion of the medial and lateral rectus muscles).
3. Pain is exacerbated with ocular movements because the extraocular muscles insert into the sclera itself.
4. Anterior uveitis can occur in up to 40% of patients because the uvea is immediately adjacent to the sclera.

5. Check intraocular pressure (IOP) to rule out acute ACG as another cause of painful red eye.

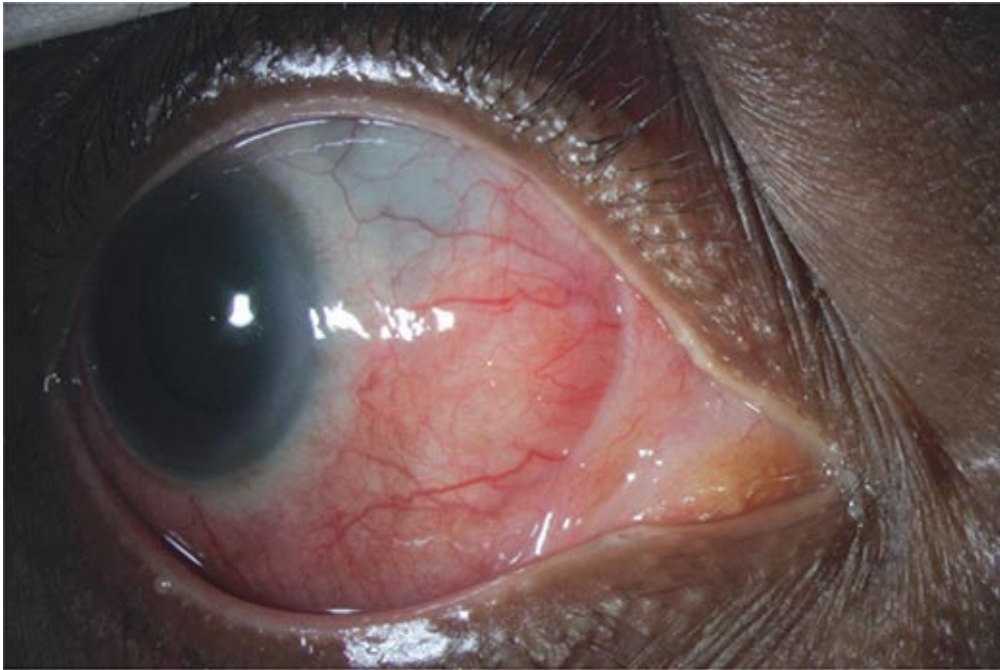


FIGURE 2.35 ■ Scleritis. A generalized vascular injection is seen. A bluish hue is also seen superiorly due to scleral thinning. The vessels do not move when the overlying conjunctiva is moved with a cotton-tipped applicator. (Photo contributor: Jeffrey Goshe, MD.)



FIGURE 2.36 ■ Scleritis. A 55-year-old female with scleritis of the left eye associated with rheumatoid arthritis. Note dilation of the deep conjunctival and episcleral vessels and blue hue suggesting thinning of the sclera temporally. (Used with permission from Brice Critser, CRA, The University of Iowa and



FIGURE 2.37 ■ Sectorial Scleritis. Deep-boring pain experienced by this patient distinguishes this segmental area of erythema from episcleritis. (Photo contributor: Kevin J. Knoop, MD, MS.)

EPISCLERITIS

Clinical Summary

Episcleritis is a common and benign inflammation of the episclera, typically affecting young and middle-aged adults. Seventy percent of cases occur in females. The episclera lies just beneath the bulbar conjunctiva. Its vessels are large, run in a radial direction, and can be seen beneath the overlying conjunctiva. Episcleral and conjunctival vessels blanch with the use of topical 5% phenylephrine drops, unlike deep episcleral vessels.



FIGURE 2.38 ■ Episcleritis. A localized area of hyperemia is seen involving the temporal sclera. (Photo contributor: Robert Trieff, MD.)

Patients may complain of foreign-body sensation, mild tenderness, irritation, mild photophobia, and excessive lacrimation. Pain is unusual but can occur, particularly in chronic cases. One-half of cases are bilateral. Eye findings are notable for a localized pink or bright red conjunctival injection, with involvement of the vessels in the superficial episcleral vascular plexus. Visual acuity is normal.

Episcleritis is usually an isolated condition, although it may be associated with a number of systemic diseases, including rheumatoid arthritis, inflammatory bowel disease, lupus, and vasculitis.

Management and Disposition

For many patients, the condition is self-limited and will resolve within 3 weeks, with or without treatment. For mild cases, use over-the-counter artificial tears. Topical or oral NSAIDs may also be used. For those with recurrent or recalcitrant lesions, referral to the ophthalmologist is indicated.

Pearls

1. It is important to differentiate episcleritis from sight-threatening scleritis. Episcleritis presents with only mild pain and bright red episcleral vessels, which will blanch with topical phenylephrine.
2. Conjunctivitis is a more common cause of red eye, and symptoms usually include morning crusting. Injection that is localized rather than diffuse is more likely to be episcleritis.

ACUTE ANGLE-CLOSURE GLAUCOMA

Clinical Summary

Acute angle-closure glaucoma (ACG) is secondary to narrowing or closure of the anterior chamber angle, resulting in increased IOP, with subsequent damage to the optic nerve. Normally, aqueous humor drains out of the anterior chamber via Schlemm canal in the anterior chamber angle. In ACG, this flow is impeded, and the IOP rises from a normal range of 10 to 21 mm Hg to 50 mm Hg or higher.

ACG presents as an acutely inflamed eye. Nausea and vomiting are common and may be the presenting complaints. Eye pain and headache vary in severity. As the IOP reaches the 50 to 60 mm Hg range, fluid is forced into the cornea, resulting in corneal edema. Patients report blurred vision and rainbow-colored halos around lights. Clinical findings include tearing, perilimbal injection (“ciliary flush”), a cloudy (“steamy”) cornea, a nonreactive mid-dilated pupil, anterior chamber inflammation, and an increased IOP. Using a penlight or slit-lamp microscopy, the anterior chamber may appear shallow.

Management and Disposition

ACG is an emergency that requires ophthalmology consultation, and treatment is directed at reducing the IOP. Aqueous outflow is increased by topical myotics (pilocarpine) that pull the peripheral iris taut away from the anterior chamber angle. Decreased production of aqueous is accomplished with topical β -blockers (timolol), an α -adrenergic agonist (apraclonidine), or a carbonic anhydrase inhibitor (acetazolamide or methazolamide systemically, dorzolamide

topically). Osmotic agents reduce the aqueous volume within the eye and include glycerol, oral isosorbide, and IV mannitol. Latanoprost, a prostaglandin derivative, increases aqueous outflow through nontrabecular meshwork pathways.

The definitive treatment of ACG is laser peripheral iridectomy, done usually after the IOP normalizes.

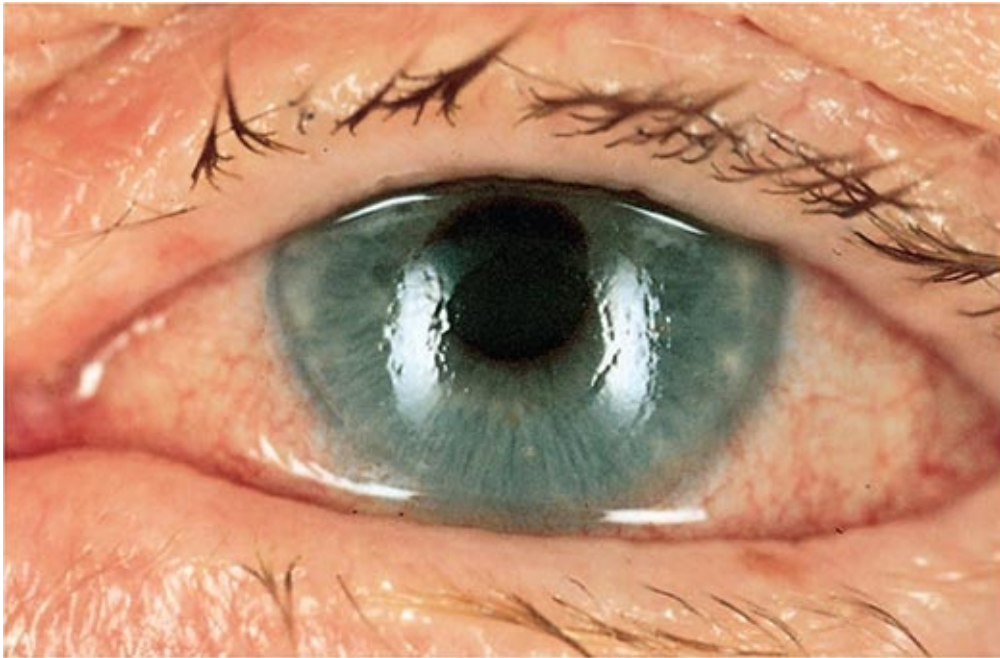


FIGURE 2.39 ■ Acute Angle-Closure Glaucoma. The cornea is edematous, manifest by the indistinctness of the iris markings and the irregular corneal light reflex. Conjunctival hyperemia is also present. (Photo contributor: Kevin J. Knoop, MD, MS.)

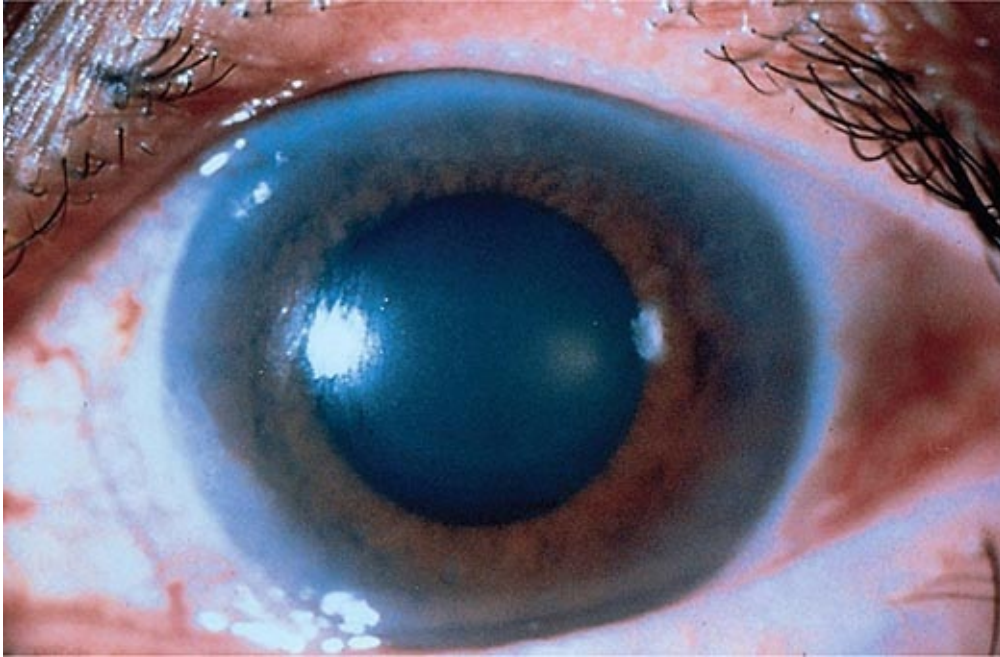


FIGURE 2.40 ■ Acute Angle-Closure Glaucoma. Note the cloudy or “steamy” appearance of the cornea and the midposition pupil. Conjunctival hyperemia is not as evident. (Photo contributor: Gary Tanner, MD.)

Pearls

1. Vision loss and blindness can occur during the course of the attack (hours to days). ACG is a true ophthalmologic emergency.
2. Medicines with anticholinergic effects are capable of inducing pupillary dilation, which might provoke an angle-closure attack. These include over-the-counter decongestants, motion sickness medications, antipsychotics, and antidepressants.
3. ACG often occurs in the evening, when lower light levels cause mydriasis.
4. ACG patients may easily be misdiagnosed initially as having a migraine headache or a CNS catastrophe because they may present with severe headache and vomiting.
5. The elevated IOP in acute ACG can be reduced pharmacologically by three mechanisms: 1st, by opening the closed angle with myotics; 2nd, by reducing aqueous formation with β -blockers, α -agonists, or carbonic anhydrase inhibitors; and 3rd, by reducing the aqueous volume within the eye by osmotic agents.

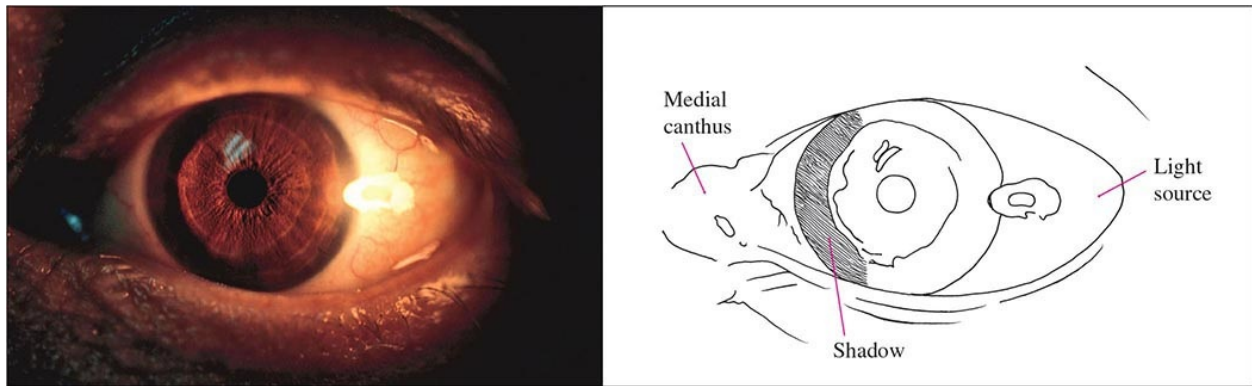


FIGURE 2.41 ■ Penlight Test. A penlight, held laterally and directed nasally, projects a shadow on the nasal side of an iris with a shallow anterior chamber. This patient presented with acute angle-closure glaucoma. (Photo contributor: Alan B. Storrow, MD.)

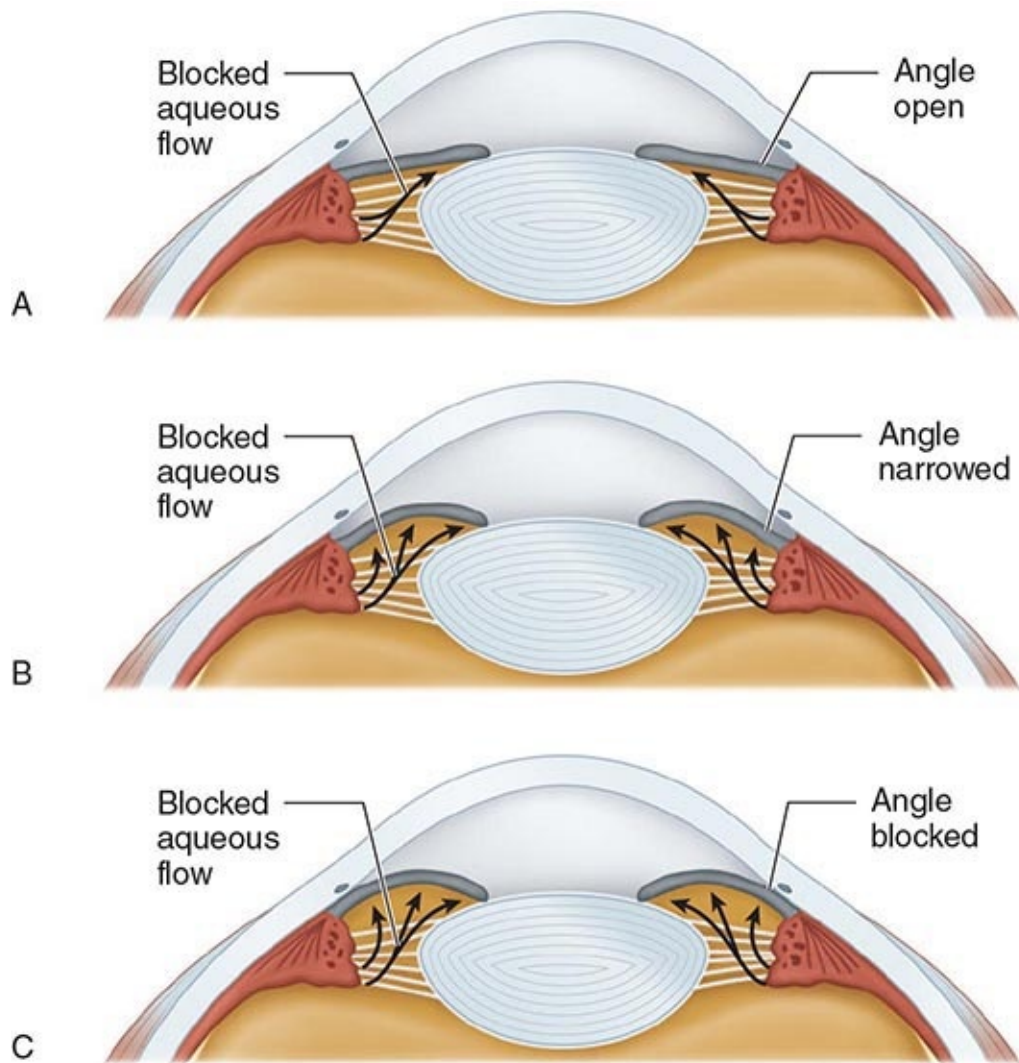


FIGURE 2.42 ■ Iris Bombé. (A) A physiologic block normally occurs where the posterior surface of the iris touches the lens. (B) When this block is increased, the iris as a result bows forward (iris bombé) and the angle is narrowed and pressure increases. (C) As pressure increases, the angle becomes blocked.

ANTERIOR UVEITIS (IRITIS)

Clinical Summary

The uvea is the middle layer of the eye. Inflammation of the anterior portion (iris and ciliary body) is called anterior uveitis or iritis. The posterior portion includes the choroid. Approximately half of cases of iritis are associated with systemic disease. These include inflammatory disorders (rheumatoid arthritis, Behçet disease, sarcoid), HLA-B27–associated conditions (ankylosing spondylitis, inflammatory bowel disease, Reiter syndrome), and infectious causes (zoster, tuberculosis, toxoplasmosis, AIDS).

Clinical features include conjunctival hyperemia, hyperemic perilimbal vessels (“ciliary flush”), miosis, decreased visual acuity, photophobia, tearing, and pain. Leukocytes in the anterior chamber are characteristic. The slit lamp may demonstrate a hypopyon, cells, flare, and keratic precipitates (agglutinated inflammatory cells adherent to the posterior corneal endothelium). These appear either as fine gray-white deposits or as a large, flat, greasy-looking ones (“mutton fat”). The IOP may be decreased due to decreased aqueous production by the inflamed ciliary body or increased secondary to inflammatory debris within the trabeculae of the anterior chamber angle obstructing outflow.

Management and Disposition

The patient’s history forms the basis for the evaluation and laboratory testing and should focus on rheumatic illness, dermatologic problems, bowel disease, infectious exposures, and sexual history. Treatment is nonspecific. Topical cycloplegics and corticosteroids may be prescribed in conjunction with the ophthalmologist. Antibiotics are not usually prescribed.



FIGURE 2.43 ■ Uveitis. Conjunctival injection, miosis, and cloudy anterior chamber with discoloration of iris compared to other eye in patient with anterior uveitis four days after intraocular injection. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 2.44 ■ Anterior Uveitis. Atraumatic left eye pain, photophobia, and limbal injection, in a middle-aged male with inflammatory bowel disease. Miosis is also present, a hallmark of uveitis. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Iritis is usually associated with a miotic pupil.
2. When uveitis is associated with a systemic disorder, the associated condition

is usually evident. Common exceptions include sarcoidosis and syphilis. A chest x-ray looking for sarcoidosis and serologic testing for syphilis are reasonable.

3. Patients with recurrent uveitis should undergo workup for systemic inflammatory disease.
4. Topical analgesics do not significantly ameliorate the pain of anterior uveitis.
5. Consider sympathetic ophthalmia with unexplained uveitis and a history of eye trauma.

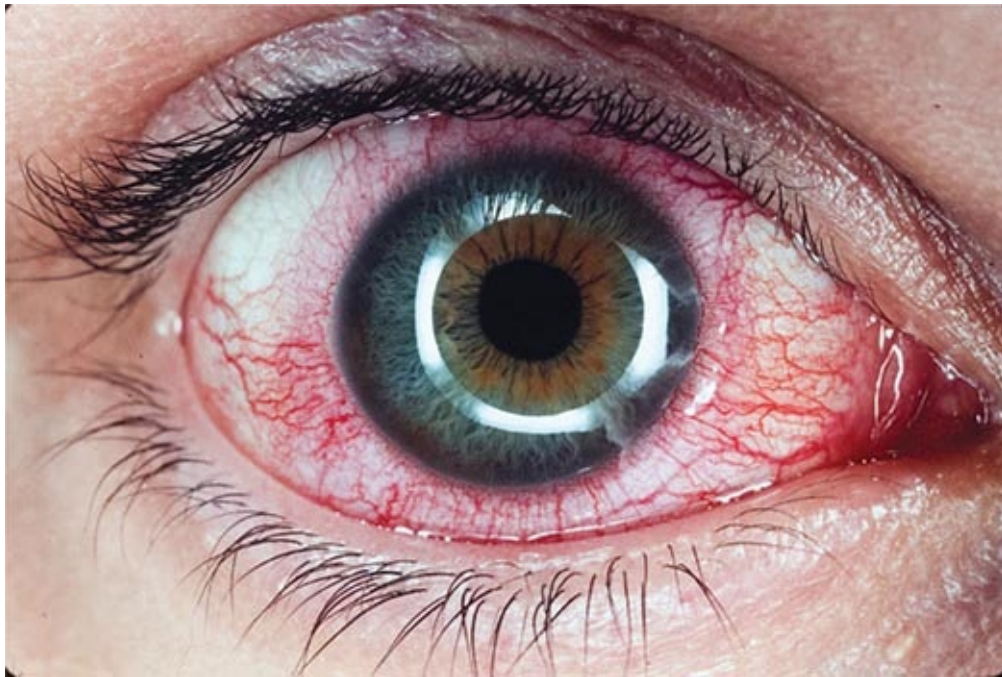


FIGURE 2.45 ■ Anterior Uveitis. Marked conjunctival injection and perilimbal hyperemia (“ciliary flush”) are seen in this patient with recurrent iritis. (Photo contributor: Frank Birinyi, MD.)



FIGURE 2.46 ■ Hypopyon. A thin layering of white blood cells is present in the inferior anterior chamber. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)



FIGURE 2.47 ■ Anterior Chamber Cells. Cells in the anterior chamber are a sign of inflammation or bleeding and appear similar to particles of dust in a sunbeam. They are best seen with a narrow slit-lamp beam directed obliquely across the anterior chamber. (Used with permission from Spalton DJ, Hitchings RA, Hunter PA, eds. *Atlas of Clinical Ophthalmology*. 2nd ed. London, UK: Mosby-Wolfe Limited; 1994.)

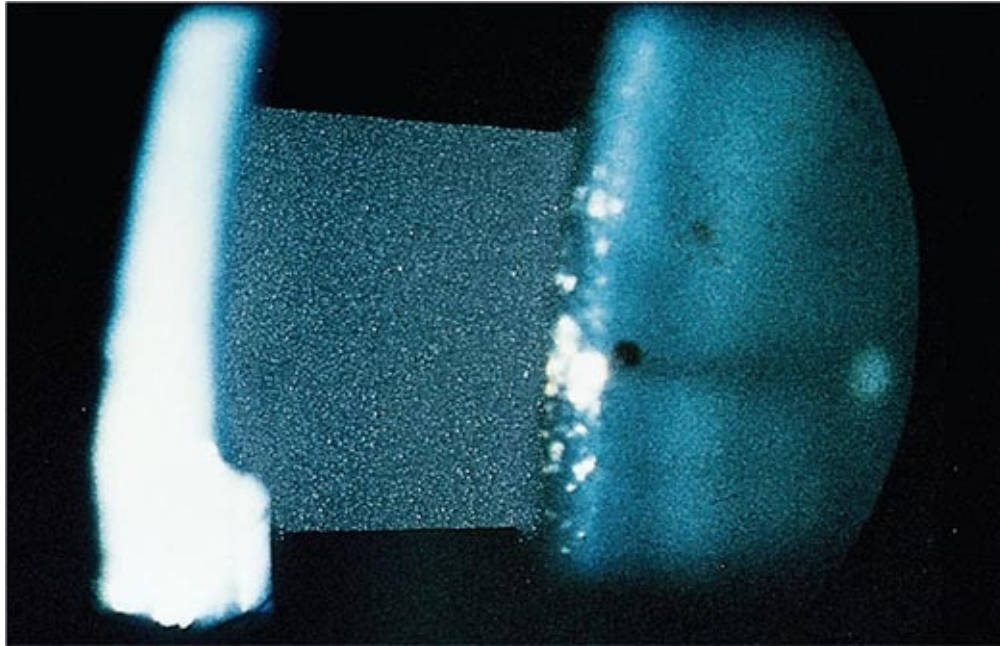


FIGURE 2.48 ■ Anterior Chamber Flare. Flare in the anterior chamber represents an elevated concentration of plasma proteins from inflamed, leaking intraocular blood vessels. Flare seen in a slit-lamp beam appears similar to a car headlight cutting through the fog. (Used with permission from Spalton DJ, Hitchings RA, Hunter PA, eds. *Atlas of Clinical Ophthalmology*, 2nd ed. London, UK: Mosby-Wolfe Limited; 1994.)

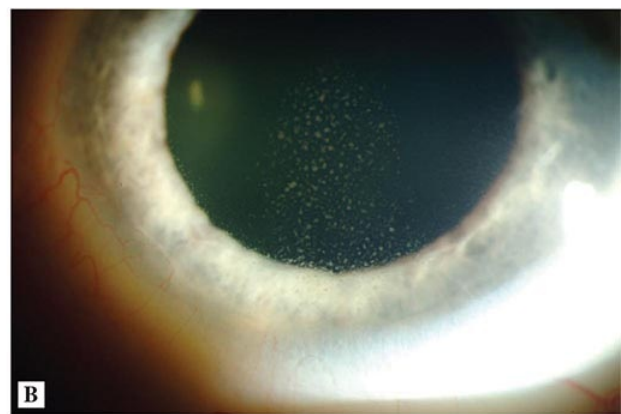
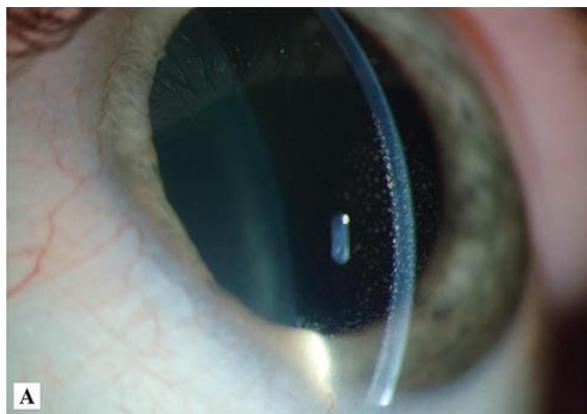


FIGURE 2.49 ■ Keratic Precipitates. Deposits of cells on the *endothelial* layer of the cornea are seen in these photographs with a slit-lamp beam (**A**), and under diffuse light (**B**). (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

ENDOPHTHALMITIS

Clinical Summary

Endophthalmitis is an infection of the globe. It is a vision-threatening medical emergency. The condition typically results from exogenous inoculation of bacteria or fungi following eye surgery or trauma. Less commonly, endogenous infection can occur and may result from the extension of a corneal infection. Patients with endophthalmitis will present with pain and decreased visual acuity. For postsurgical patients, the majority of cases will present within 1 week of the surgery.

On examination, conjunctival chemosis, hyperemia, and decreased visual acuity are noted. Some cases may present with hypopyon as sequelae of intraocular inflammation. The diagnosis of endophthalmitis is clinical.

Management and Disposition

An ophthalmologist should be consulted emergently for the management of endophthalmitis. Intravitreal antibiotics will be required. Common pathogens associated with endophthalmitis include coagulase-negative *staphylococci*, *streptococci*, and *Bacillus cereus*. Intravitreal vancomycin plus ceftazidime is a common antibiotic regimen. Vitrectomy may be required.

Pearls

1. Most cases of endophthalmitis occur postoperatively within the 1st week following surgery.
2. Trauma-associated endophthalmitis can occur after penetrating globe trauma with the likelihood of infection increasing with delayed laceration closure or retained intraocular foreign bodies.
3. Intravitreal antibiotics administered by an ophthalmologist are required for the treatment of endophthalmitis.



FIGURE 2.50 ■ Endophthalmitis. Severe endophthalmitis and hypopyon as a postoperative complication. (Photo contributor: Lawrence B. Stack, MD.)

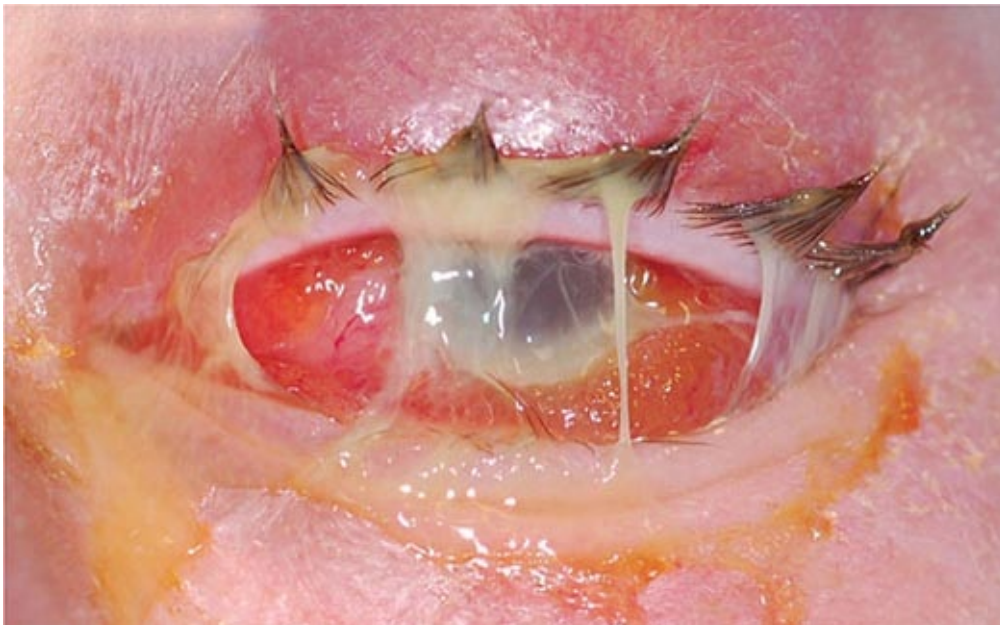


FIGURE 2.51 ■ Endophthalmitis. Severe endophthalmitis and orbital cellulitis as a complication of IV drug abuse. (Photo contributor: David Effron, MD.)

SYMPATHETIC OPHTHALMIA

Clinical Summary

This rare disorder presents as an insidious or acute anterior uveitis of both eyes (exciting and sympathizing eye) after recent or remote trauma to the exciting eye. The initial injury is usually from prior penetrating eye trauma with delayed wound closure or, less commonly, from prior eye surgery.

The disease is thought to be an autoimmune inflammatory response against choroidal melanocytes released due to trauma. It may develop rapidly or insidiously, presenting from days to years after the inciting traumatic event. Most cases develop within 1 year.

Earliest symptoms include eye floaters and loss of accommodation. The disease may progress to severe uveitis with redness, pain, and photophobia in the sympathizing, noninjured eye.

Classically, there is a granulomatous anterior chamber reaction with mutton-fat keratic precipitates seen on the corneal endothelium.

Management and Disposition

Diagnosis is suspected clinically. Consider other causes of granulomatous uveitis such as sarcoidosis, tuberculosis, and syphilis. Consult ophthalmology for management, which may include topical steroids, evisceration, or enucleation of the eye with prior trauma.

Pearls

1. Consider sympathetic ophthalmia with unexplained uveitis and a history of penetrating eye trauma.
2. Solicit a history of eye trauma or prior eye surgery when evaluating eye pain, blurred vision, and redness.
3. Early intervention can be sight saving.



FIGURE 2.52 ■ Sympathetic Ophthalmia. This patient with prior ocular trauma (untreated globe rupture) OS presented with pain and redness OD, thought to be due to sympathetic ophthalmia. OS enucleation and treatment with steroids resolved condition. (Photo contributor: R. Jason Thurman, MD.)

ANISOCORIA

Clinical Summary

Anisocoria is a disparity of pupil size. To determine the abnormal pupil, compare pupil sizes in light and dark. It is accentuated in the paretic muscle. If the iris sphincter (or its innervation) is involved, the anisocoria will be increased in bright light. If the iris dilator muscle (or its innervation) is affected, it will be more pronounced in darkness. Up to 20% of normal individuals have physiologic anisocoria of 1 to 2 mm.

Other causes of anisocoria with an abnormally large pupil include mydriatic drops, contamination from a scopolamine patch, an Adie pupil, and ocular trauma with iris sphincter damage. A dilated pupil due to anticholinergic agents (eg, atropine) does not react to light, although a dilated pupil due to

sympathomimetics still has some response.

An abnormally small pupil may be secondary to Horner syndrome, chronic Adie pupil (8 weeks or more after the event), iritis, and eye drops (pilocarpine). Miosis secondary to pupillary sphincter muscle spasm may be transiently observed after ocular trauma, followed by mydriasis.

Management and Disposition

Evaluation is dependent on clinical presentation. A patient with the acute onset of 3rd-nerve palsy with associated headache or trauma should be evaluated as a neurosurgical emergency.

Pilocarpine may be helpful to differentiate the etiology of pupil dilation. With low concentrations (0.125%), an Adie pupil will constrict more than the unaffected eye secondary to denervation supersensitivity. With higher concentrations (1%), a pupil that fails to constrict is most likely secondary to topical anticholinergic mydriatics (scopolamine, atropine, or cyclopentolate). Unlike the mydriasis seen with intracranial pathology, pharmacologic mydriasis is not associated with pain, ptosis, or diplopia. Other pathology within the iris sphincter muscle that prevents constriction to 1% pilocarpine includes iris muscle trauma and synechiae.

Pearls

1. A patient with a dilated pupil, ptosis, and abnormal extra-ocular movements should be evaluated emergently for an aneurysm or expanding supratentorial mass with tentorial herniation.
2. With physiologic anisocoria, the pupil size disparity is the same in light and dark, and there are no other ocular findings.
3. A driver's license or ID badge may be helpful to document a preexisting anisocoria.
4. Some brands of eye makeup contain belladonna alkaloids, which can cause mydriasis.
5. An Adie pupil initially is dilated but may become miotic over time. It is a benign condition that affects young adults, affects women more often than men, and is associated with decreased reflexes.



FIGURE 2.53 ■ Posttraumatic Anisocoria. Marked chronic anisocoria secondary to prior trauma as a child. (Photo contributor: R. Jason Thurman, MD.)

HERPES ZOSTER OPHTHALMICUS

Clinical Summary

Reactivation of endogenous latent varicella-zoster virus within the trigeminal ganglion with neuronal spread through the ophthalmic branch results in crops of grouped vesicles on the forehead and periocularly.

Patients typically present with periocular rash and an injected eye, along with a watery discharge. The most common corneal lesion is punctate epithelial keratitis, in which the cornea has a ground-glass appearance because of stromal edema. Pseudodendrites, also very common, form from mucous deposition, are usually peripheral, and stain moderate to poorly with fluorescein. These may be differentiated from the dendrites of HSV in that the pseudodendrites lack the rounded terminal bulbs at the end of the branches and are broader and more plaquelike. Anterior stromal infiltrates may be seen in the 2nd or 3rd week after the acute infection. Follicles (hyperplastic lymphoid tissue that appears as gray or white lobular elevations, particularly in the inferior cul-de-sac) and regional adenopathy may or may not be present. Iritis is seen in approximately 40% of patients.



FIGURE 2.54 ■ Herpes Zoster Ophthalmicus. A vesicular rash in the distribution of the ophthalmic division (V1) of the trigeminal nerve is seen. The presence of the lesion near the tip of the nose (Hutchinson sign) increases the risk of ocular involvement. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Treat patients with epithelial defects with topical broad-spectrum antibiotics to prevent secondary infection. Initiate oral antivirals within 72 hours of onset, and treat for 7 to 10 days. Use cycloplegics if an iritis is present. Artificial tears or ointment may be helpful, and narcotic analgesics may be required. Ophthalmologic consultation is indicated.

Pearls

1. Ocular complications may follow the rash by many months to years. These complications have a highly variable presentation that can mimic almost any ophthalmic condition.
2. Recurrence is more common in the immunocompromised host.

3. Perform a careful eye exam with corneal staining. Nearly two-thirds of patients will develop ocular lesions.
4. Corneal hypesthesia and the appearance of dendrites with fluorescein staining are seen in both herpes zoster ophthalmicus and herpes simplex keratitis.
5. Patients with skin lesions on the tip of the nose (Hutchinson sign) are at high risk for ocular involvement. However, the eye may be involved without a nasal lesion.



FIGURE 2.55 ■ Herpes Zoster Ophthalmicus. A large circular dendrite is seen in this patient with ocular involvement from herpes zoster virus. (Photo contributor: Alexandra Bingnear, RN.)



FIGURE 2.56 ■ Herpes Zoster Ophthalmicus. Grouped vesicles on the forehead and eyebrow are seen with conjunctival injection indicating ocular involvement. (Photo contributor: Lawrence E. Heiskell, MD, FACEP, FAAFP.)



FIGURE 2.57 ■ Herpes Zoster Ophthalmicus. Scabbed over lesions indicating late-stage herpes zoster ophthalmicus in the V1 distribution are seen. Marked conjunctival erythema and corneal ulcerations are also seen. (Photo contributor: Cindy Chang, MD.)

OCULAR HERPES SIMPLEX

Clinical Summary

Ocular herpetic disease may be neonatal, primary, or recurrent. Neonatal disease occurs secondary to passage through an infected birth canal and is usually HSV

type 2. Primary ocular herpes may present as a blepharitis (grouped eyelid vesicles on an erythematous base), conjunctivitis, or keratoconjunctivitis. Patients with keratoconjunctivitis commonly note pain, irritation, foreign-body sensation, redness, photophobia, tearing, and occasionally decreased visual acuity. Follicles and preauricular adenopathy may be present. Initially, the keratitis is diffuse and punctate, but after 24 hours, fluorescein demonstrates either serpiginous ulcers or multiple diffuse epithelial defects. True dendritic ulcers are rarely seen in primary disease.

Most ocular herpetic infections are manifestations of recurrent disease rather than a primary ocular infection. These may be triggered by ultraviolet laser treatment, topical ocular medications (β -blockers, prostaglandins), and immunosuppression (especially ophthalmic topical glucocorticoids). Recurrent disease most commonly presents as keratoconjunctivitis with a watery discharge, conjunctival injection, irritation, blurred vision, and preauricular lymph node involvement. Corneal involvement initially is punctate, but evolves into a dendritic keratitis. The linear branches classically end in bead-like extensions called terminal bulbs. Fluorescein dye demonstrates primarily the corneal defect; the terminal bulbs are best seen with rose stain. In addition to the dendritic pattern, fluorescein stain may instead take on a geographic or ameboid shape, secondary to widening of the dendrite. Most patients (80%) with herpes simplex keratitis have decreased or absent corneal sensation in the area of the dendrite or geographic ulceration. Deeper corneal stromal inflammation may also occur (disciform keratitis). Recurrent disease can also present with iritis or with blepharitis, with vesicles grouped in focal clusters.



FIGURE 2.58 ■ Ocular Herpes Simplex. This 10-year-old has recurrent ocular herpes simplex since age 6. Vesicles should not be mistaken for hordeola. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

There is a high association in neonatal ocular herpes infections between ocular HSV disease and serious systemic or neurologic infection, and an emergency pediatric or infectious disease consult is necessary. IV acyclovir is indicated, and the ocular disease itself may be treated with topical antivirals. Other sexually transmitted diseases such as chlamydia or gonorrhea should be explored.

Treatment of those with primary ocular herpes (beyond the neonatal period) presenting as blepharitis or periocular dermatitis consists of good local hygiene and a prophylactic topical antiviral such as trifluorothymidine or idoxuridine ointment. Patients with corneal involvement should additionally receive topical antibiotics to prevent secondary bacterial infection.



FIGURE 2.59 ■ Ocular Herpes Simplex. Grouped vesicles on an erythematous base with periorbital erythema are seen in this patient with a history of ocular herpes simplex. Honey-colored crusts suggest secondary impetigo. (Photo contributor: Lawrence B. Stack, MD.)

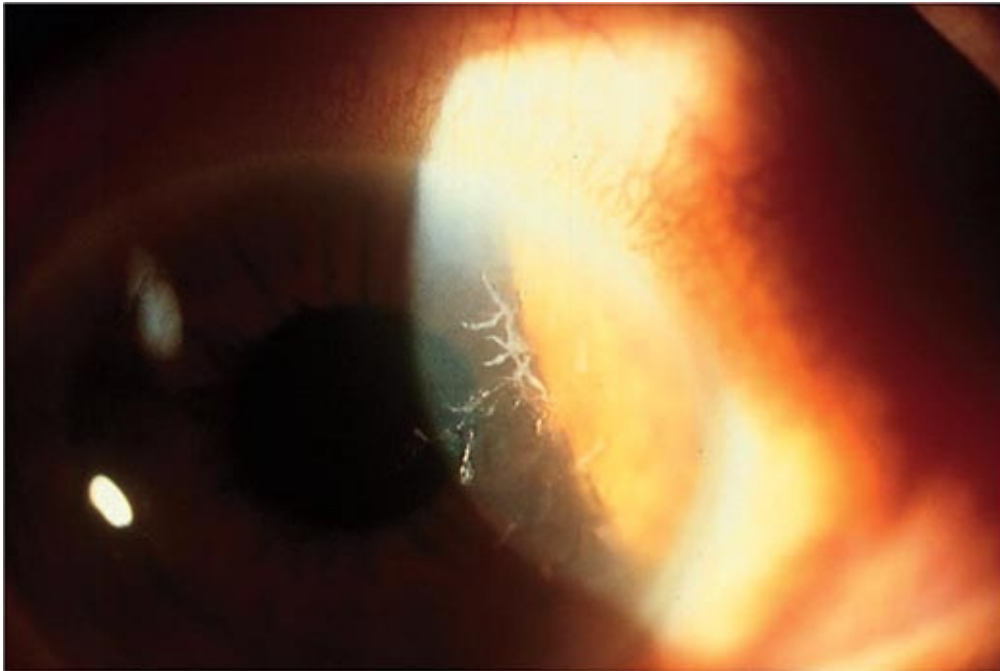


FIGURE 2.60 ■ Herpes Simplex Keratitis. Unstained dendritic lesions. (Photo contributor: Lawrence B. Stack, MD.)

In those with recurrent disease, topical antivirals (trifluri-dine, ganciclovir, acyclovir, brivudine) are effective, as is oral acyclovir. Ophthalmology consultation is required in patients with ocular HSV disease. Episodes of

recurrent stromal disease may be limited by the long-term use of low-dose oral antivirals.

Pearls

1. Most ocular HSV infections are manifestations of recurrent disease.
2. Corneal disease is the most prevalent form of ocular HSV disease.
3. Oral antivirals are now frequently used for keratitis because of their convenience, although topical optical antivirals are equally effective.
4. HSV dendrites, when stained with fluorescein, appear as branching lesions with terminal bulbs.
5. Corneal hypesthesia may be easily overlooked in the initial evaluation of a red eye. If a topical anesthetic has been given, a reexamination 1 hour later is helpful for evaluating hypesthesia.

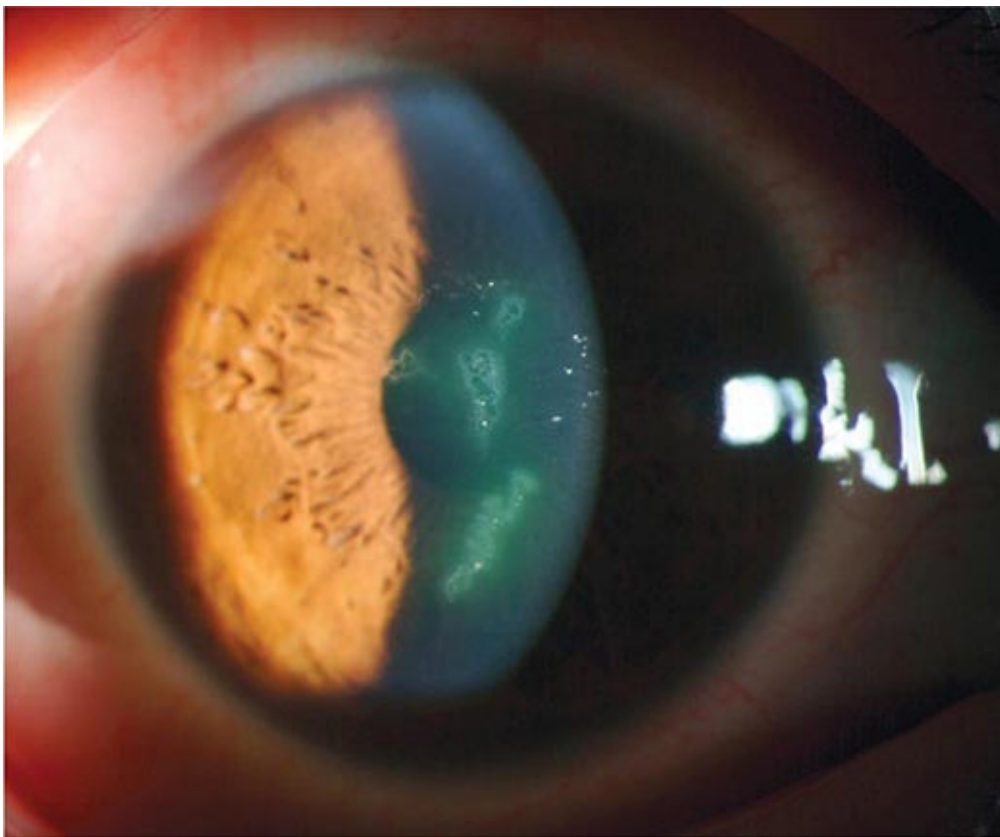


FIGURE 2.61 ■ Herpes Simplex Keratitis. A slit-lamp view of dendritic lesions under white light. (Used with permission from The University of Iowa and EyeRounds.org.)

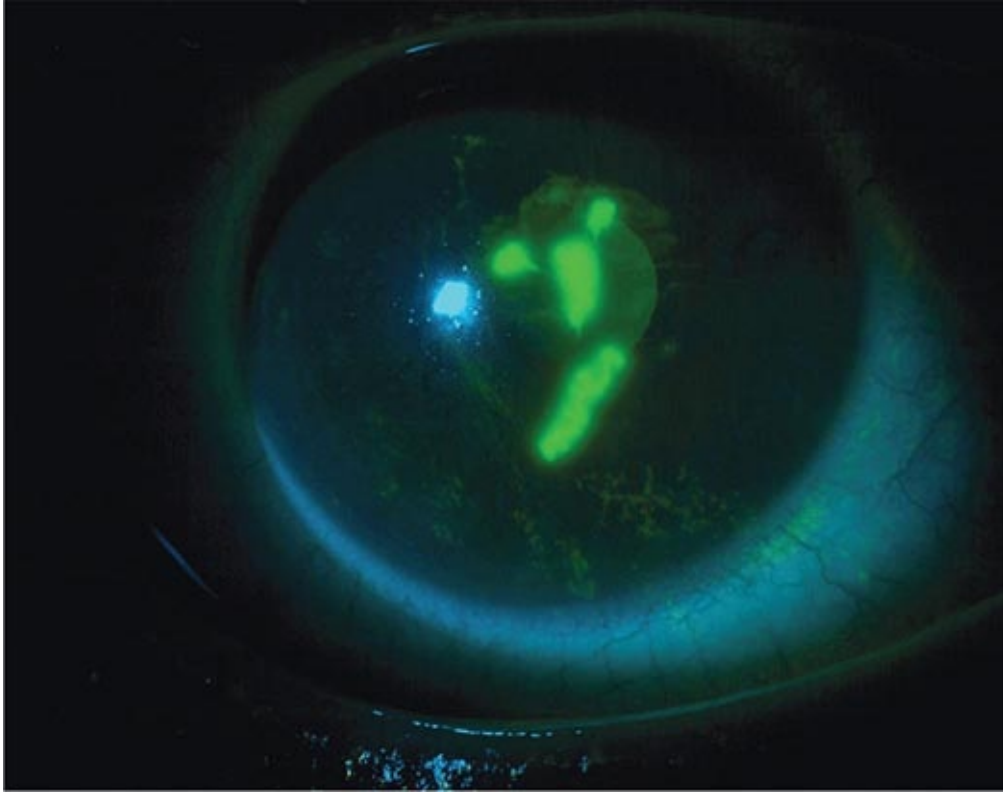


FIGURE 2.62 ■ Herpes Simplex Keratitis. Epithelial dendrites seen in [Fig. 2.61](#) after fluorescein staining. (Used with permission from The University of Iowa and [EyeRounds.org](#).)

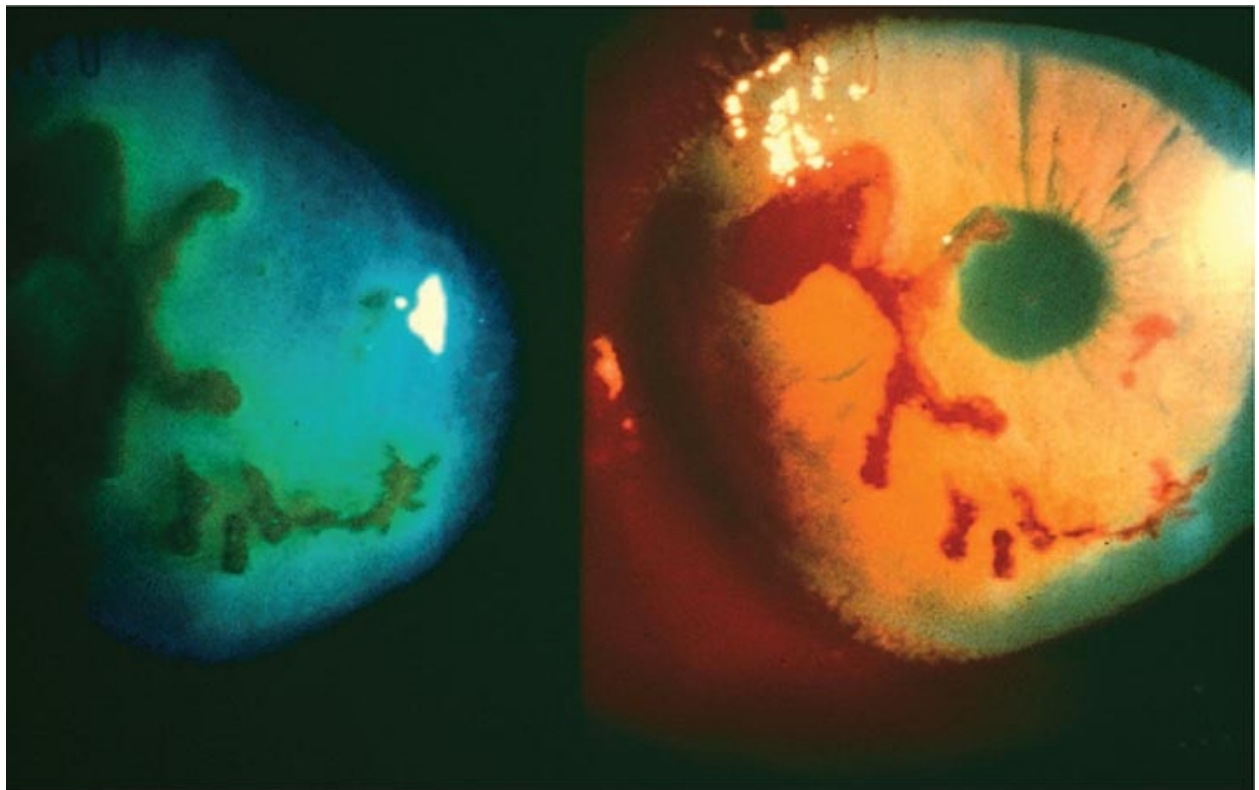


FIGURE 2.63 ■ Herpes Simplex Keratitis. Fluorescein (*left*) and rose bengal (*right*) stains demonstrate characteristic dendritic patterns. Whereas fluorescein staining is used to detect epithelial defects, rose bengal staining additionally demonstrates degenerating or dead epithelial cells and is particularly good for demonstrating the club-shaped terminal bulbs at the end of each branch. (Used with permission from the American Academy of Ophthalmology. *External Disease and Cornea: A Multimedia Collection*. San Francisco; 1994. © American Academy of Ophthalmology.)

CORNEAL ULCER

Clinical Summary

A corneal ulcer is an inflammatory and ulcerative keratitis. Common infectious etiologies include bacteria (*Staphylococcus*, *Streptococcus*, *Pseudomonas*) and viruses (HSV, adenovirus). Bacterial corneal ulcers are commonly associated with extended-wear contact lenses. Rare causes of corneal ulcers include fungal infections and *Acanthamoeba*, a ubiquitous protozoan associated with contaminated contact lens solutions. Fungal infections may also arise from trauma involving vegetable matter such as a tree branch. *Acanthamoeba* infections may also occur from swimming in lakes, especially while wearing contact lenses.

Patients present with pain, photophobia, decreased vision, discharge, and a foreign-body sensation. Ocular findings include a corneal infiltrate, typically a round white spot, with conjunctival hyperemia, meiosis, and chemosis. Slit-lamp bio-microscopy may demonstrate an epithelial defect with fluorescein uptake. Anterior chamber findings can include cells and flare, keratic precipitates, and a hypopyon.

Management and Disposition

Corneal ulcers are an ophthalmologic emergency requiring emergent ophthalmology consultation. Stains and cultures should be obtained as expeditiously as possible. Intensive topical treatment using fortified antibiotics is the most effective treatment route, initially given every 30 to 60 minutes. For mild cases, a single fluoroquinolone agent may suffice. For more severe cases, dual therapy using a cephalosporin or vancomycin combined with an aminoglycoside is recommended. Clinical improvement is usually noted after 2 to 3 days. Systemic antibiotics are used in cases where the sclera is involved

(*Pseudomonas*) or if there is a high risk of concurrent systemic disease (*Neisseria*, *Haemophilus*). Cycloplegics are recommended if there is an accompanying iritis. Steroids and eye patching are contraindicated. A contact lens wearer must discontinue contact lens wear.

Pearls

1. A corneal ulcer is an ophthalmologic emergency.
2. Extended-wear contact lens use is a risk factor for corneal ulcer.
3. *Pseudomonas aeruginosa*, associated with thick yellow-green or blue-green mucopurulent tenacious exudate, is capable of destroying the cornea within 6 to 12 hours.
4. *Acanthamoeba* should be suspected in contact lens wearers with contaminated lens solutions or who swim wearing their contact lens. Classically, these patients have pain out of proportion to their clinical findings.
5. Infectious ulcers tend to develop centrally, away from the vascular supply and immune system of the limbus.



FIGURE 2.64 ■ Corneal Ulcer. An elliptical ulcer at 5-o'clock position near the limbus is seen. This location is atypical for a bacterial ulcer. The patient presented with painful red eyes and normal uncorrected vision, but wore cosmetic soft contact lenses. Bilateral corneal ulcers were diagnosed, which cleared after treatment with topical ciprofloxacin. Note that the ciliary flush seen in the nasal portion of the limbus is not to be mistaken for conjunctivitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

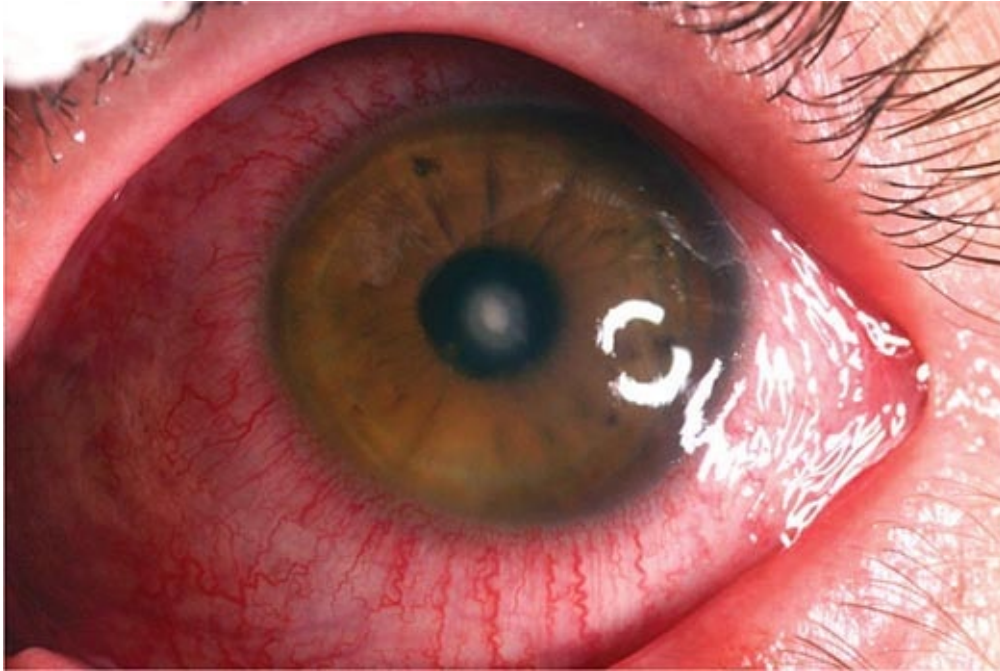


FIGURE 2.65 ■ Corneal Ulcer. A white circular corneal infiltrate is seen in the central visual axis in this contact lens wearer. (Photo contributor: Kevin J. Knoop, MD, MS.)

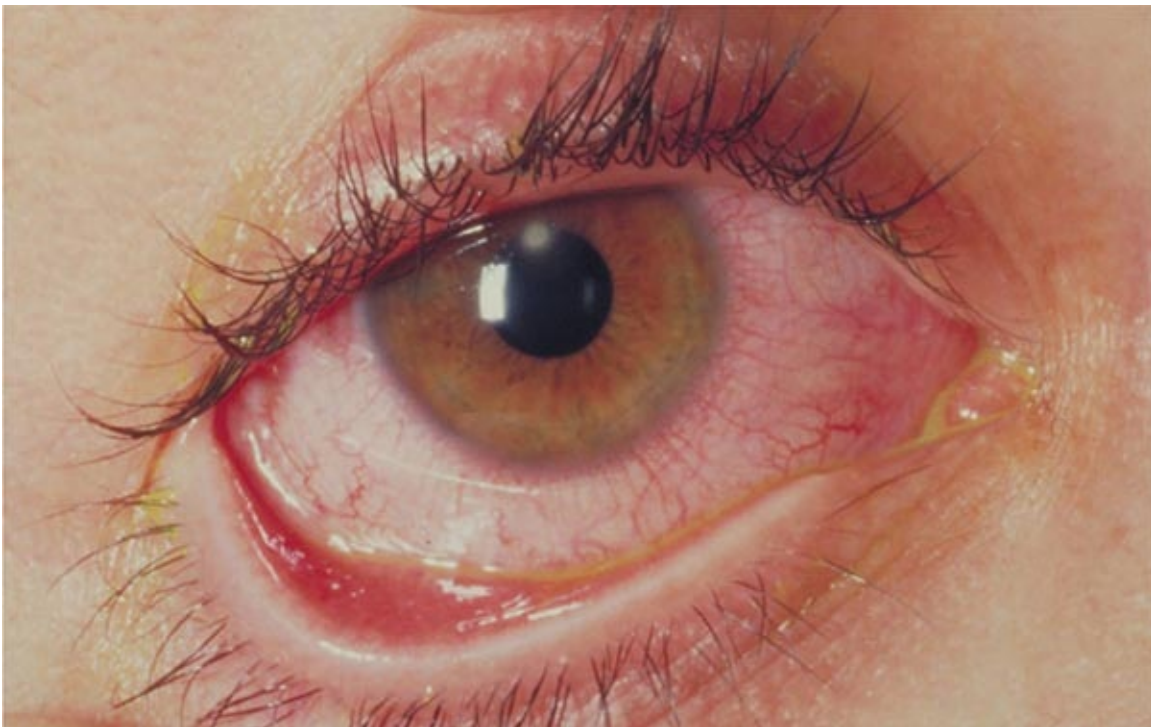


FIGURE 2.66 ■ Corneal Ulcer. A small circular corneal infiltrate is seen adjacent to the white flash photography reflection. Diffuse conjunctival hyperemia with a nasal ciliary flush is seen. (Photo contributor: Lawrence B. Stack, MD.)

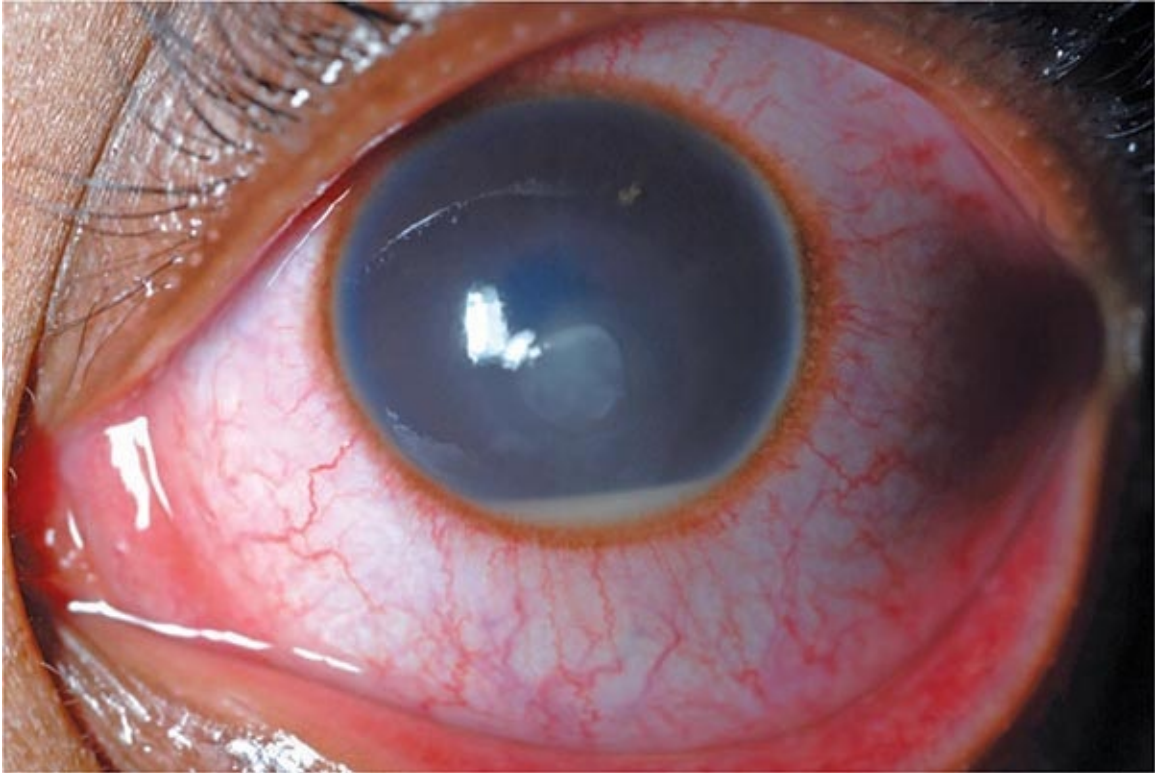


FIGURE 2.67 ■ Corneal Ulcer with Hypopyon. A hypopyon has developed from a corneal ulcer seen near the visual axis. The conjunctiva is inflamed and a ciliary flush is present. (Photo contributor: R. Jason Thurman, MD.)

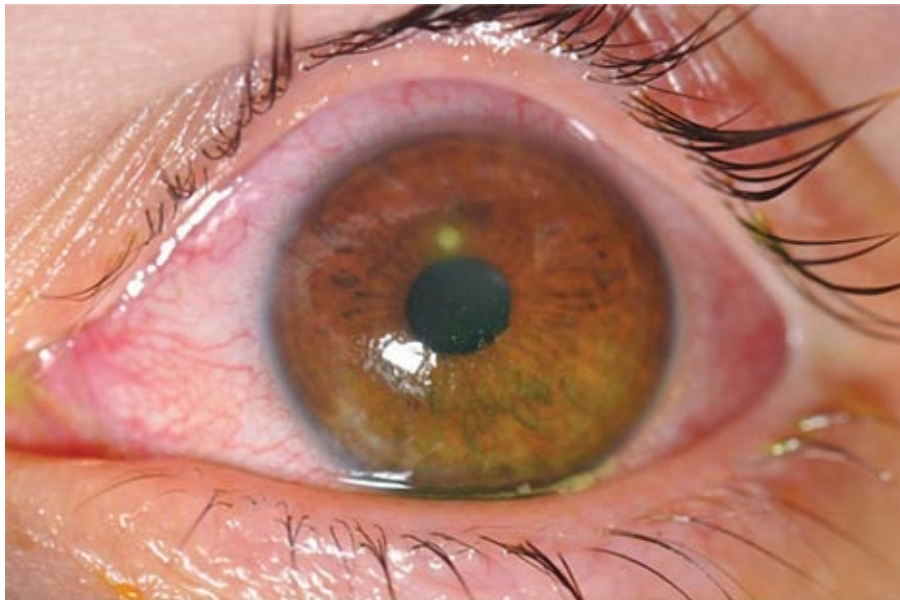


FIGURE 2.68 ■ Corneal Ulcer. Diminished visual acuity and pain due to the corneal ulcer just superior to the visual axis contributed to his motor vehicle crash and airbag deployment, causing a large corneal abrasion of the inferior third of the cornea. (Photo contributor: Lawrence B. Stack, MD.)

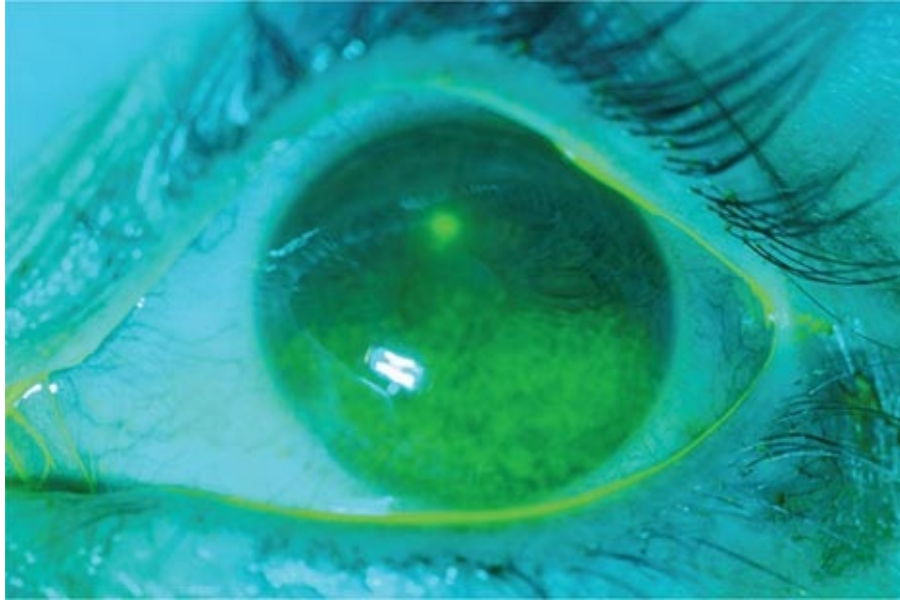


FIGURE 2.69 ■ Corneal Ulcer. Fluorescein stain uptake of the cor-neal ulcer just above the visual axis and “exposure pattern” abrasion of the inferior third of the corneal surface due to airbag deployment. Consider concurrent alkali injury from the airbags. (Photo contributor: Lawrence B. Stack, MD.)

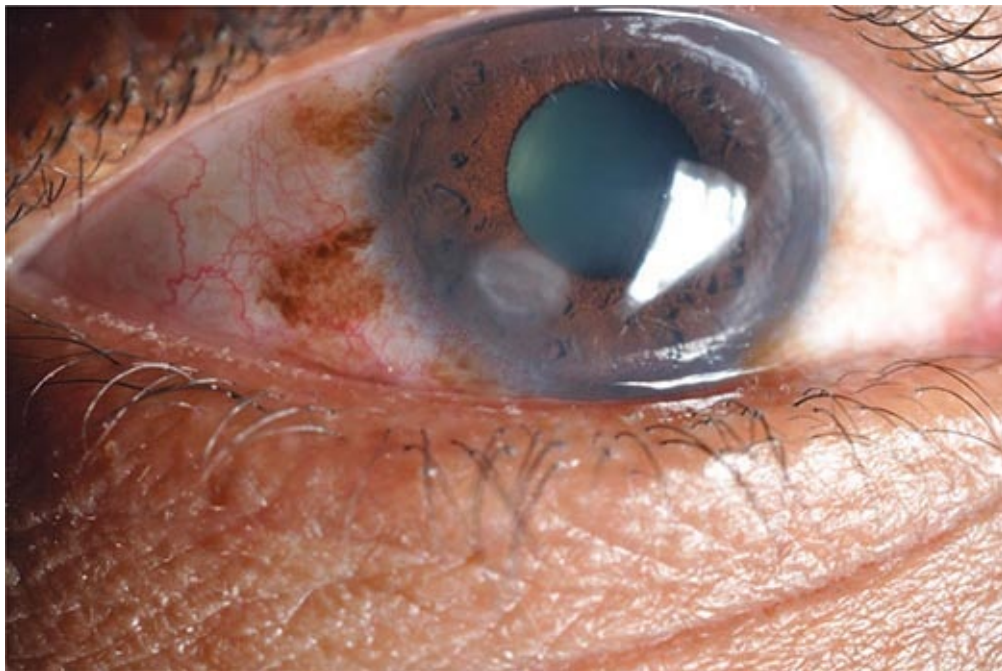


FIGURE 2.70 ■ Corneal Ulcer. A large oval opaque corneal ulcer is seen at 8-o’clock position toward the limbus in a contact-lens wearing patient. (Photo contributor: Lawrence B. Stack, MD.)

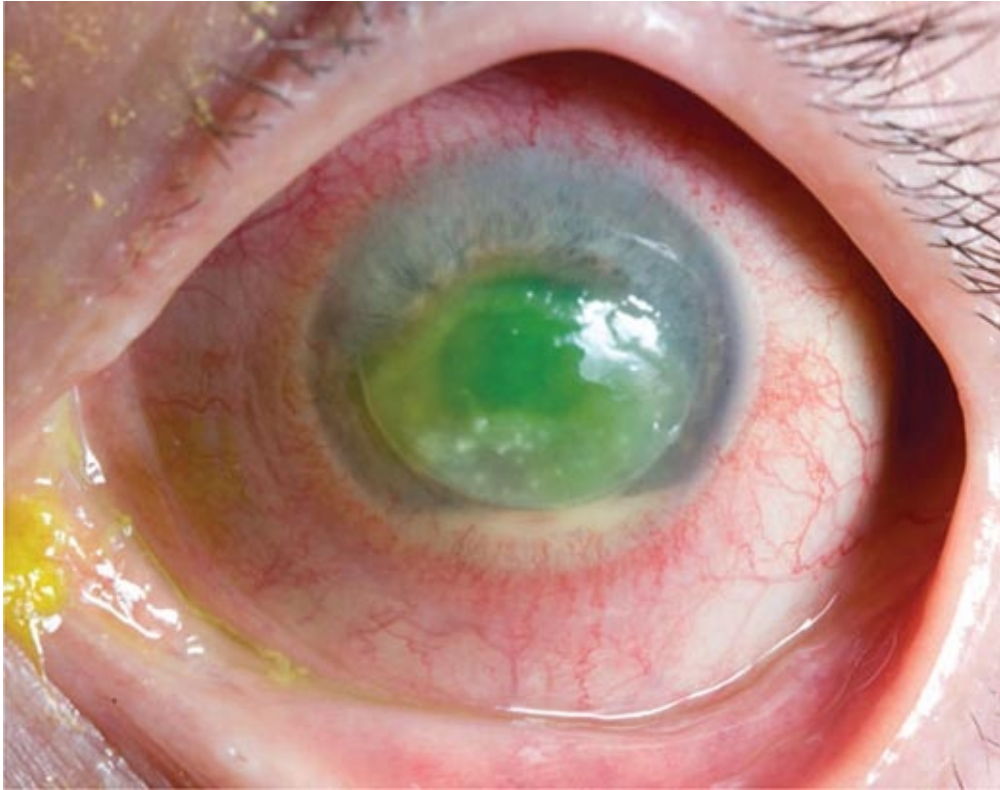


FIGURE 2.71 ■ Corneal Ulcer with Hypopyon. A middle-aged female accidentally splashed dishwashing detergent in her eye 1 week prior to ED visit. A large inferior corneal ulcer, with corneal edema and hypopyon, is seen. (Photo contributor: Eugene Eiland, MD.)

AFFERENT PUPILLARY DEFECT

Clinical Summary

Pupil size is controlled in the midbrain, from which efferent nerves travel to both pupils, resulting in symmetric pupils, even with a unilateral light stimulus. The perception of the light stimulus, however, may be decreased by disease within anterior visual structures such as the retina, optic nerve, chiasm, optic tract, and midbrain pathways. With diminished afferent stimulation, less light is “perceived,” and pupil contraction diminishes (ie, dilates) as a result. In this situation, the affected side is said to demonstrate an afferent pupillary defect (APD).

An APD is best appreciated by the swinging flashlight test, which discloses differences in afferent stimuli between the two eyes. A flashlight is directed onto one pupil and then the other. The normal response is a prompt constriction.

(Generally, this is followed by a slight “release” dilation.) During the brief interval required to move the light from one to the other eye, both pupils begin to dilate. Thus, under normal circumstances, when the light reaches the second eye, reflex constriction again occurs. With a diseased eye or pathway, the diseased side perceives less light and the mid-brain therefore sets the pupil size to be larger, and as a result, the pupils dilate (or continue to dilate following the “release” dilation).

Although sensitive, the swinging flashlight test is not specific, since the pathology may be anywhere in the visual pathway from the retina to the midbrain. The test cannot distinguish between unilateral afferent disease and asymmetric bilateral disease. Typically, a positive finding indicates optic nerve disease such as ischemic optic neuropathy, optic neuritis (including that seen with multiple sclerosis), retrobulbar optic neuritis, and glaucoma.

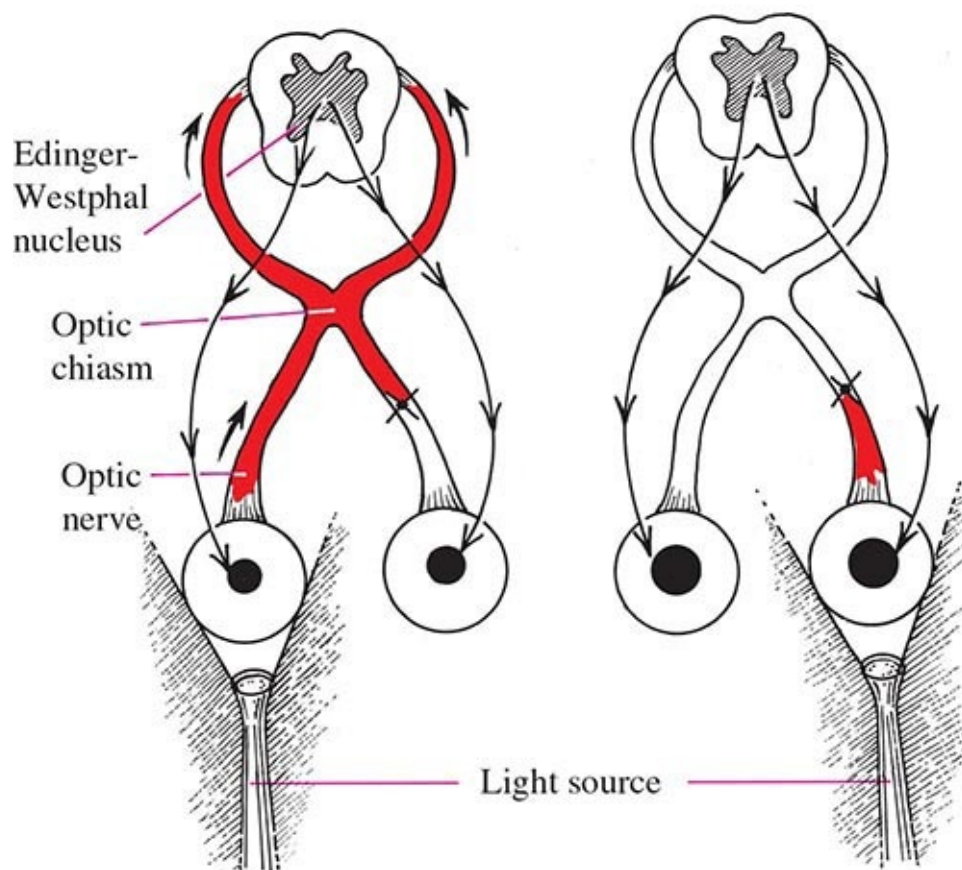


FIGURE 2.72 ■ Afferent Pupillary Defect. Schematic representation of an afferent pupillary defect (APD) due to neurologic lesion in the anterior visual pathway.

Management and Disposition

The finding of an afferent pupillary defect is nonspecific. A fundoscopic exam may show occlusion of the central retinal vein or central retinal artery or a dense hemorrhage in the vitreous. Otherwise, a neurologic evaluation (history, physical examination, and computed tomography [CT] scan) is important to assess for treatable conditions. In the absence of gross ocular or neurologic disease, a clinically stable patient may be discharged from the ED with ophthalmology follow-up.

Pearls

1. An APD does not cause anisocoria because any change in light input results in a bilaterally symmetric output from the midbrain.
2. In the swinging flashlight test, both pupils will dilate when the flashlight moves from the normal pupil to the affected pupil, since pupil size is centrally mediated in the midbrain.
3. Bilaterally symmetric disease produces no APD, since the basis for the swinging flashlight test is asymmetry of disease.
4. A dense vitreous hemorrhage is capable of producing a mild defect. Lesser ocular pathology such as branch retinal vein or artery occlusion or a cataract is unlikely to produce a clinically detectable APD.
5. Dim room illumination may be helpful when performing the swinging flashlight test. The patient should focus on an object 15 feet away to avoid the pupil constriction normally seen with accommodation.



FIGURE 2.73 ■ Marcus Gunn Pupil. These photographs demonstrate an afferent pupillary defect of the left pupil. When the light shines in the affected left eye (**B**), the pupils are less constricted than when the light shines in the right eye (**A**). Thus, when the light is swung from the right pupil to the left, the pupils appear to dilate. The anisocoria here is subtle. (Photo contributor: Frank Birinyi, MD.)

THYROID EYE DISEASE

Clinical Summary

Thyroid eye disease (TED) is typically seen in individuals with Graves disease, but also is found in Hashimoto thyroiditis. Similar to conjunctival icterus, it is a sign of underlying disease that must be explored. TED has many eye signs but is typically characterized by eyelid retractions, lid lag, redness (erythema), conjunctivitis, and bulging or “bug” eyes (exophthalmos). This is due to the autoimmune process of the disease, where auto-antibodies target eye muscle fibroblasts, which change into fat, expand, and become inflamed, leading to manifestations of TED.



FIGURE 2.74 ■ Thyroid Eye Disease. Lateral lid retraction (temporal flare) with scleral show is seen in this patient with chronic hyperthyroidism. (Used with permission from The University of Iowa and EyeRounds.org.)



FIGURE 2.75 ■ Thyroid Eye Disease. Marked chemosis is seen in this patient with hyperthyroidism. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

TED is diagnosed clinically when presenting to the ED. Thyroid hormones (triiodothyronine, free thyroxine, thyroid-stimulating hormone) and imaging (CT or magnetic resonance imaging [MRI] orbits) can support the diagnosis. Once diagnosed, regulating hormone levels is key and referral to an endocrinologist is key for long-term management of the underlying disease. However, TED will progress independently of the thyroid disease and should be managed separately. Systemic steroids in conjunction with orbital radiation and immunotherapy (eg, rituximab, teprotumumab) can be used initially before considering surgical interventions.

Pearls

1. TED is a sign of underlying thyroid disease that is looming.
2. Early recognition of TED is crucial for determining the appropriate management for preservation of vision.

INTERNUCLEAR OPHTHALMOPLÉGIA (INO)

Clinical Summary

Internuclear ophthalmoplegia (INO) is a specific gaze deficit caused by lesions to the medial longitudinal fasciculus (MLF) of the dorsomedial brain stem. It presents with horizontal eye movement deficits characterized by poor adduction in the affected eye and abduction nystagmus in the contralateral eye.

Occasionally INO can be present bilaterally. Smooth binocular vision and the ability to visually track require synchronized ocular movements. The MLF coordinates neuronal signaling between cranial nerves III, IV, and VI to achieve synchronous eye movements. The majority of INO cases are associated with multiple sclerosis, although mass lesions, trauma, and cerebrovascular disease are potential causes as well. The diagnosis of INO is made by neurologic examination, specifically assessing horizontal eye movement. Findings can be subtle and may require specialized neurologic testing.



FIGURE 2.76 ■ Internuclear Ophthalmoplegia. Internuclear ophthalmoplegia deficits are present bilaterally and are a common presentation in multiple sclerosis patients. Horizontal gaze is impaired, with poor adduction seen with leftward gaze (**A**) and rightward gaze (**B**). (Photo contributor: David Effron, MD.)

Management and Disposition

Depending on the underlying cause, symptoms may resolve over months or persist indefinitely. Eye patching may be used to provide symptom relief from diplopia. For patients with multiple sclerosis, treatment of the underlying demyelinating disorder may improve symptoms. All patients with INO require neurologic consultation and MRI. An acute ischemic stroke as a potential cause for new-onset INO should be investigated and may require hospital admission.

Pearls

1. INO is the most common ocular movement abnormality in multiple sclerosis.
2. Ischemic infarction can result in INO and should be investigated with a complete stroke evaluation, especially in older patients.
3. Bilateral INO is commonly secondary to multiple sclerosis.

HORNER SYNDROME

Clinical Summary

Horner syndrome (miosis, ptosis, and anhidrosis) is secondary to loss of ocular sympathetic innervation. Ptosis is less than 2 mm, the result of paralysis of Müller muscle, innervated by the sympathetic pathway. Anhidrosis is often not apparent to patients or clinicians. A pupillary finding specific in Horner syndrome is dilation lag. Because the dilator muscle is weak, the pupil dilates more slowly than the normal pupil.

This loss of ocular sympathetic innervation can be produced by a lesion anywhere along a three-neuron sympathetic pathway, from the hypothalamus down through the brain stem to the cervical cord, in the apex of the chest, along the carotid sheath, and in the cavernous sinus or orbit. Isolated Horner syndrome presenting with head or neck pain suggests an internal carotid artery dissection.

Management and Disposition

Associated signs and symptoms help direct the ED workup. A patient with cranial nerve abnormalities requires CT or MRI imaging and admission. In the setting of cervical spine trauma, neck immobilization and appropriate imaging studies are instituted. Consider carotid artery dissection in neck pain without trauma.

Pearls

1. Classic signs of a Horner syndrome include miosis and mild ptosis. Anhidrosis is less readily appreciated, and occurs with first- or second-order lesions only.

2. Miosis and dilation lag are accentuated in a darkened room.
3. A Pancoast tumor usually is caused by bronchogenic carcinoma and may present, as its presenting sign, as a Horner syndrome.
4. Cluster headaches, with autonomic sympathetic system dysfunction, are capable of producing an ipsilateral Horner syndrome.
5. Ipsilateral extraocular paresis, especially a 6th-nerve palsy, without other brain stem signs localizes pathology to within the cavernous sinus.



FIGURE 2.77 ■ Horner Syndrome. Unilateral miosis and ptosis are seen in this patient with Horner syndrome and sarcoma metastatic to the spine. (Photo contributor: Frank Birinyi, MD.)

MYASTHENIA GRAVIS

Clinical Summary

Myasthenia gravis is an autoimmune disorder of neuromuscular transmission that results in weakness of multiple muscle groups. The disease is antibody-mediated with a T-cell–modulated attack on postsynaptic acetylcholine receptors at the neuro-muscular junction. The two clinical presentations of myasthenia gravis are ocular and generalized disease. In ocular disease, weakness is limited

to the eyelids and extraocular muscles, whereas in generalized disease, bulbar (eg, dysarthria, dysphagia), limb, and respiratory muscles may be involved in addition to the eyes. More than half of myasthenia gravis patients present with ptosis and/or diplopia. Ptosis can alternate from eye to eye with severity waxing and waning. Extraocular muscle weakness may manifest as binocular diplopia, with early symptoms presenting simply as blurry vision. The pupils are always spared in myasthenia gravis, differentiating it from other extra-ocular movement disorders.

Management and Disposition

Patients with suspected myasthenia gravis warrant neurologic consultation. Initial therapy for patients includes oral acetylcholinesterase inhibitors (pyridostigmine). For severe disease (myasthenic crisis) that is rapidly progressive or results in respiratory compromise, therapies can include plasma exchange, IV immunoglobulin, and systemic glucocorticoids.

Pearls

1. Ocular manifestations (ptosis, extraocular muscle deficits) may be the 1st sign of myasthenia gravis and should be recognized in the ED.
2. The “ice pack test” can be used in evaluation of patients with ptosis. Place a bag of ice over the closed lid for 2 minutes. Improvement of ptosis is a positive test result; neuromuscular transmission improves with decreased temperature.
3. Myasthenic crisis is life threatening, resulting in severe oropharyngeal and/or respiratory muscle weakness requiring intubation and ventilatory support.



FIGURE 2.78 ■ Myasthenia Gravis with Ptosis. Patient with ptosis of left eye associated with myasthenia gravis before the ice pack test. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 2.79 ■ Myasthenia Gravis with Ptosis. Patient with resolved ptosis of left eye after ice pack test. (Photo contributor: Lawrence B. Stack, MD.)

THIRD-NERVE PALSY

Clinical Summary

The 3rd cranial nerve innervates all the extraocular muscles except the lateral rectus and superior oblique (LR6, SO4). It also controls the levator palpebrae muscle and supplies parasympathetics to the pupillary constrictor and ciliary muscles. Therefore, the clinical examination is notable for a dilated and

unreactive pupil, limited extraocular movements, and ptosis. The eye rests in a position of abduction because of unopposed action of the lateral rectus.

Third-nerve dysfunction can result from lesions anywhere along its path from the oculomotor nucleus in the midbrain, within the subarachnoid space, traversing the cavernous sinus, and terminating in the extraocular muscles within the orbit. Contralateral hemiparesis suggests brain stem involvement (Weber syndrome). Pathology within the subarachnoid space causing a 3rd-nerve palsy includes compression of the nerve by a posterior communicating artery aneurysm, uncal herniation, or compressive neoplasm.

Pathology within the cavernous sinus causing a 3rd-nerve palsy includes carotid artery aneurysm, cavernous sinus thrombosis, and carotid-cavernous fistula. Third-nerve lesions here are often accompanied by lesions involving the 4th, 5th (ophthalmic branch), and 6th cranial nerves. Orbital pathology such as inflammation, trauma, or neoplasm should be suspected when orbital findings such as chemosis, conjunctival injection, or proptosis are seen.

Isolated 3rd-nerve palsies (“pupil-sparing”) are usually caused by microvascular ischemia. Diabetes, hypertension, and advanced age are risk factors. These typically present with intact pupillary function probably because of the superficial location of the pupillomotor fibers.

Management and Disposition

In patients with brain stem involvement, CT or MRI is indicated. Associated fever, headache, and altered consciousness should prompt CT scanning and subsequent lumbar puncture. MRI with gadolinium is preferred for evaluation of the cavernous sinus, and CT scanning is recommended for suspected orbital pathology.

Of particular concern is the sudden onset of 3rd-nerve palsy accompanied by a “thunderclap” headache, stiff neck, and depressed level of consciousness. Even those with “pupil sparing” should be evaluated as a neurosurgical emergency with emergent neuroimaging to evaluate for aneurysm and uncal herniation. If subarachnoid hemorrhage is not found and suspicion of aneurysmal leak remains high, a lumbar puncture should be considered.

In the setting of head trauma and oculomotor palsy, the workup should proceed expeditiously, with measures to reduce intracranial pressure.

Pearls

1. Patients with the abrupt onset of a “thunderclap” headache and 3rd-nerve palsy require immediate evaluation for an aneurysm. The posterior communicating artery is a common site.
2. In patients over 50 with 3rd-nerve palsies whose pupil is unaffected (“pupil sparing”), the etiology is usually hyper-tensive or diabetic vascular disease.
3. Mild or moderate pain is common in ischemic lesions.
4. Third-nerve palsy is unlikely to cause isolated mydriasis. Other etiologies such as tonic pupil, iris sphincter damage, and pharmacologic mydriasis are more likely.
5. Eighty percent of carotid-cavernous fistulas result from trauma and may present weeks after minor trauma. Findings include an ocular bruit, pain, pulsatile exophthalmos, and chemosis.



FIGURE 2.80 ■ Isolated Third-Nerve Palsy. This 60-year-old woman with diabetes mellitus presents with an isolated third-nerve palsy on the right. She is attempting to look leftward. Her symptoms began as an isolated pain above her right orbit 10 days prior at which time her double vision with leftward gaze began. (Photo contributor: Lawrence B. Stack, MD.)

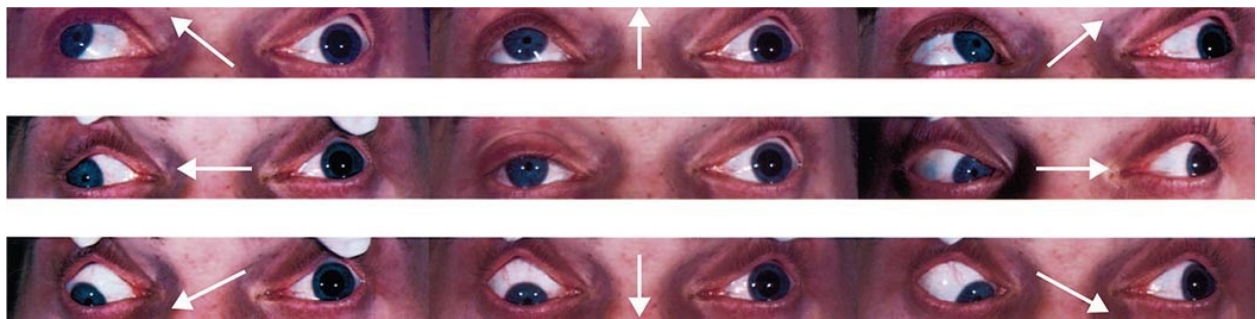


FIGURE 2.81 ■ Third-Nerve Palsy. This composite shows the defects of a left third cranial nerve palsy. Conjugate eye movement is present only when the affected eye gazes laterally to the affected side (intact lateral rectus). When gaze is directly ahead, exotropia is seen secondary to the unopposed lateral rectus muscle of the affected side. (Photo contributor: Frank Birinyi, MD.)



FIGURE 2.82 ■ Third-Nerve Palsy. Decreased motility of the right eye in all directions except for abduction in a patient with a right pupil-sparing third-nerve palsy secondary to poorly controlled hypertension and diabetes. (Used with permission from The University of Iowa and EyeRounds.org.)

SIXTH-NERVE PALSY

Clinical Summary

The abducens nerve innervates the lateral rectus muscle and is the most common single muscle palsy, causing loss of abduction and resultant horizontal diplopia, worse in ipsilateral gaze. Associated findings are dependent on the location of the lesion. Within the pons, involvement of the corticospinal tract results in contralateral hemiparesis. The abducens has the longest intracranial course of any nerve, and therefore is vulnerable to stretching or compression secondary to

elevated intracranial pressure, trauma, neurosurgical manipulation, and cervical traction. Also, any meningeal process (infectious, inflammatory, or neoplastic) can affect this portion of the 6th nerve. Aneurysmal compression is uncommon.

Prior to entering the cavernous sinus, the nerve crosses the petrous portion of the temporal bone. Trauma with temporal bone fracture can result in a combination of sixth- and seventh-nerve palsies. Cavernous sinus pathology is suggested by the involvement of the internal carotid artery, venous drainage of the eye and orbit, trochlear and oculomotor nerves, the 1st division of the trigeminal nerve, and the ocular sympathetics. Microvascular changes secondary to diabetes, hypertension, and giant cell arteritis can compromise function.

Management and Disposition

Associated signs and symptoms guide the ED workup. CT or MRI is indicated if brain stem or cavernous sinus involvement is suspected. Pathology localizing to the subarachnoid space should prompt consideration for CT scanning and subsequent spinal tap. In the elderly, an isolated 6th-nerve palsy is likely ischemic, transient, and not indicative of underlying neurologic disease. In these cases, a glucose and erythrocyte sedimentation rate are appropriate; these patients can be followed as outpatients provided close follow-up is arranged.

There is no treatment for the palsy itself except for patching the affected eye if diplopia is bothersome.

Pearls

1. An isolated sixth-nerve palsy is commonly due to microvascular disease, not an aneurysm.
2. Basilar skull fractures of the temporal bone are capable of producing a sixth-nerve palsy.
3. A sixth-nerve palsy associated with a Horner is usually localized to the cavernous sinus, since sympathetic fibers, as they traverse from the internal carotid artery to the oculomotor nerve, may briefly accompany the abducens nerve.

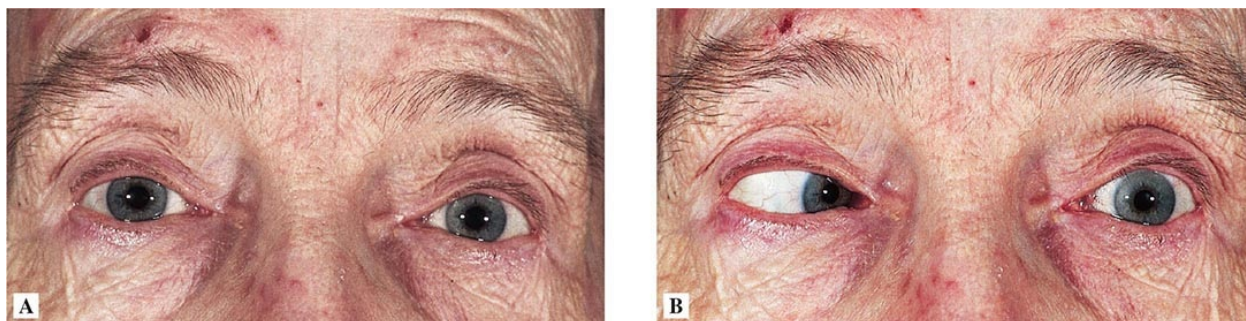


FIGURE 2.83 ■ Sixth-Nerve Palsy. Loss of abduction of the left eye is seen in lateral gaze. (Photo contributor: Frank Birinyi, MD.)



FIGURE 2.84 ■ Sixth-Nerve Palsy. Isolated sixth-nerve palsy of the right eye after acute head trauma. (Photo contributor: Kevin J. Knoop, MD, MS.)

¹The authors acknowledge the special contribution of Frank Birinyi, MD, Thomas F. Mauger, MD, Andrew Hendershot, MD, Marc E. Levsky, MD, and Paul DeFlorio, MD, for portions of this chapter written for the previous editions of this book.

Chapter 3

FUNDUSCOPIC FINDINGS

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Papilledema. Blurred disk margins indicate papilledema. A small subhyaloid hemorrhage is also seen inferior to the disk at 5-o'clock position. (Photo contributor: Aaron Sobol, MD.)

NORMAL FUNDUS

Clinical Summary

Disk

The disk is pale pink, approximately 1.5 mm in diameter, with sharp, flat margins. The physiologic cup is located within the disk and usually measures less than six-tenths the disk diameter. The cups should be approximately equal in both eyes.

Vessels

The central retinal artery and central retinal vein travel within the optic nerve, branching near the surface into the inferior and superior branches of arterioles and venules, respectively. Normally the walls of the vessels are not visible; the column of blood within the walls is visualized. The venules are seen as branching, dark red lines. The arterioles are seen as bright red branching lines, approximately two-thirds or three-fourths the diameter of the venules.

Macula

This is an area of the retina located temporal to the disk; it is void of visible vessels. The fovea is an area of depression approximately 1.5 mm in diameter (similar to the optic disk) in the center of the macula. The foveola is a tiny pit located in the center of the fovea. These areas correspond to central vision.

Background

The background fundus is red; there is some variation in the color, depending on the amount of individual pigmentation and the visibility of the choroidal vessels beneath the retina.

Pearls

1. Fundal examination should be an integral part of any eye examination.
2. The cup-to-disk ratio is slightly larger in the African American population.
3. The normal fundus should be void of any hemorrhages, exudates, or tortuous vasculature.

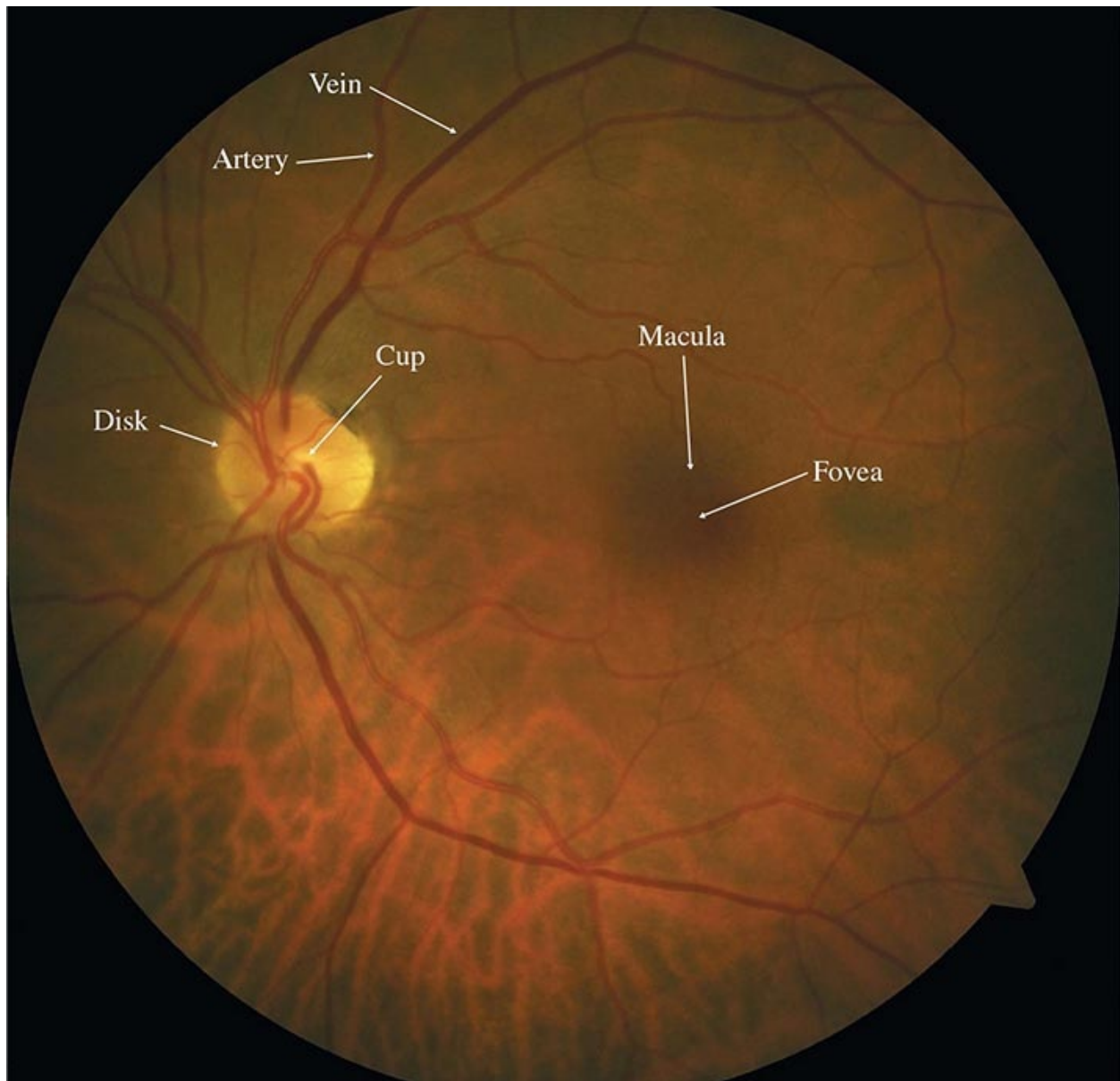


FIGURE 3.1 ■ Normal Fundus. The disk has sharp margins and is normal in color, with a small central cup. Arterioles and venules have normal color, sheen, and course. Background is in normal color. The macula is enclosed by arching temporal vessels. The fovea is located by a central pit. (Photo contributor: Jeffrey Goshe, MD.)

AGE-RELATED MACULAR DEGENERATION

Clinical Summary

Age-related macular degeneration increases in incidence with each decade over

50 and is evidenced by accumulation of either hard drusen (small, discrete, round, punctate nodules) or soft drusen (larger, pale yellow or gray, without discrete margins that may be confluent). Most patients with drusen have good vision, although there may be decreased visual acuity and distortion of vision. There may be associated pigmentary changes and atrophy of the retina. Vision may slowly deteriorate if atrophy occurs.

Patients with early or late degenerative changes of the macula are at risk of developing choroidal neovascularization (CNV), which is associated with distortion of vision, blind spots, and decreased visual acuity. Macular appearance may show dirty gray lesions, hemorrhage, retinal elevation, and exudation.

Management and Disposition

Patients with drusen need ophthalmologic evaluation every 6 to 12 months or sooner if visual distortion or decreasing visual acuity develops. If a patient complains of deterioration of visual acuity or image distortion, prompt ophthalmic evaluation is warranted.

Pearls

1. Age-related macular degeneration is the leading cause of blindness in the United States in patients older than age 65 years.
2. Patient may have normal peripheral vision.
3. Untreated CNV can lead to visual loss within a few days.
4. Patients frequently complain of distortion with CNV.

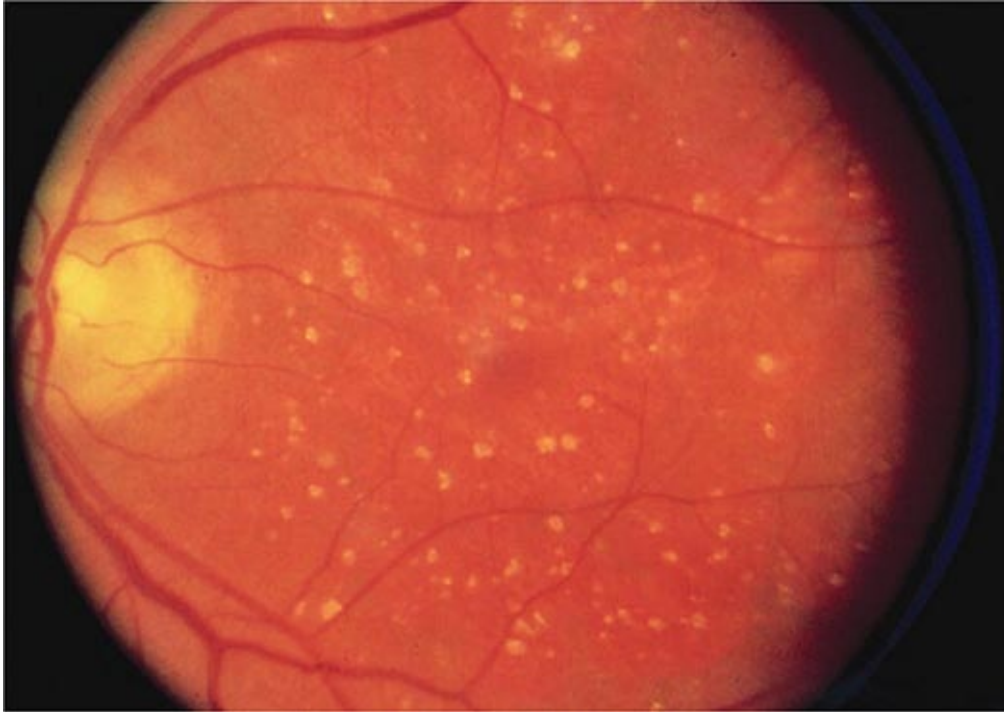


FIGURE 3.2 ■ Age-Related Macular Degeneration, Drusen. Typical macular drusen and retinal pigment epithelial (RPE) atrophy (scalloped pigment loss) in age-related macular degeneration. (Photo contributor: Richard E. Wyszynski, MD.)



FIGURE 3.3 ■ Age-Related Macular Degeneration, Drusen. Drusen are clustered in the center of the macula. (Photo contributor: Richard E. Wyszynski, MD.)



FIGURE 3.4 ■ Age-Related Macular Degeneration. Hemorrhage seen beneath the retina in association with subretinal neovascularization. Note the retinal vessels are superficial to the hemorrhage, which lies just beneath the retina. (Photo contributor: Richard E. Wyszynski, MD.)

EXUDATE

Clinical Summary

Hard exudates are refractile, yellowish deposits with sharp margins composed of fat-laden macrophages and serum lipids. Occasionally the lipid deposits form a partial or complete ring (called a circinate ring) around the leaking area of pathology. If the lipid leakage is located near the fovea, a spoke or star-type distribution of the hard exudates may be seen.

Cotton wool spots, or soft “exudates,” are actually microinfarctions of the retinal nerve fiber layer, and appear white with soft or fuzzy edges.

Inflammatory exudates are secondary to retinal or chorio-retinal inflammation.

Hard exudation and cotton wool spots are associated with vascular diseases such as diabetes mellitus, hypertension, and collagen vascular diseases but can be seen with papilledema and other intrinsic ocular conditions. Inflammatory exudates are seen in patients with such diseases as sarcoidosis and

toxoplasmosis.

Management and Disposition

Routine referral for ophthalmologic and medical workup is appropriate.

Pearl

1. Hard exudates that are intraretinal may easily be confused with drusen occurring near Bruch membrane, which separates the retina from the choroid.



FIGURE 3.5 ■ Hard Exudates. Collection of yellow lipid deposits with sharp margins are seen. Also seen are dot-blot hemorrhages and copper wiring, which are more typical in diabetes mellitus and hyper-tension, respectively. (Photo contributor: Jeffrey Goshe, MD.)

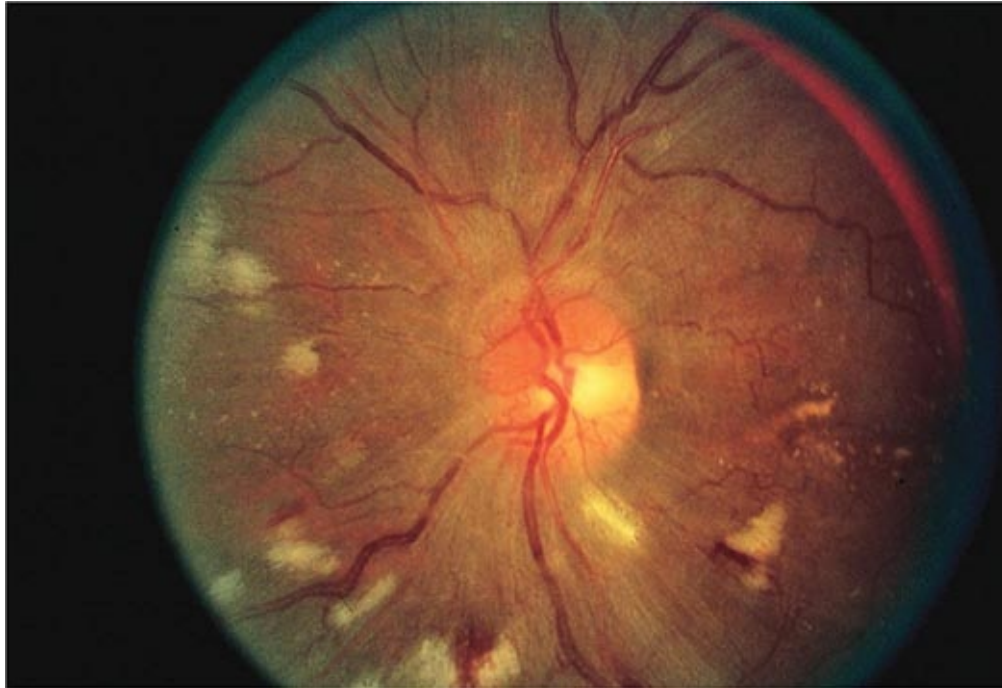


FIGURE 3.6 ■ Cotton Wool Spots. White lesions with fuzzy margins, seen here approximately one-fifth to one-fourth disk diameter in size. Orientation of cotton wool spots generally follows the curvilinear arrangement of the nerve fiber layer. Intraretinal hemorrhages and intraretinal vascular abnormalities are also present. (Photo contributor: Richard E. Wyszynski, MD.)

ROTH SPOTS

Clinical Summary

Roth spots are retinal hemorrhages with a white or yellow center. They may be seen in patients with a host of diseases such as anemia, leukemia, multiple myeloma, diabetes mellitus, collagen vascular disease, other vascular diseases, intracranial hemorrhage in infants, septic retinitis, and carcinoma. Flame-shaped or splinter hemorrhages or dot-blot hemorrhages may resemble Roth spots.

Management and Disposition

Routine referral for general medical evaluation is appropriate.

Pearls

1. Roth spots are not pathognomonic for any particular disease process and can represent a variety of clinical conditions.
2. These lesions represent red blood cells surrounding inflammatory cells.

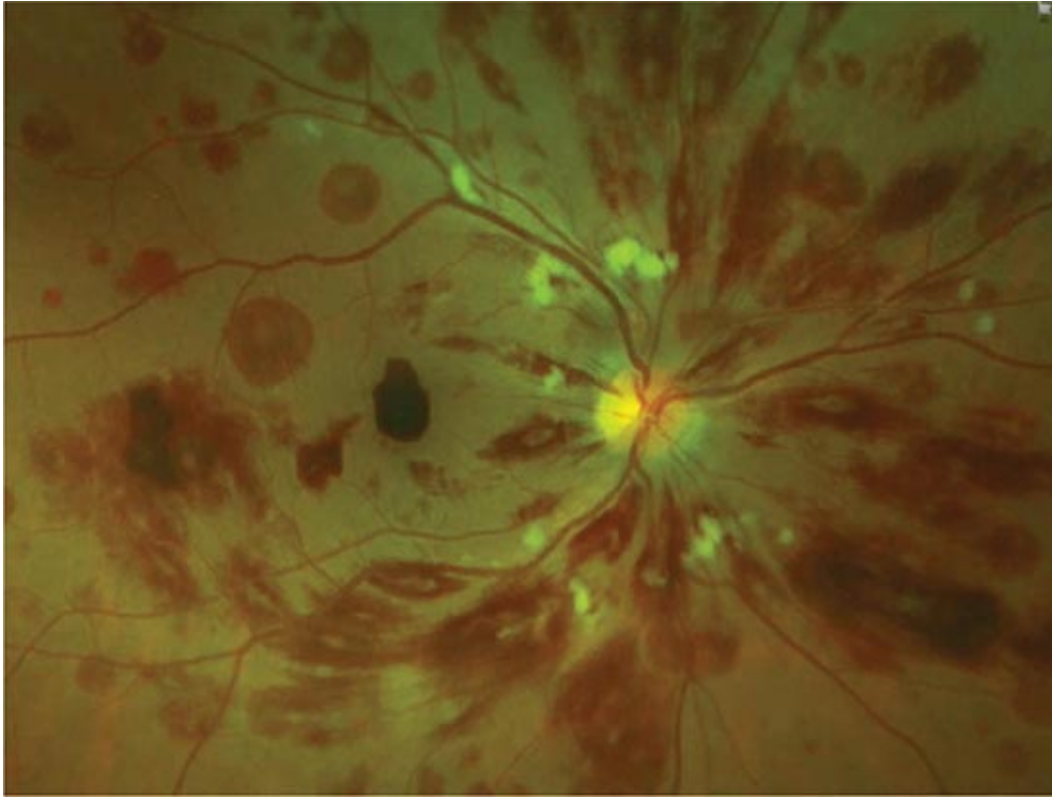


FIGURE 3.7 ■ Roth Spots. Multiple retinal hemorrhages with pale centers are seen. (Photo contributor: Tal Rubinstein, MD.)

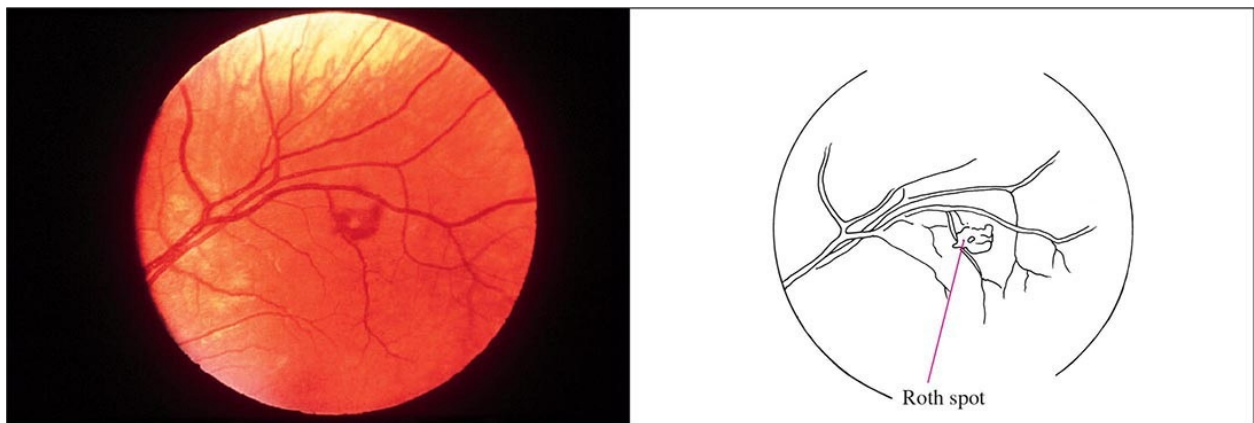


FIGURE 3.8 ■ Roth Spot. Retinal hemorrhage with pale center. (Photo contributor: William E. Cappaert, MD.)

EMBOLI

Clinical Summary

Plaques, if present, are often found at arteriolar bifurcations. Patients may have signs and symptoms of vascular disease such as carotid bruits or stenosis, aortic stenosis, aneurysms, or atrial fibrillation. Amaurosis fugax, a transient loss of vision often described as a curtain of darkness obscuring vision with sight restoration within a few minutes, may be present in the history.

Cholesterol emboli (Hollenhorst plaques), associated with generalized atherosclerosis, often from carotid atheroma, are bright, highly refractile plaques. Platelet emboli (carotid artery or cardiac thrombus) are white and very difficult to visualize. Calcific emboli (cardiac valvular disease) are irregular and white or dull gray and much less refractile.

Management and Disposition

Referral for routine general medical evaluation is appropriate unless the patient presents with signs or symptoms consistent with showering of emboli, transient ischemic attack, or cerebrovascular accident, in which case admission should be considered.

Pearls

1. Retinal emboli may produce a loss of vision, either transient or permanent in nature.
2. Arteriolar occlusion may occur in either a central or a peripheral branch location.
3. Occurrence of retinal emboli should prompt the clinician to search for an embolic source as the event may be a precursor to impending ischemic stroke.

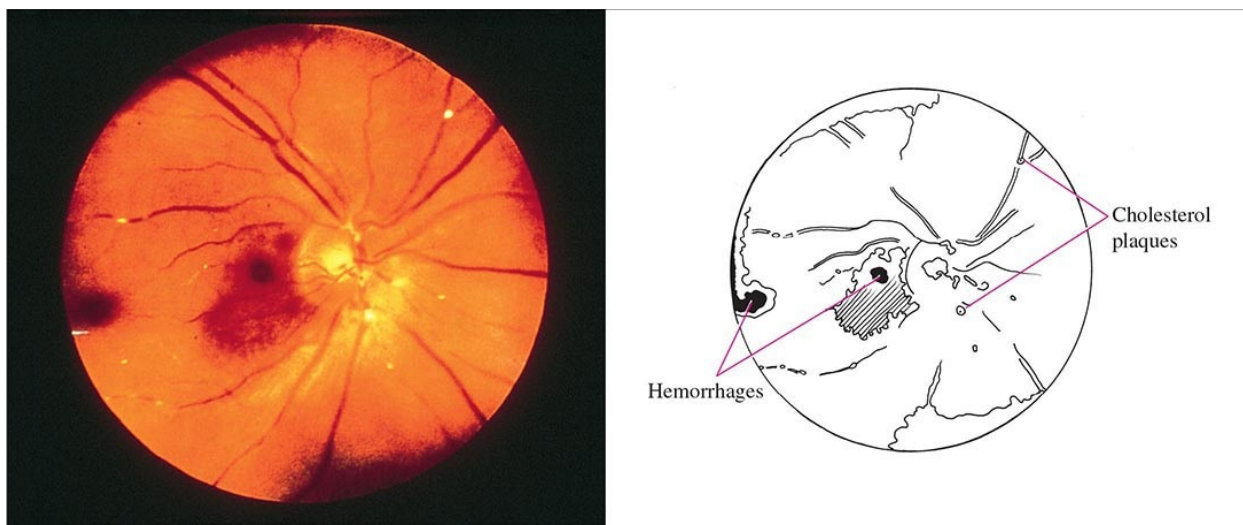


FIGURE 3.9 ■ Emboli. Refractile cholesterol plaques usually lodge at vessel bifurcations. (Photo contributor: William E. Cappaert, MD.)

CENTRAL RETINAL ARTERY OCCLUSION

Clinical Summary

In central retinal artery occlusion (CRAO), the typical patient experiences a sudden painless monocular loss of vision, either segmental or complete. Visual acuity may range from finger counting or light perception to complete blindness. Fundal findings may include the following: fundal paleness caused by retinal edema (the fovea does not have the edema and thus appears as a cherry-red spot); narrow and irregular retinal arterioles; and a “boxcar” appearance of the retinal venules.

Management and Disposition

Attempts to restore retinal blood flow may be beneficial if performed in a very narrow time window after the acute event. This may be accomplished by (1) decreasing intraocular pressure (IOP) with topical β -blocker eye drops or intravenous acetazolamide; or (2) ocular massage, applied with cyclic pressure on the globe for 10 seconds, followed by release and then repeated. Urgent consultation with an ophthalmologist is indicated to determine if more aggressive acute therapy (paracentesis) is warranted. However, such aggressive treatment rarely alters the poor prognosis. Medical evaluation and treatment of

associated findings may be warranted. Tissue plasminogen activator may be considered for lysis of an occluding thrombus.

Pearls

1. Visual decrement may be caused by a “low-flow” state (vs total occlusion). As this cannot be identified on presentation and can present hours later, immediate treatment and consultation are indicated regardless of the time of onset.
2. Sudden, painless monocular vision loss is typical.
3. CRAO may be associated with temporal arteritis. This diagnosis should be strongly considered in all patients presenting with signs and symptoms of CRAO who are older than 55 years.

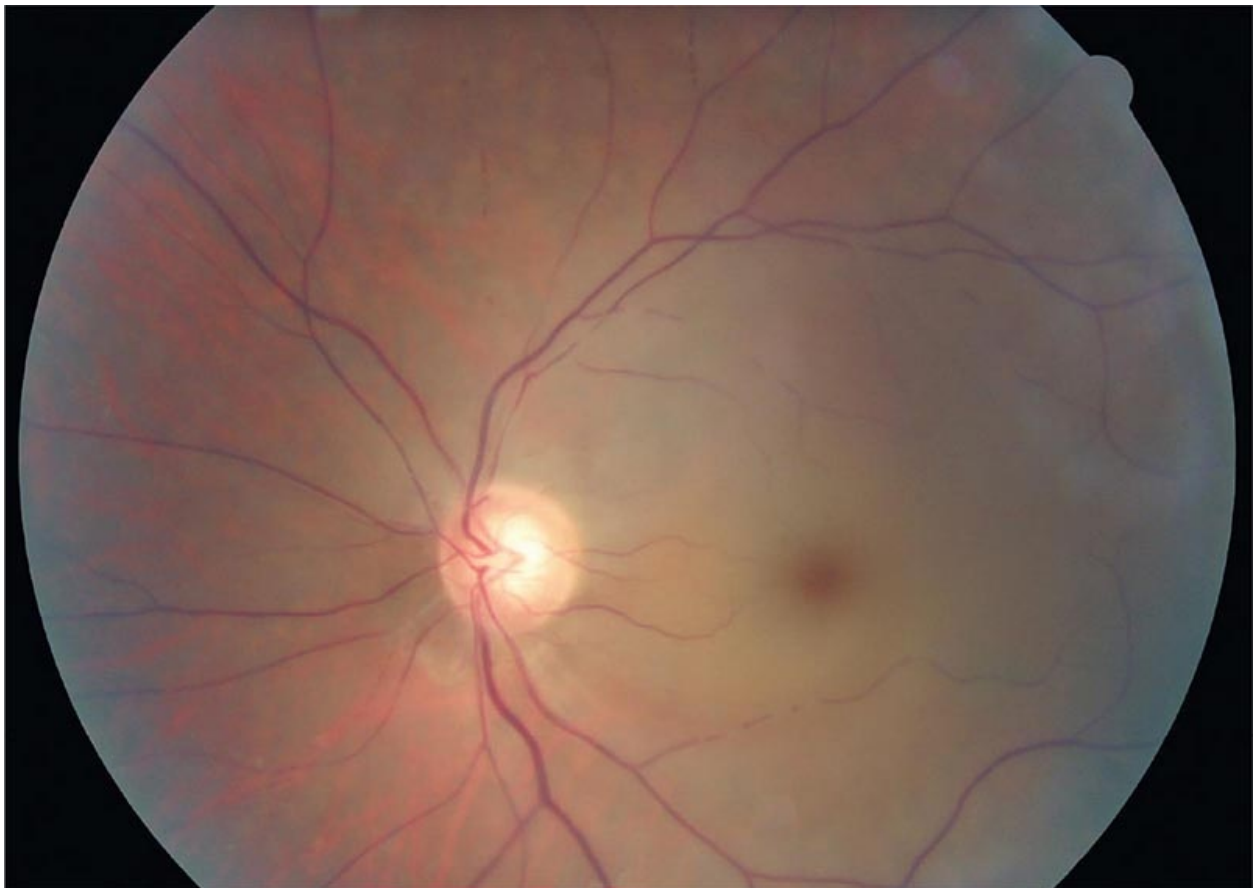


FIGURE 3.10 ■ Central Retinal Artery Occlusion. The retinal pallor caused by retinal edema is well demonstrated, contrasting with the “cherry-red spot” of the nonedematous fovea. Note the vascular narrowing and the “boxcar” appearance of the venules. (Photo contributor: Aaron Sobol, MD.)

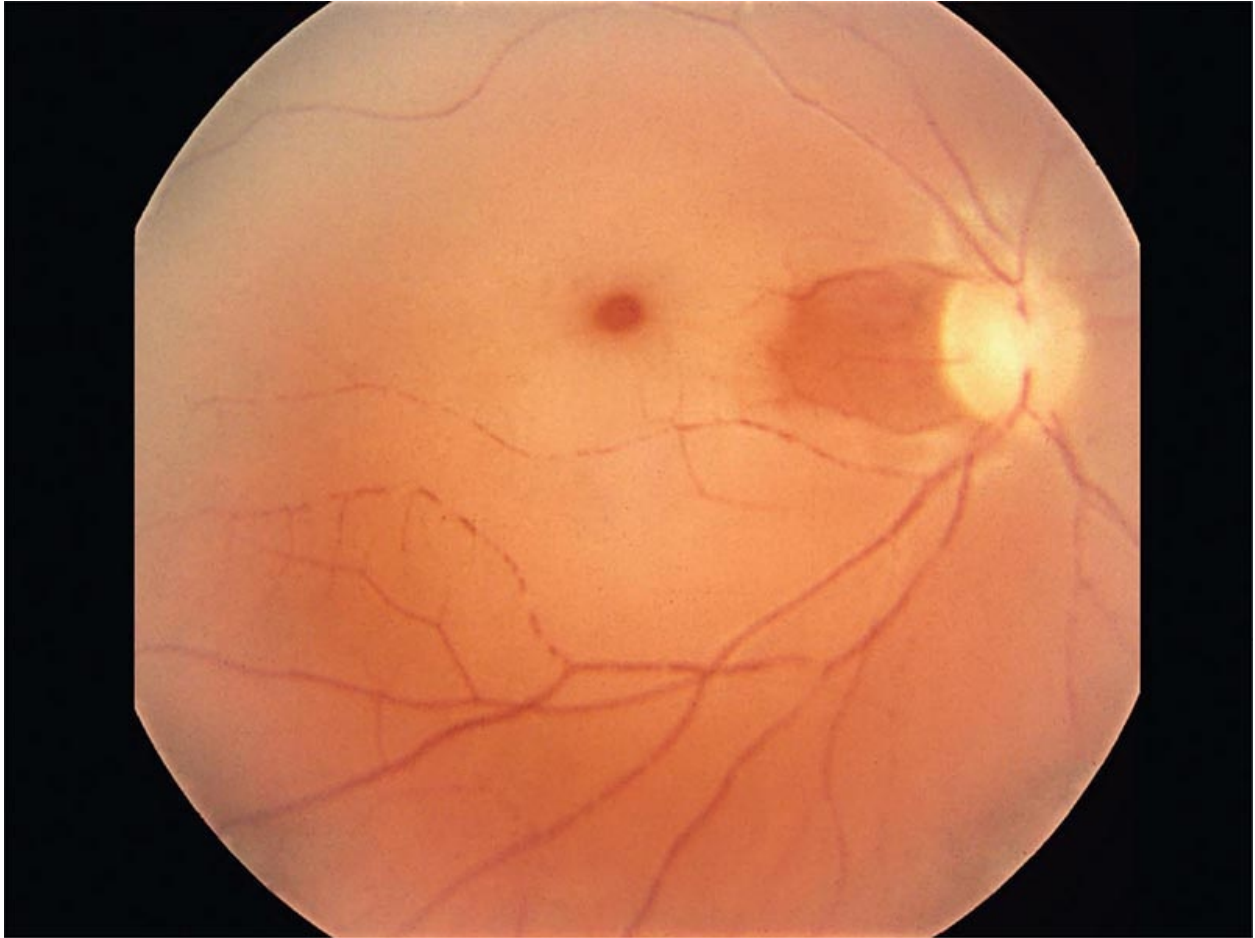


FIGURE 3.11 ■ Central Retinal Artery Occlusion with Cilioretinal Artery Sparing. “Hyperemia” of the fundus on the temporal side of the disk and sparing of the macular region owing to the presence of a patent cilioretinal artery. (Photo contributor: Thomas R. Hedges III, MD.)



FIGURE 3.12 ■ Cilioretinal Artery Occlusion. Absence of blood flow with “boxcar” appearance is seen against a backdrop of retinal ischemia (whitening) in this branch occlusion. The remainder of the retina appears normal. (Used with permission from The University of Iowa and EyeRounds.org.)

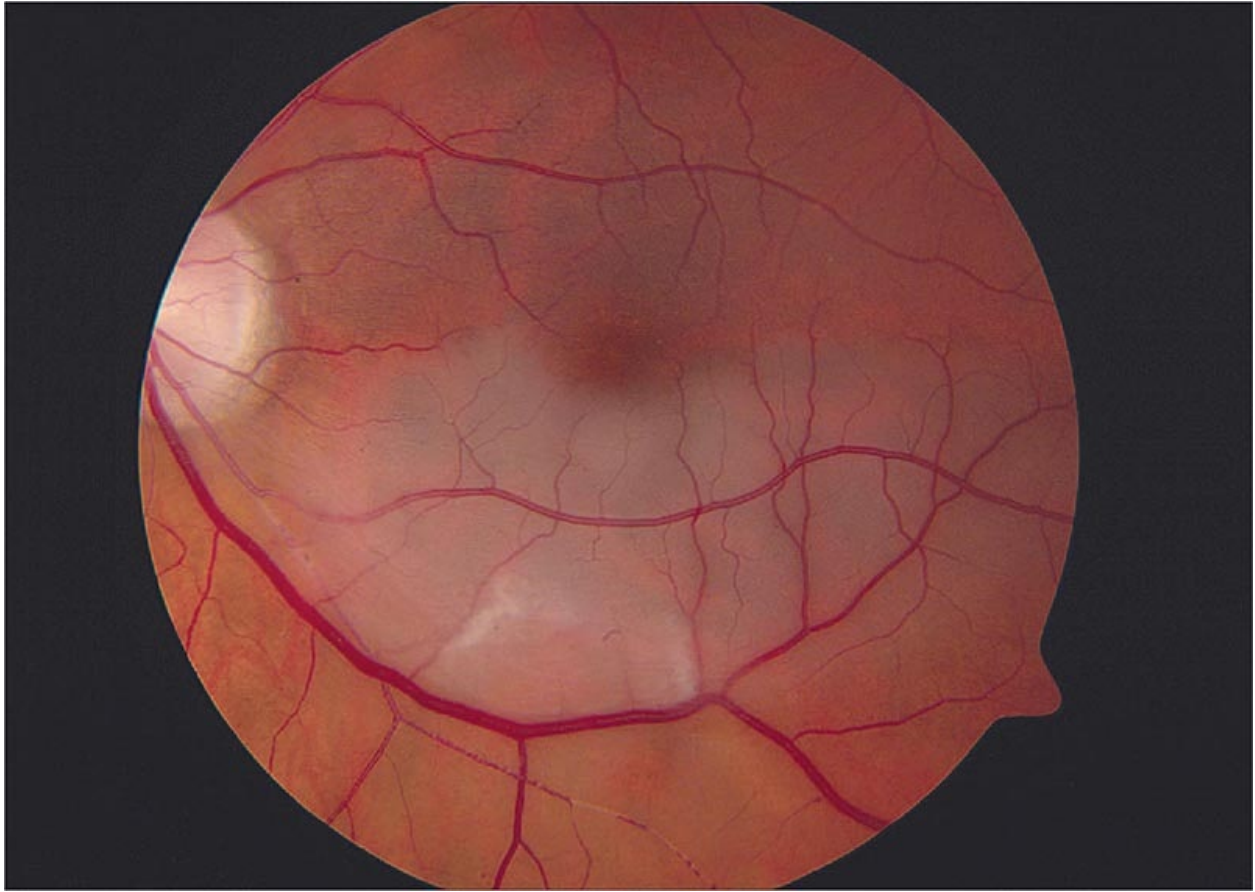


FIGURE 3.13 ■ Branch Central Retinal Artery Occlusion. Retinal whitening (owing to ischemia) and absence of blood flow in the inferotemporal branch retinal arteriole. (Photo contributor: Arun D. Singh, MD.)

CENTRAL RETINAL VEIN OCCLUSION

Clinical Summary

Patients are usually older individuals and complain of sudden, painless visual loss in one eye. The vision loss is usually not as severe as CRAO and may vary from normal to hand motion. Funduscopy in a classic, ischemic central retinal vein occlusion (CRVO) shows a “blood and thunder” fundus: hemorrhages (including flame, dot, or blot, preretinal, and vitreous) and dilation and tortuosity of the venous system. The arterial system often shows narrowing. The disk margin may be blurred. Cotton wool spots and edema may be seen.

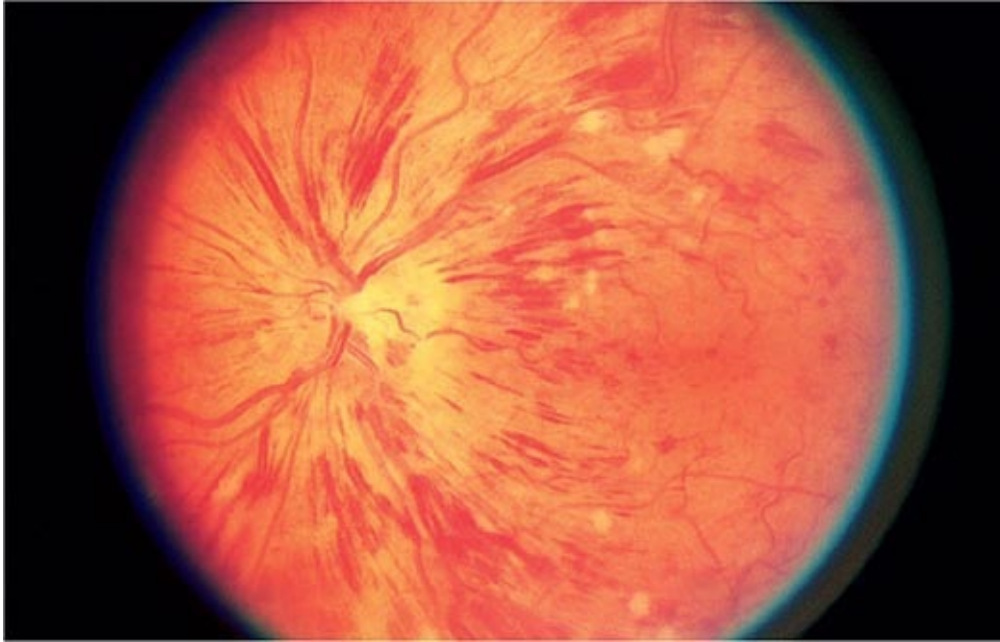


FIGURE 3.14 ■ Central Retinal Vein Occlusion. The amount of hemorrhage is the most striking feature in this photograph. Also note the blurred disk margin, the dilation and tortuosity of the venules, and the cotton wool spots. Retinal edema is suggested by blurring of the retinal details. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

Management and Disposition

Treatment is rarely effective in preventing or reversing the damage done by the occlusion and is directed toward systemic evaluation to identify and treat contributing factors, hopefully decreasing the chance of contralateral CRVO. Ophthalmologic evaluation is necessary to confirm the diagnosis, estimate the amount of ischemia, and follow the patient so as to minimize sequelae of possible complications such as neovascularization and neovascular glaucoma.

Pearls

1. Sudden, painless visual loss in one eye should be evaluated promptly to determine its etiology.
2. Look for the classic “blood and thunder” funduscopy findings.
3. Consider the differential diagnosis of acute *painful* (glaucoma, retrobulbar neuritis) versus *painless* vision loss (CRAO, anterior ischemic optic neuropathy, retinal detachment, sub-retinal neovascularization, and vitreous hemorrhage).

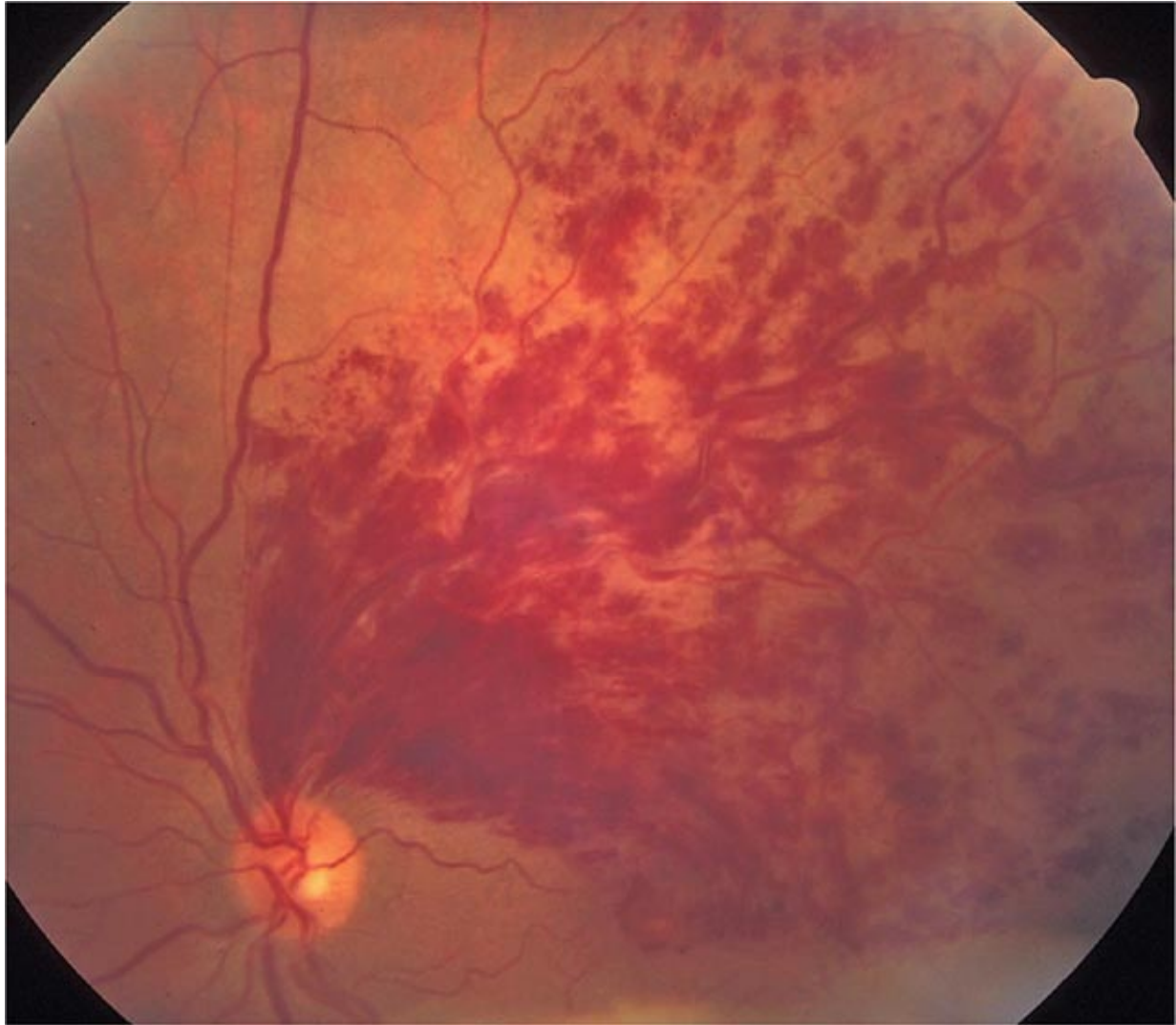


FIGURE 3.15 ■ Central Retinal Vein Branch Occlusion. The hemorrhage seen is limited to a sector of the fundus, indicating that a branch occlusion has occurred. There is less edema, and a large portion of the fundus is unaffected. (Photo contributor: Richard E. Wyszynski, MD.)

HYPERTENSIVE RETINOPATHY

Clinical Summary

Fundus changes that may be seen with hypertension include generalized and focal narrowing of arterioles, generalized arteriolar sclerosis (resembling copper or silver wiring), arteriovenous crossing changes, hemorrhages (usually flame-shaped), retinal edema and exudation, cotton wool spots, microaneurysms, and disk edema.

Diabetic retinopathy, many hematologic and vascular diseases, traumas, localized ocular pathology, and papilledema should all be considered.

Management and Disposition

The patient's hypertension should be appropriately treated, and a search for other end-organ damage should be considered. The patient should be referred for appropriate long-term blood pressure management.

Pearls

1. Hypertensive arteriolar findings may be reversible if organic changes have not occurred in the vessel walls.
2. Always consider hypertensive retinopathy in the differential diagnosis of papilledema.



FIGURE 3.16 ■ Severe Hypertensive Retinopathy. Marked papilledema, diffuse flame hemorrhages, and cotton wool spots with hard exudates in a patient with severe hypertensive disease. (Photo contributor: Jeffrey Goshe, MD.)

DIABETIC RETINOPATHY

Clinical Summary

The early ocular manifestations of diabetes mellitus are referred to as background diabetic retinopathy (BDR). Fundus findings include flame or splinter hemorrhages (located in the superficial nerve fiber layer) or dot and blot hemorrhages (located deeper in the retina), hard exudates, retinal edema, and microaneurysms. If these signs are located in the macula, the patient's visual acuity may be decreased or at risk of becoming compromised, requiring laser treatment. Proliferative diabetic retinopathy can show BDR changes plus cotton wool spots, intraretinal microvascular abnormalities, and venous beading. Proliferative diabetic retinopathy is demonstrated by neovascularization at the disk (NVD) or elsewhere (NVE). These require laser therapy owing to risk of severe visual loss from sequelae: vitreous hemorrhage, tractional retinal detachment, and severe glaucoma.

Many vascular and hematologic diseases—such as collagen vascular disease, sickle cell trait, hypertension, hypo-tension, anemia, leukemia, and inflammatory and infectious states—and ocular conditions can be associated with some or all of the above signs.

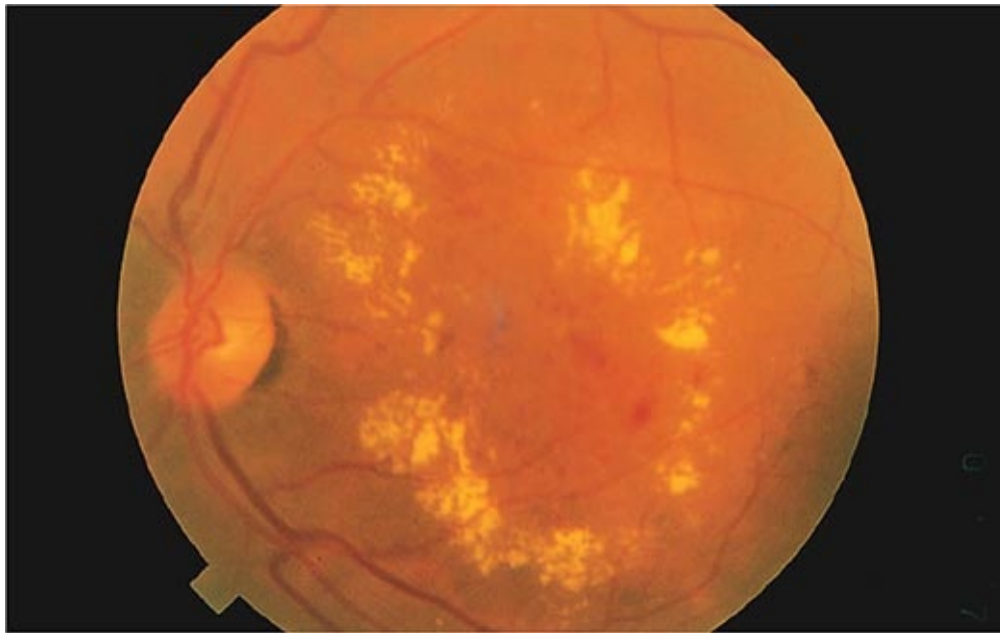


FIGURE 3.17 ■ Background Diabetic Retinopathy. An example of diabetic maculopathy with a typical circinate lipid ring. (Photo contributor: Richard E. Wyszynski, MD.)

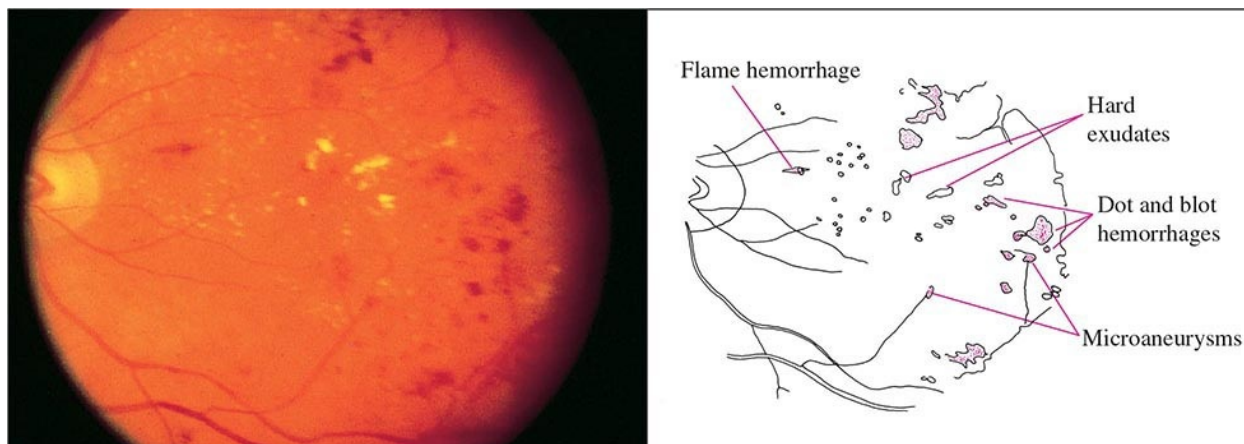


FIGURE 3.18 ■ Background Diabetic Retinopathy. Hard exudates, dot hemorrhages, blot hemorrhages, flame hemorrhages, and microaneurysms are present. Because these changes are located within the macula, this is classified as diabetic maculopathy. (Photo contributor: Richard E. Wyszynski, MD.)

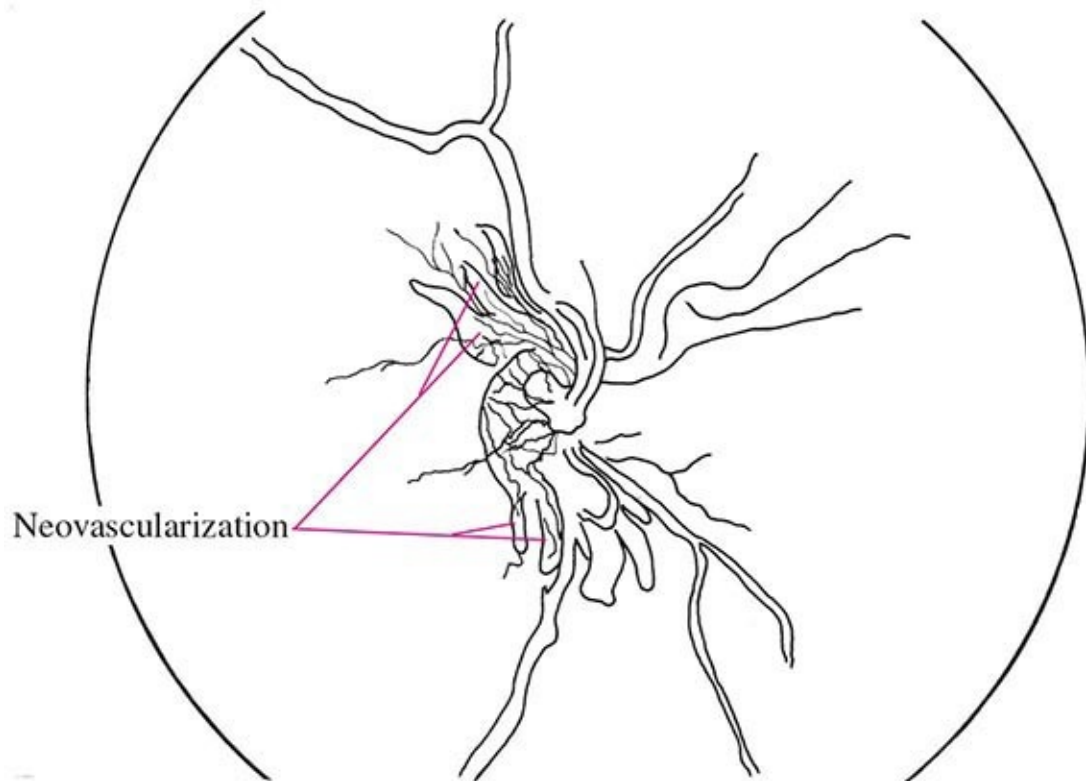


FIGURE 3.19 ■ Proliferative Diabetic Retinopathy. In addition to the signs seen in background and preproliferative diabetic retinopathy, neovascularization is seen here coming off the disk. (Photo

contributor: Richard E. Wyszynski, MD.)

Management and Disposition

Routine ophthalmologic referral for laser or surgical treatment is indicated.

Pearls

1. Periodic ophthalmologic evaluations are recommended for all diabetic patients.
2. Microaneurysms typically appear 10 years after the initial onset of diabetes, although they may appear earlier in patients with juvenile diabetes.
3. Control of blood sugar alone does not prevent the development of retinopathy.
4. Blurred vision can also occur from acute increases in serum glucose, causing lens swelling and a refractive shift even in the absence of retinopathy.

VITREOUS HEMORRHAGE

Clinical Summary

Patients may complain of floaters followed by the sudden loss or deterioration of vision in the affected eye, although bilateral hemorrhage can occur. The red reflex is diminished or absent, and the retina is obscured because of the bleeding. Large sheets or three-dimensional collections of red to red-black blood may be detected.

Multiple underlying etiologies include proliferative diabetic retinopathy, retinal or vitreous detachments, hematologic diseases, trauma (ocular or shaken impact syndrome), subarachnoid hemorrhage (SAH), collagen vascular disease, infections, macular degeneration, and tumors.

Management and Disposition

Refer to an ophthalmologist and an appropriate physician for associated conditions. Ophthalmic observation, photocoagulation, and surgery are all therapeutic options. Bed rest may help to increase visualization of the fundus.

Pearl

1. The patient's vision may improve somewhat after a period of sitting or standing as the blood layers out.

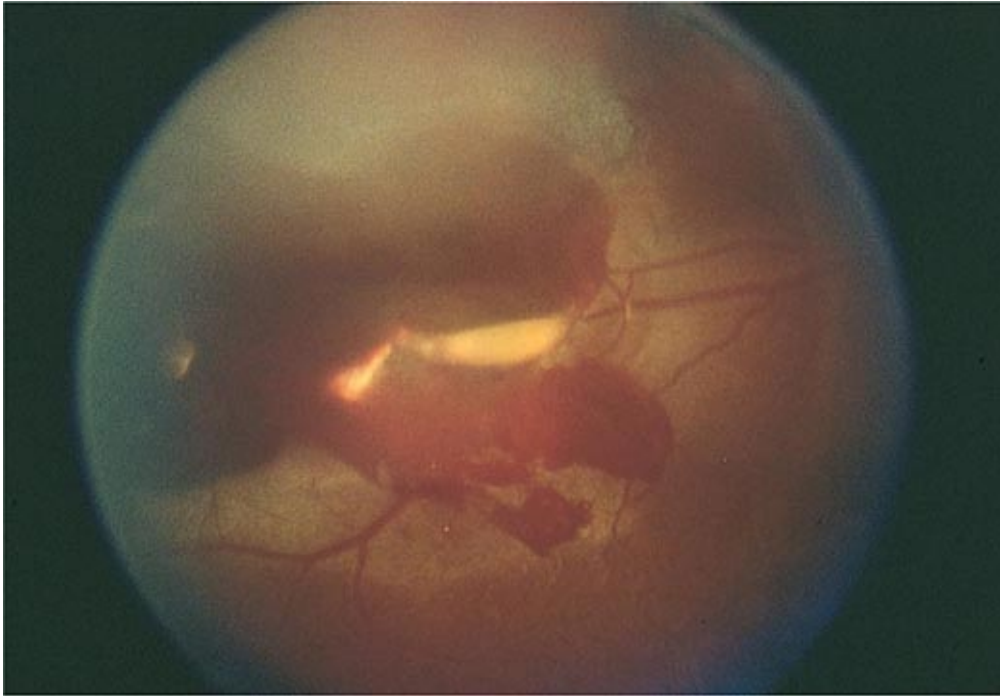


FIGURE 3.20 ■ Vitreous Hemorrhage. Large amount of vitreous hemorrhage associated with metallic intraocular foreign body. The large quantity of blood obscures visualization of retinal details. (Photo contributor: Richard E. Wyszynski, MD.)



FIGURE 3.21 ■ Vitreous Hemorrhage. A smaller amount of vitreous hemorrhage is more easily photographed. Gravitational effect on the vitreous blood creates the appearance of a flat meniscus (keel-shaped blood) in this patient with vitreous hemorrhage associated with proliferative diabetic retinopathy. (Photo contributor: Richard E. Wyszynski, MD.)

RETINAL DETACHMENT

Clinical Summary

Patients often complain of monocular, decreased visual function and may describe a shadow or curtain descending over the eye. Other complaints include cloudy or smoky vision, floaters, or momentary flashes of light. Monocular visual field defects may be noted, and central visual acuity is diminished with macular involvement. Fundal examination may reveal a billowing or tentlike elevation of retina compared with adjacent areas. The elevated retina often appears gray. Retinal holes and tears may be seen, but often the holes, tears, and retinal detachment cannot be seen without indirect ophthalmoscopy.

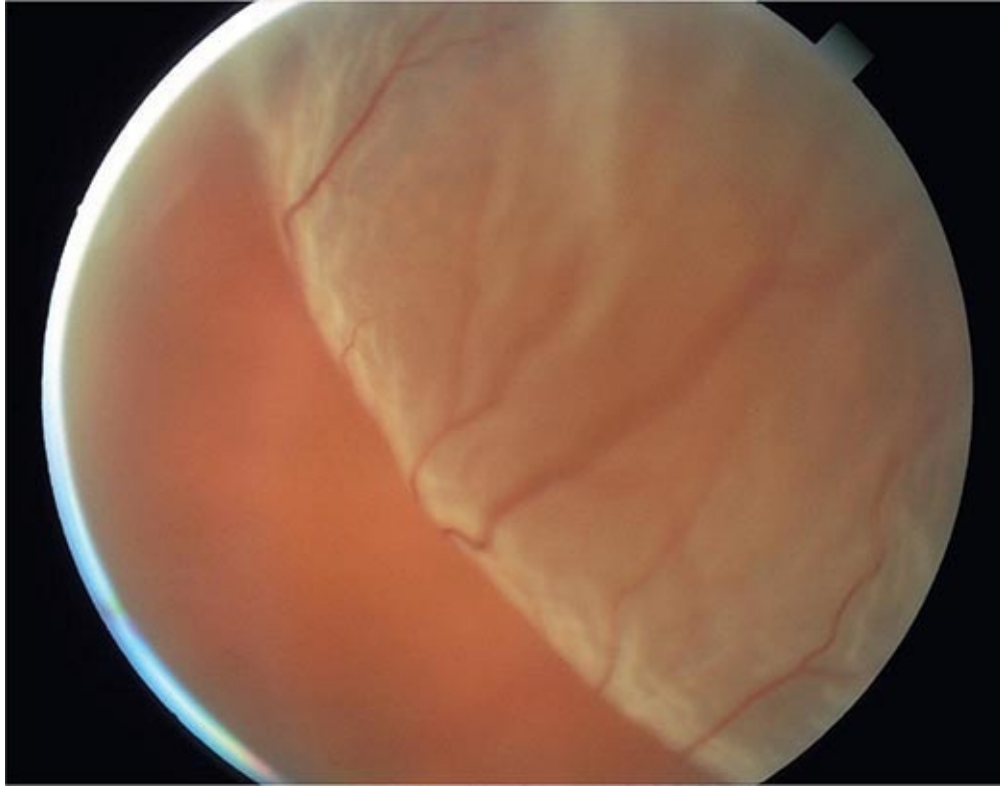


FIGURE 3.22 ■ Retinal Detachment. A fold of detached retina is seen drooping well in front of the posterior pole, giving the appearance of a “three-dimensional” fundus. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

Retinal detachments caused by retinal tears or holes can be associated with trauma, previous ocular surgery, nearsightedness, family history of retinal detachment, and Marfan disease. Retinal detachments caused by traction on the retina by an intraocular process can be because of systemic influences in the eye, such as diabetes mellitus or sickle cell trait. Occasionally retinal detachments are caused by tumors or exudative processes that elevate the retina. Symptoms of “light flashes” may occur with vitreous changes in the absence of retinal pathology. Patients may note flashes of light occurring only in a darkened environment because of the mechanical stimulation of the retina from the extraocular muscles, usually in a nearsighted individual.

Management and Disposition

Urgent ophthalmologic evaluation and treatment are warranted.

Pearls

1. Often patients have had sensation of flashes of light that occur in a certain area of a visual field in one eye, corresponding to the pathologic pulling on the corresponding retina.
2. Visual loss may be gradual or sudden.

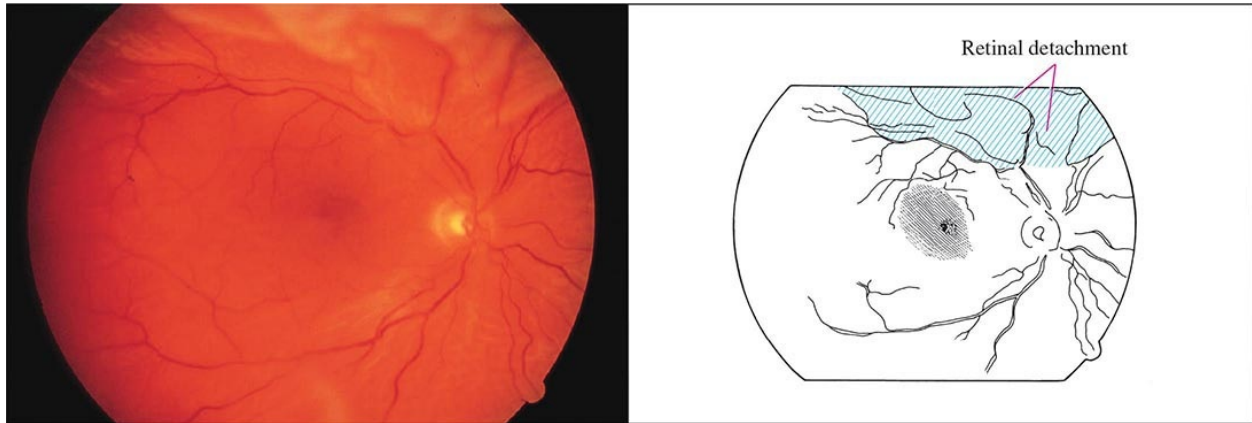


FIGURE 3.23 ■ Retinal Detachment. Undulating, out-of-focus, elevated retina is seen with few vessels in focus. (Photo contributor: Richard E. Wyszynski, MD.)

CYTOMEGALOVIRUS RETINITIS

Clinical Summary

Patients may complain of the gradual and usually painless onset of the following visual sensations: floaters, scintillating scotomas (quivering blind spots), decreased peripheral visual field, and metamorphopsia (wavy distortion of vision). Cytomegalovirus (CMV) infiltrates appear as focal, small (but may be larger, confluent) white lesions in the retina that look like cotton wool spots. CMV is a necrotizing virus that is spread hematogenously, so that damage is concentrated in the retina adjacent to the major vessels and the optic disk. Often hemorrhage is involved with significant retinal necrosis (dirty white with a granular appearance), giving the “pizza pie” or “cheese and ketchup” appearance. Optic nerve involvement and retinal detachments can be present.

The differential includes other infections such as toxoplasmosis, other herpesviruses, syphilis, and occasionally other opportunistic infections.

Management and Disposition

Reversal, if possible, of immunosuppression; antiviral agents have been used effectively to treat this condition.

Pearls

1. HIV retinopathy consists of scattered retinal hemorrhages and scattered, multiple cotton wool spots that resolve over time, whereas CMV lesions will typically progress.
2. Although exposure to the CMV virus is widespread, the virus rarely produces a clinically recognized disease in nonimmunosuppressed individuals.

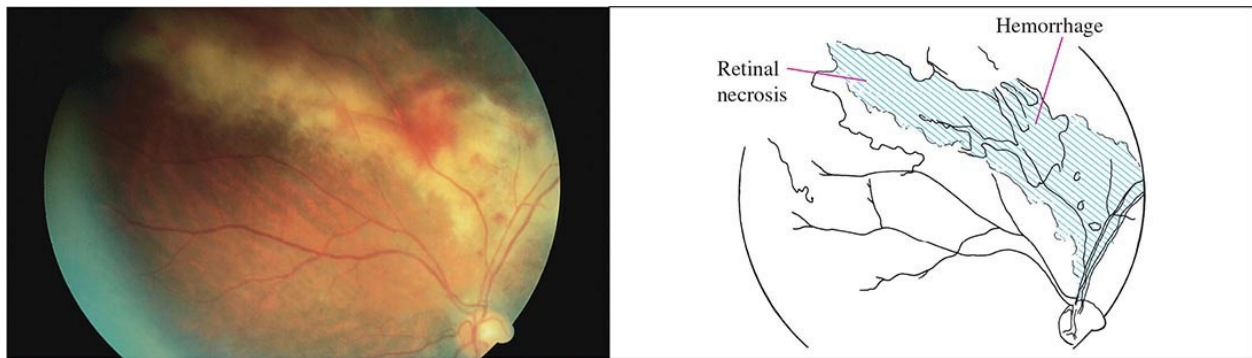


FIGURE 3.24 ■ CMV Retinitis. “Pizza pie” or “cheese and ketchup” appearance is demonstrated by hemorrhages and the dirty, white, granular-appearing retinal necrosis adjacent to major vessels. (Photo contributor: Richard E. Wyszynski, MD.)



FIGURE 3.25 ■ CMV Retinitis. Hemorrhages and dirty white retinal necrosis are seen extending from the optic disk to the peripheral fundus. (Photo contributor: Jeffrey Goshe, MD.)

PAPILLEDEMA

Clinical Summary

Papilledema involves swelling of the optic nerve head, usually in association with elevated intracranial pressure. The optic disks are hyperemic with blurred disk margins; the venules are dilated and tortuous. The optic cup may be obscured by the swollen disk. There may be flame hemorrhages and infarctions (white, indistinct cotton wool spots) in the nerve fiber layer and edema in the surrounding retina.

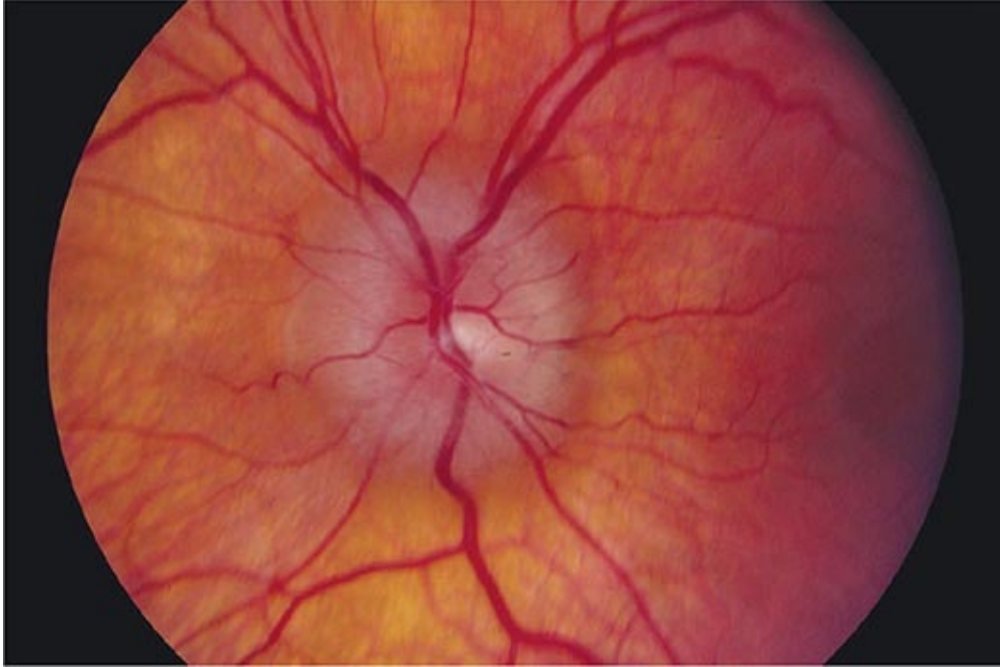


FIGURE 3.26 ■ Papilledema. Note blurred disk margins and congested disk. (Photo contributor: Arun D. Singh, MD.)

Ocular inflammation (eg, papillitis), tumors or trauma, central retinal artery or vein occlusion, optic nerve drusen, and marked hyperopia may present with similar findings.

Management and Disposition

Expeditious ophthalmologic and medical evaluation is warranted.

Pearls

1. The top of a swollen disk and the surrounding unaffected retina will not both be in focus on the same setting on direct ophthalmoscopy.
2. Papilledema is a bilateral process, although it may be slightly asymmetric. A unilateral swollen disk suggests a localized ocular or orbital process.
3. Vision is usually normal acutely, although the patient may complain of transient visual changes. The blind spot is usually enlarged.
4. Diplopia from 6th cranial nerve palsy can be associated with increased intracranial pressure and papilledema.

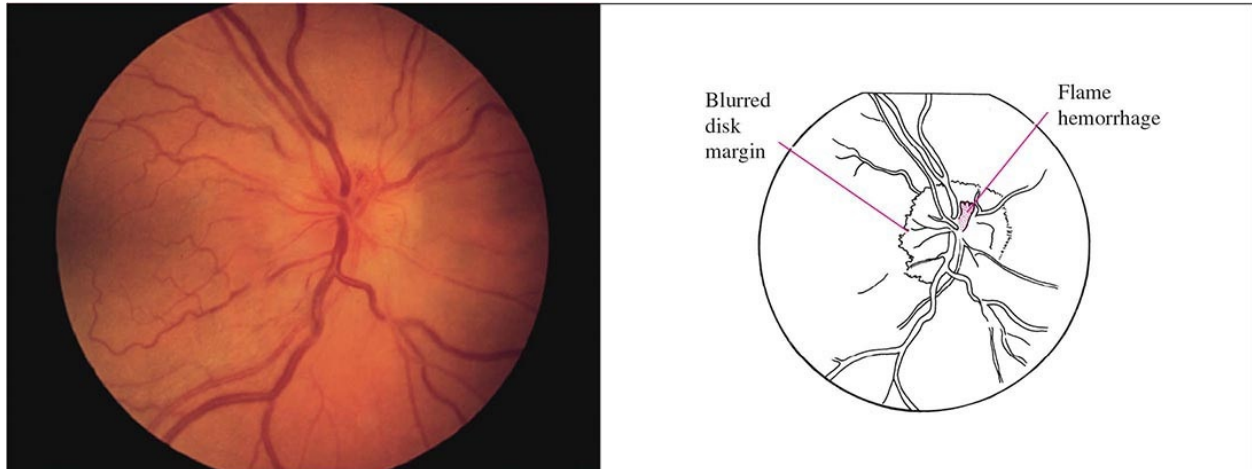


FIGURE 3.27 ■ Papilledema. Disk is hyperemic and swollen with loss of sharp margins. The venules are dilated and tortuous. The cup is obscured. A small flame hemorrhage is seen at 12- to 1-o'clock position on the disk margin. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

OPTIC NEURITIS

Clinical Summary

Most cases of optic neuritis are retrobulbar and involve no changes in the fundus, or optic disk, during the acute episode. With time, variable optic disk pallor may develop. Typical retrobulbar optic neuritis presents with sudden or rapidly progressing monocular vision loss in patients younger than 50 years. There is a central visual field defect that may extend to the blind spot. Pain on movement of the globe is common. The pupillary light response is diminished in the affected eye. Over time the vision improves partially or completely; minimal or severe optic atrophy may develop. Papillitis, inflammation of the intraocular portion of the optic nerve, will accompany disk swelling, with a few flame hemorrhages and possible cells in the vitreous.

Optic neuritis must be differentiated from papilledema (bilateral disk swelling, typically with no acute visual loss with the exception of transient visual changes), ischemic neuropathy (pale, swollen disk in an older individual with sudden monocular vision loss), tumors, and metabolic or endocrine disorders. Most cases of optic neuritis are of unknown etiology. Some known causes of optic neuritis include demyelinating disease, infections (including viral, syphilis, tuberculosis, sarcoidosis), or inflammations from contiguous

structures (sinuses, meninges, orbit).

Management and Disposition

Treatment is controversial; often none is recommended. Oral steroids may worsen prognosis in certain cases. Intravenous steroids should be considered after consultation with an ophthalmologist.

Pearls

1. Monocular vision loss with pain on palpation of the globe or with eye movement and decreased color vision and color desaturation with loss of contrast are clinical clues to the diagnosis.
2. Sudden or rapidly progressing central vision loss is characteristic.
3. Most cases of acute optic neuritis are retrobulbar. Thus, ophthalmoscopy shows a normal fundus.
4. Suspect temporal arteritis in older patients.

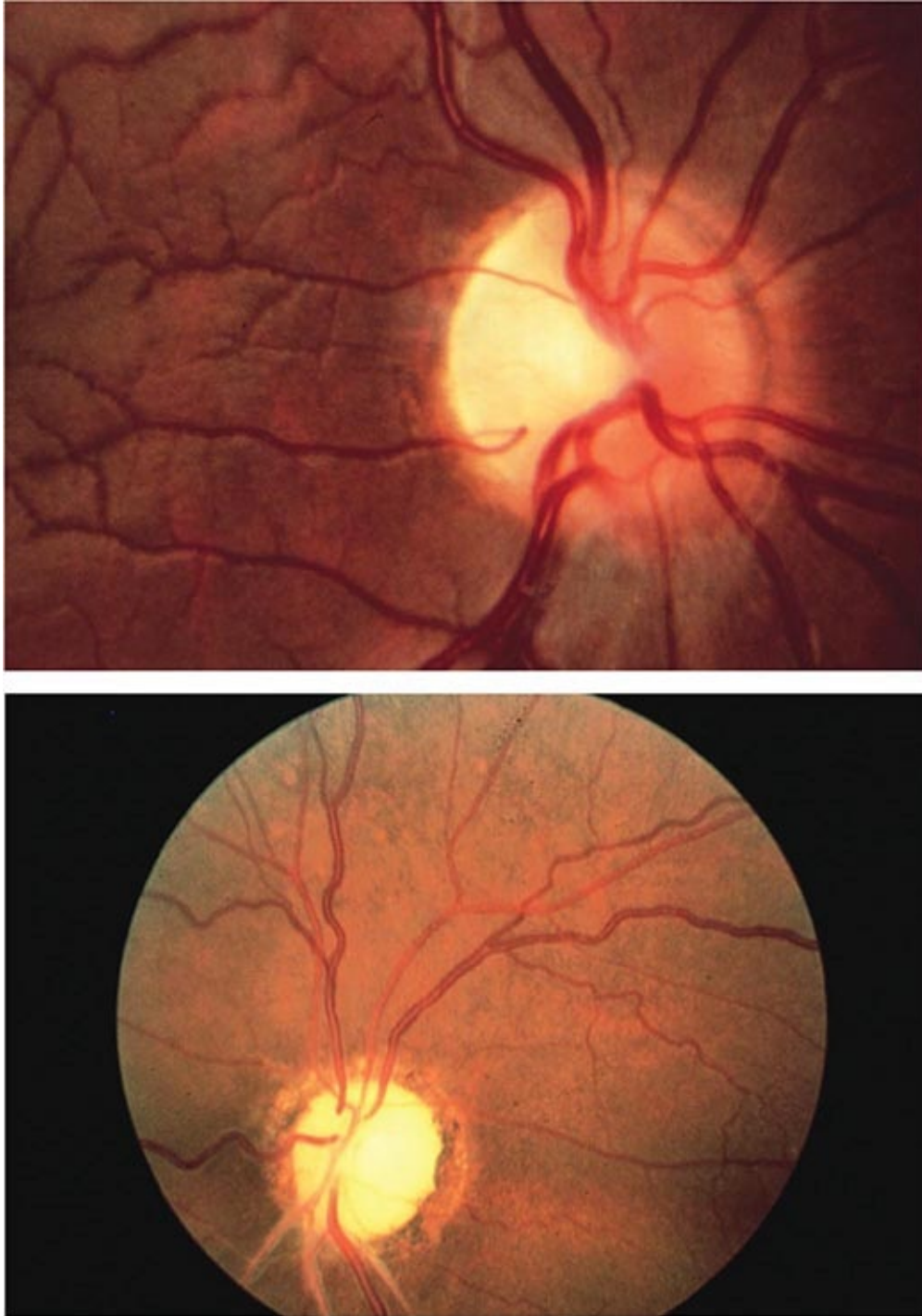


FIGURE 3.28 ■ Optic Nerve Pallor. Optic nerve pallor, either segmental (*top*) or generalized (*bottom*), is a nonspecific change that may be associated with a previous episode of optic neuritis or other insults to the optic nerve. (Photo contributor: Richard E. Wyszynski, MD.)

ANTERIOR ISCHEMIC OPTIC NEUROPATHY

Clinical Summary

Anterior ischemic optic neuropathy (AION) presents with a sudden loss of visual field (often altitudinal), usually involving fixation, in an older individual. The loss is usually stable after onset, with no improvement, and only occasionally, progressive over several days to weeks. In contrast to the hyper-emic appearance that usually accompanies optic nerve head edema, pale disk swelling is present involving a sector or the full disk, sometimes with accompanying flame hemorrhages. The cup-to-disk ratio is typically small (0.1-0.2) bilaterally.

The common, nonarteritic causes of AION (probably arteriosclerosis) need to be differentiated from arteritic (AAION) ones, such as giant cell arteritis (GCA). If untreated, the latter will involve the other eye in 75% of cases, often in a few days to weeks. These elderly individuals often have weight loss, masseter claudication, weakness, myalgias, elevated sedimentation rate, and painful scalp, temples, or forehead.

Management and Disposition

Routine ophthalmologic and medical evaluation is appropriate.

Pearls

1. Consider AION in an elderly patient with sudden, usually painless, visual field loss.
2. Rule out GCA. Patients tend to be older (age > 55 years) and may have associated CRAO or cranial nerve palsies (III, IV, or VI) with diplopia.

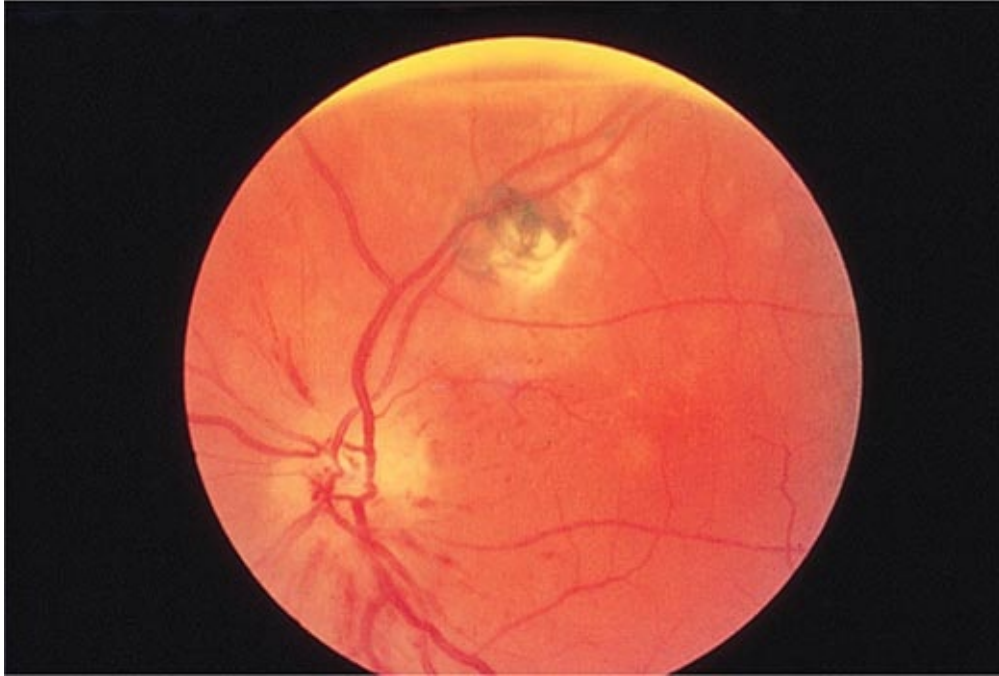


FIGURE 3.29 ■ Anterior Ischemic Optic Neuropathy. Pale disk swelling and flame hemorrhages are present. This patient also has an unrelated retinal scar owing to toxoplasmosis. (Photo contributor: William E. Cappaert, MD.)

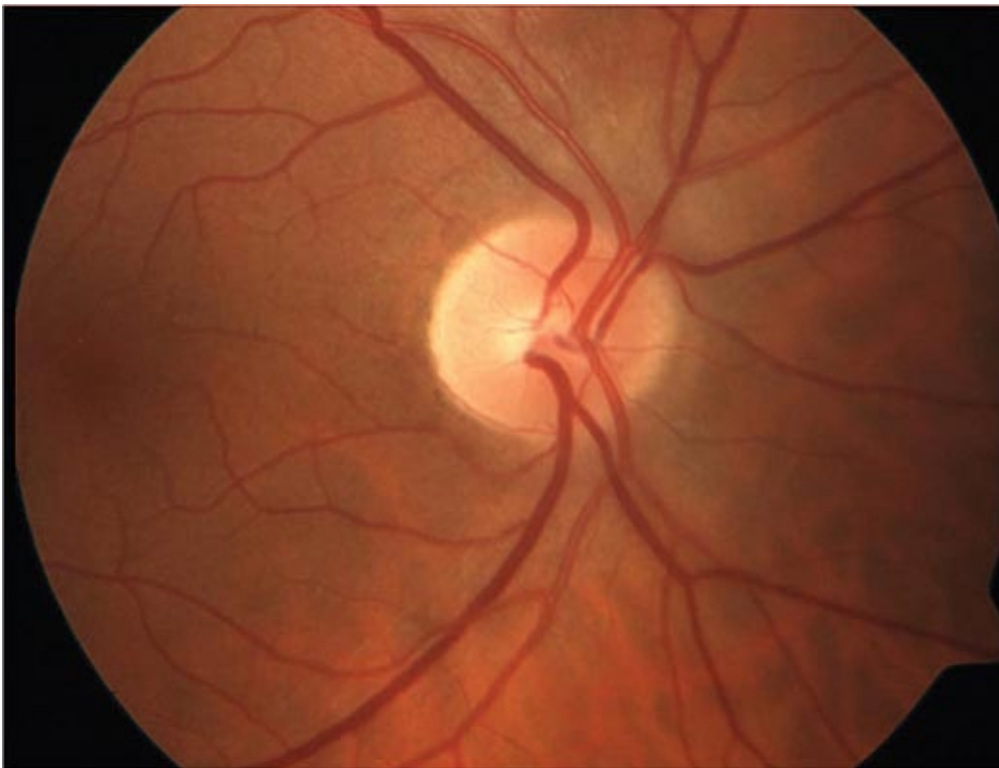


FIGURE 3.30 ■ Nonarteritic Anterior Ischemic Optic Neuropathy (NAAION). Pallor of the entire temporal disk is seen in a patient with NAAION. (Photo contributor: Jeffrey Goshe, MD.)



FIGURE 3.31 ■ Arteritic Anterior Ischemic Optic Neuropathy (AAION). Characteristic “pallid disk edema” of AAION in a patient with significant vision loss with confirmed giant cell arteritis. (Used with permission from The University of Iowa and EyeRounds.org.)

GLAUCOMA

Clinical Summary

Acute narrow or closed-angle glaucoma (ACG) results from a physical impedance of aqueous humor outflow. Symptoms range from colored halos around lights and blurred vision to severe pain (described as a headache or brow ache) with nausea and vomiting. IOP is markedly elevated. Perilimbal vessels are injected, the pupil is mid-dilated and poorly reactive to light, and the cornea may be hazy and edematous.

Two-thirds of glaucoma patients have open-angle glaucoma. Often they are asymptomatic. They may have a family history of glaucoma. Funduscopy may show asymmetric cupping of the optic nerves. The optic nerve may show notching, local thinning of tissue, or disk hemorrhage. Optic cups enlarge,

especially vertically, with progressive damage. Tissue loss is associated with visual field abnormalities. The IOP is often but not always greater than 21 mm Hg.

Management and Disposition

Acute ACG requires emergent ophthalmologic consultation and administration of medications to decrease IOP such as β -blocker drops (timolol), carbonic anhydrase inhibitors (acetazolamide), cholinergic-stimulating drops (pilocarpine), hyperosmotic agents (osmoglyn), and α -adrenergic agonists (apraclonidine). Open-angle glaucoma is treated with long-term ophthalmic evaluation and treatment with medications and laser or surgery.

Pearls

1. Nausea, vomiting, and headache may obscure the diagnosis. Use digital globe palpation routinely in patients with these complaints.
2. Open-angle glaucoma usually causes no symptoms other than gradual loss of vision.
3. Congenital glaucoma is rare. However, because of prognosis if diagnosis is delayed, consider congenital glaucoma in infants and children with tearing, photophobia, enlarged eyes, or cloudy corneas.
4. Asymmetric cupping, enlarged cups, and elevated IOP are hallmarks of open-angle glaucoma.

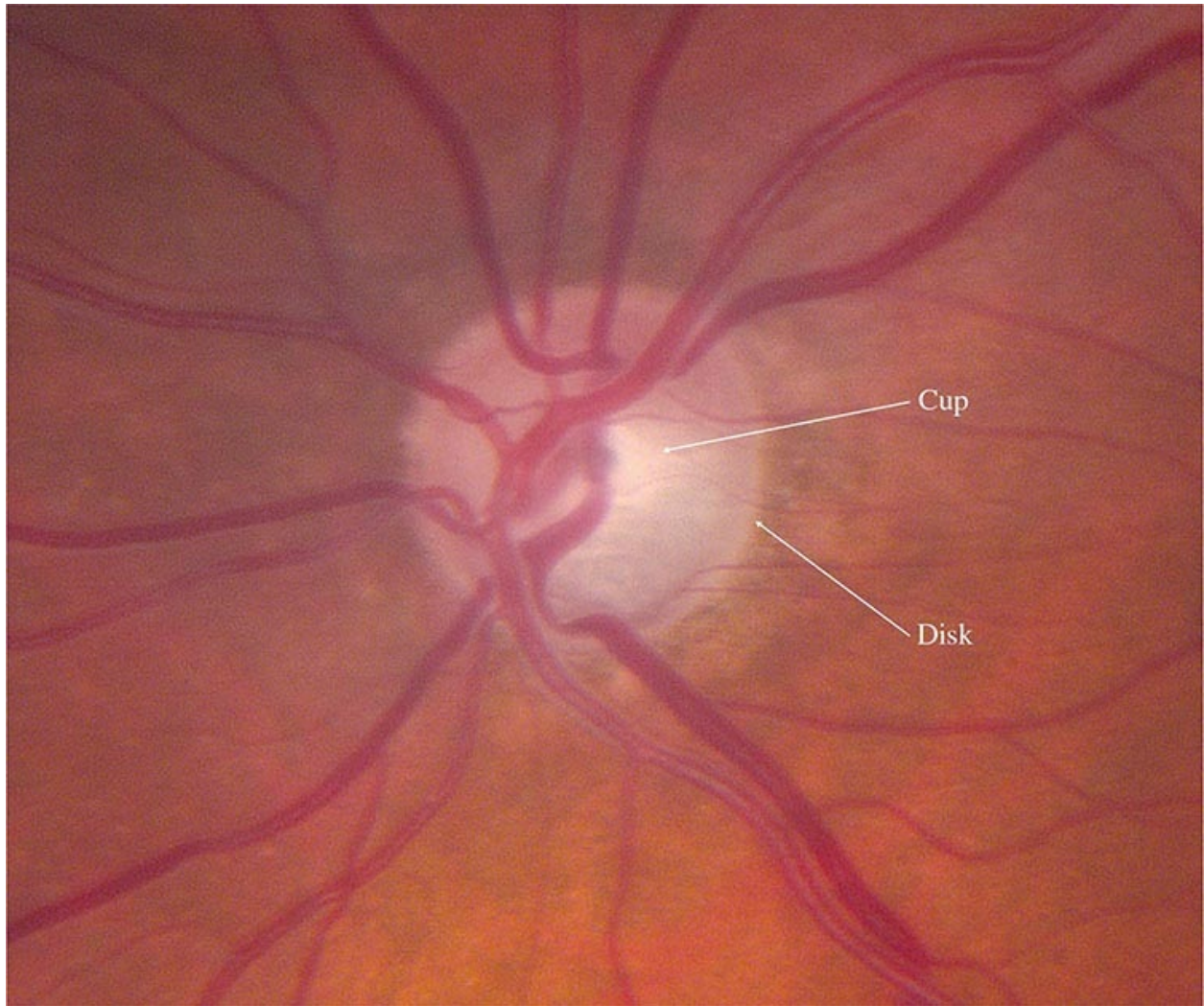


FIGURE 3.32 ■ Normal Cupping. The cup is central and is less than one-half the diameter of the disk.
(Photo contributor: Jeffrey Goshe, MD.)



FIGURE 3.33 ■ Glaucomatous Cupping. The cup is not central; it is elongated toward the rim superotemporally. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

SUBHYALOID HEMORRHAGE IN SUBARACHNOID HEMORRHAGE

Clinical Summary

Subhyaloid hemorrhage appears as extravasated blood beneath the retinal layer. These are often described as “boat-shaped” hemorrhages to distinguish them from the “flame-shaped” hemorrhages on the superficial nerve fiber layer of the retina. They may occur as a result of blunt trauma but are perhaps best known as a marker for SAH. In SAH, the hemorrhages appear as a “puff” of blood emanating from the central disk.

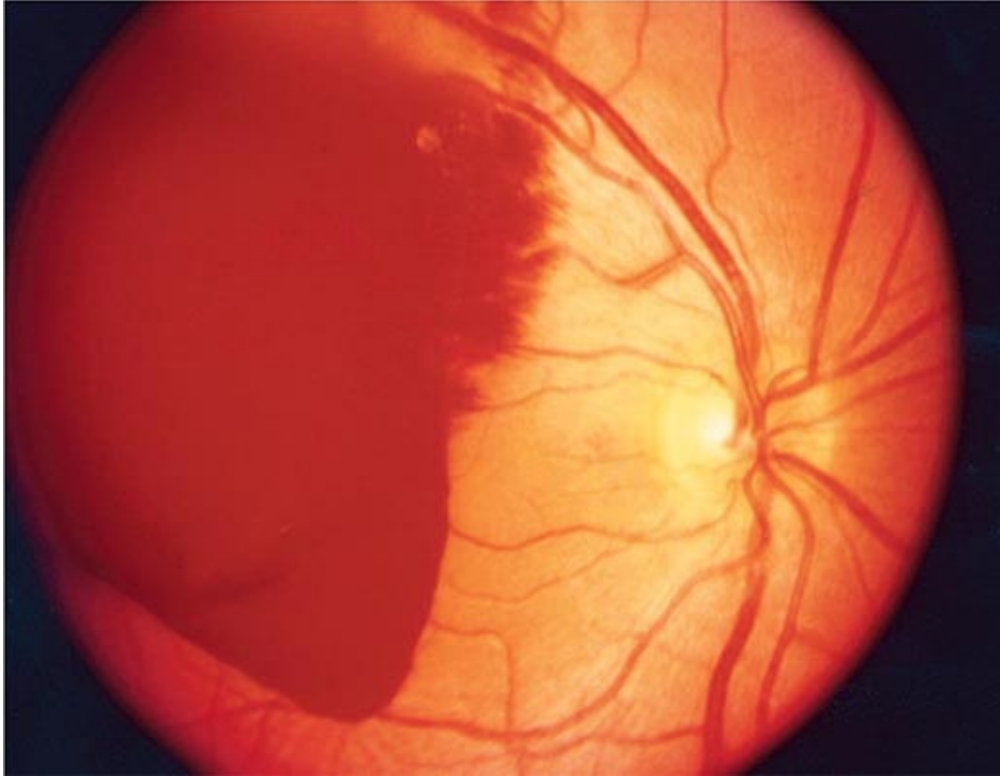


FIGURE 3.34 ■ Subhyaloid Hemorrhage. Subhyaloid hemorrhage seen on funduscopy examination in a patient with subarachnoid hemorrhage. (From Edlow JA, Caplan LR. Primary care: avoiding pitfalls in the diagnosis of subarachnoid hemorrhage. *N Engl J Med*. 2000;342:29-36. Reproduced with permission from John J. Weiter, MD, PhD.)

SAH, shaken impact syndrome, hypertensive retinopathy, and retinal hemorrhage should all be considered and aggressively evaluated.

Management and Disposition

No specific treatment is required for subhyaloid hemorrhage. Treatment is dependent on the underlying etiology. Appropriate specialty referral should be made in all cases.

Pearls

1. A funduscopy examination looking for subhyaloid hemorrhage should be included in all patients with severe headache, unresponsive pediatric patients, or those with altered mental status.
2. The appearance of a retinal hemorrhage indicates its anatomic location. A subhyaloid hemorrhage lies over the retinal vessels—thus, they cannot be

seen on fundusoscopic examination. In a subretinal hemorrhage (see Fig. 3.4), the vessels lie superficial to the hemorrhage and thus are easily seen.

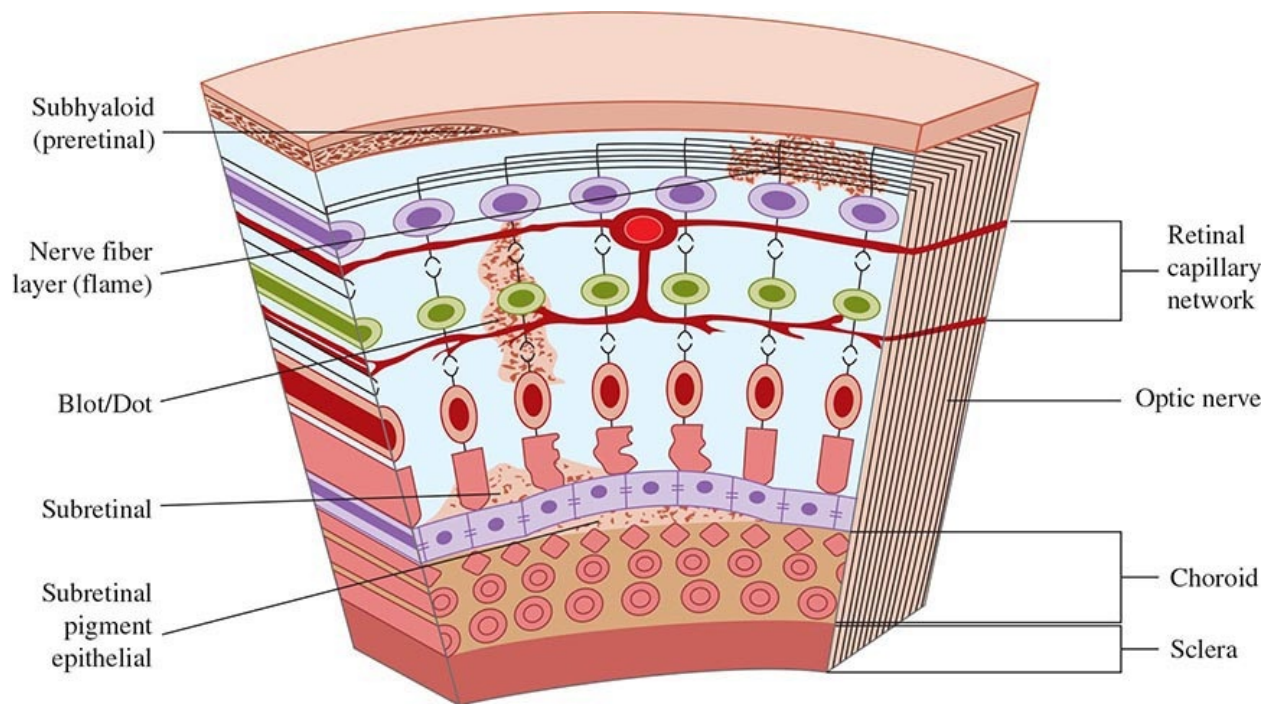
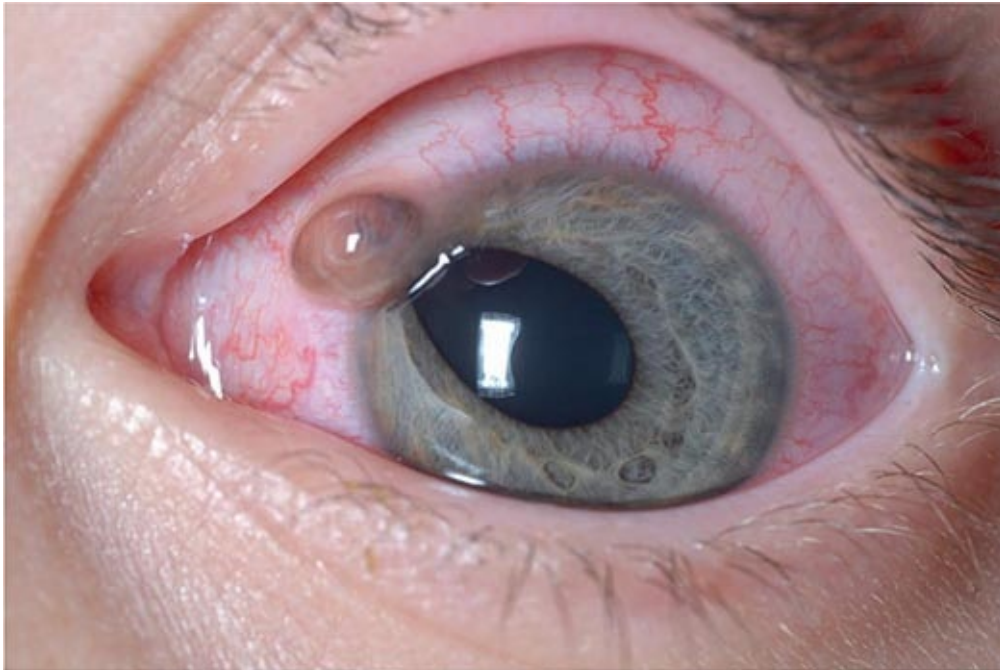


FIGURE 3.35 ■ Retinal Hemorrhages—Anatomic Location.

Chapter 4

OPHTHALMIC TRAUMA

Kevin J. Knoop
James K. Palma



Open Globe with Prolapsed Iris. Prolapsed iris seen at 10 o'clock and peaked pupil pointing to the prolapse indicating an open globe after trauma. The patient was hammering nails into a tree trunk when one ricocheted back at him. (Photo contributor: Timothy M. Brenkert, MD.)

CORNEAL ABRASION

Clinical Summary

Corneal abrasions present with acute onset of eye discomfort, tearing, and often

a foreign-body sensation. A “ciliary flush” (conjunctival injection hugging the limbus) may be seen. Large abrasions or those in the central visual axis may affect visual acuity. Photophobia and headache from ciliary muscle spasm may be present. Associated findings or complications include traumatic iritis, hypopyon, or a corneal ulcer. Fluorescein examination, preferably with a slit lamp, highlights the defect.

Management and Disposition

Instillation of topical anesthetic drops (eg, tetracaine 0.5%, proparacaine 0.5%) facilitates examination while relieving pain and blepharospasm. Brief outpatient treatment of corneal abrasions with topical anesthetics in selected patients has been shown to be safe and effective. Consider using a short-acting cycloplegic (eg, cyclopentolate 1% or homatropine 5%) to reduce pain from ciliary spasm in patients who complain of headache or photo-phobia. Consider oral opioid or nonsteroidal anti-inflammatory drug (NSAID) analgesics for pain control. NSAID eye drops (eg, diclofenac or ketorolac) are equally effective and avoid systemic side effects. Neither treatment with topical antibiotics, nor patching, nor tetanus prophylaxis for uncomplicated corneal abrasions has scientific validation. Artificial tears are safe and especially helpful for the dry eye symptoms that commonly occur after the corneal defect has healed. Emergency department and/or ophthalmologist follow-up is advised for any patient with complications or who is still symptomatic after 24 hours.

Pearls

1. Mucus may simulate fluorescein uptake, but its position changes with blinking.
2. Multiple linear corneal abrasions, the “ice-rink sign,” may result from an embedded foreign body adhered to the upper lid. Always evert the lid to evaluate this.
3. Whenever the mechanism includes grinding or striking metal, or high-velocity injuries from mowers or string trimmers, maintain a high index of suspicion for penetrating injury. Fluorescein streaming away from an “abrasion” (Seidel test) may be an indication of a corneal perforation.
4. Routine prophylactic treatment with topical antibiotics remains controversial. When used, inexpensive, broad-spectrum antibiotic drops (sulfacetamide sodium or trimethoprim/polymyxin B) allow clearer vision than lubricating

ointments, which may feel better, but blur vision. Avoid topical neomycin antibiotics because of a high risk of irritant allergy symptoms.

5. An “abrasion” in a contact lens wearer should alert one to suspect a corneal ulcer. Consult ophthalmology while the patient is in the emergency department.



FIGURE 4.1 ■ Corneal Abrasion. A small, subtle abrasion is seen at the 3-o'clock position, just across the larger white reflection from the flash. Note the brisk localized conjunctival inflammatory reaction and ciliary flush. (Photo contributor: Kevin J. Knoop, MD, MS.)

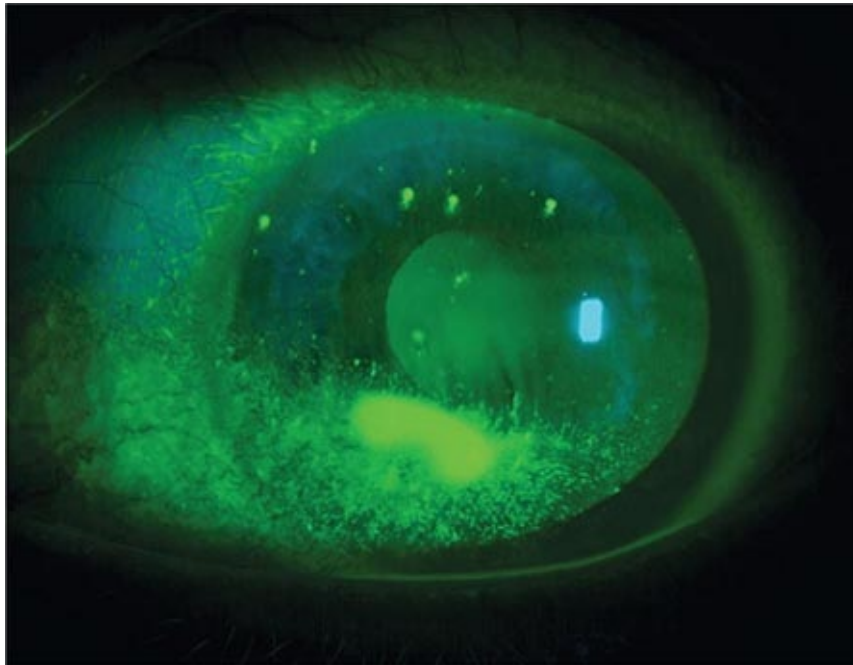


FIGURE 4.2 ■ Corneal Abrasion. Multiple punctate and one large traumatic abrasion are seen with

fluorescein uptake. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

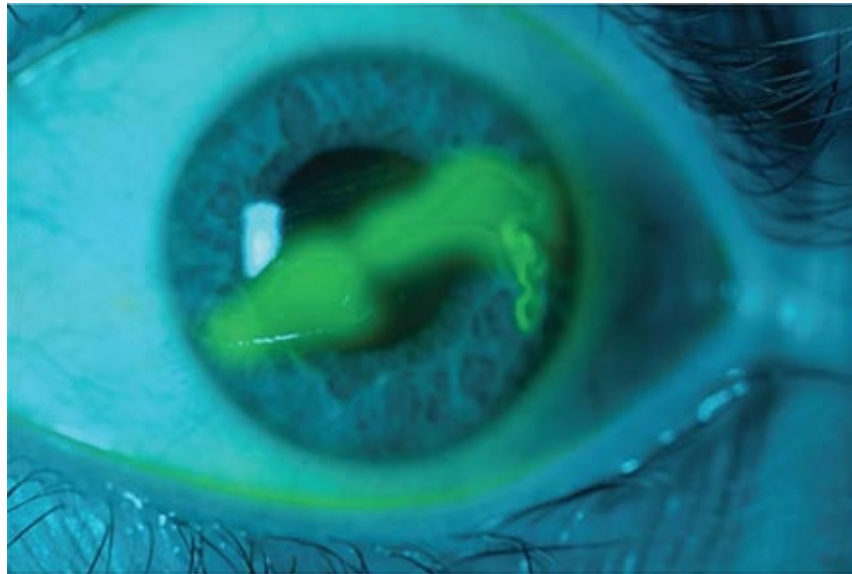


FIGURE 4.3 ■ Corneal Abrasion. Abrasions obscuring the visual axis benefit from close follow-up with an ophthalmologist to ensure adequate healing. (Photo contributor: Lawrence B. Stack, MD.)

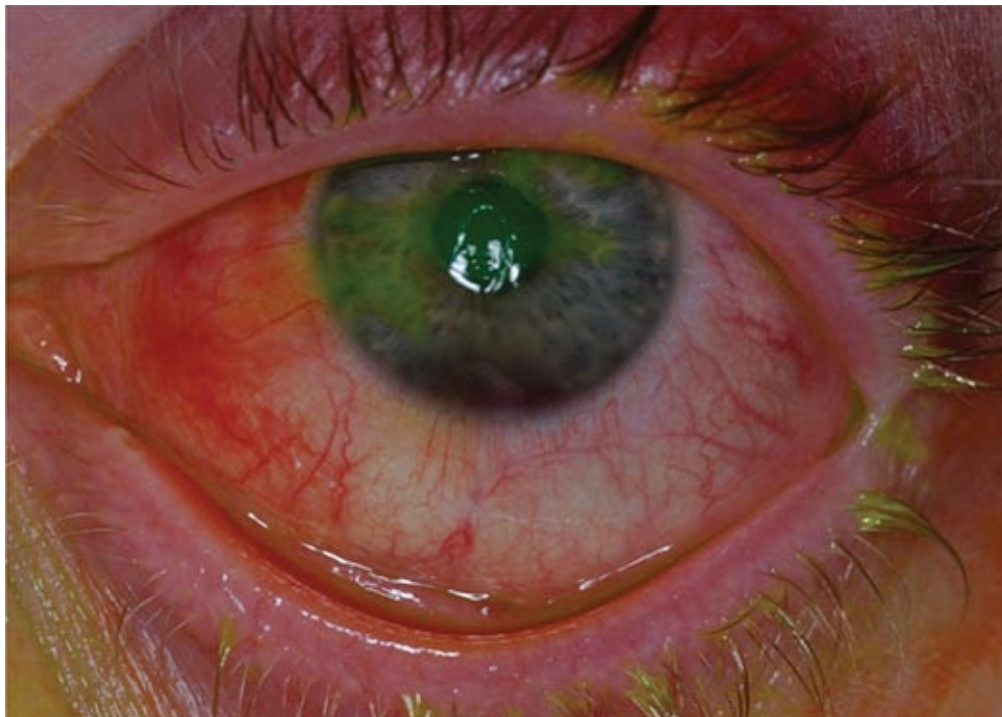


FIGURE 4.4 ■ Corneal Abrasion. This injury was due to a bungee cord impacting the eye. An irregular corneal light reflex indicates a disruption in the corneal epithelium (abrasion or perforation). A small hyphema is also seen. (Photo contributor: Kevin J. Knoop, MD, MS.)

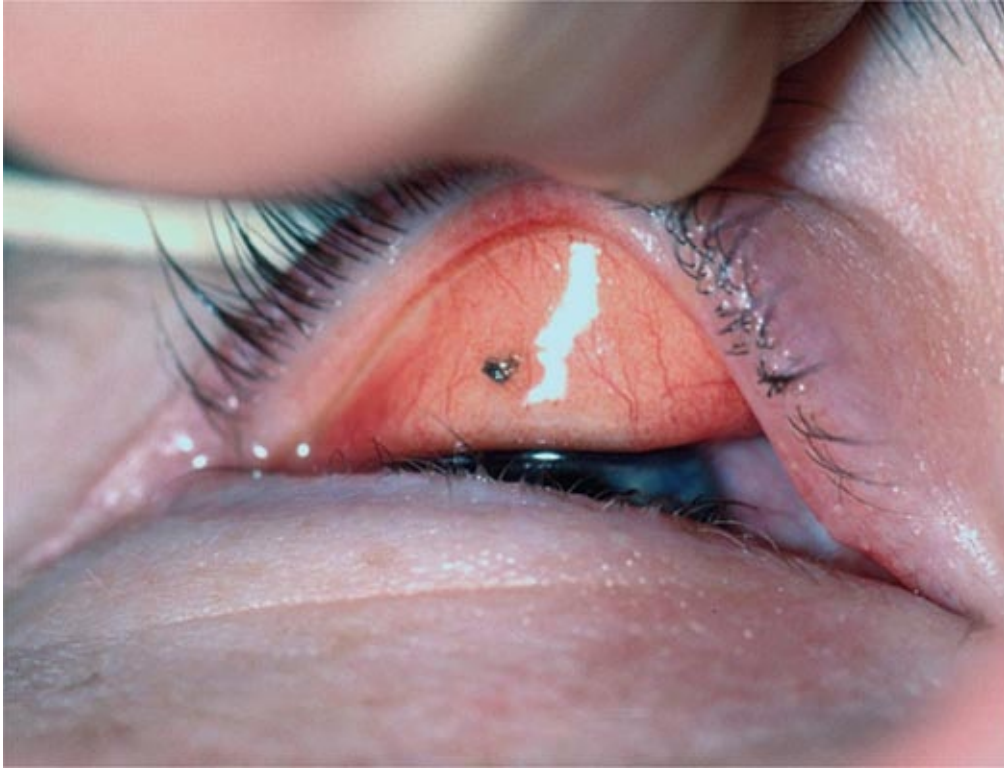


FIGURE 4.5 ■ Foreign Body under the Upper Lid. Lid eversion is an essential part of the eye examination to detect foreign bodies. (Photo contributor: Lawrence B. Stack, MD.)

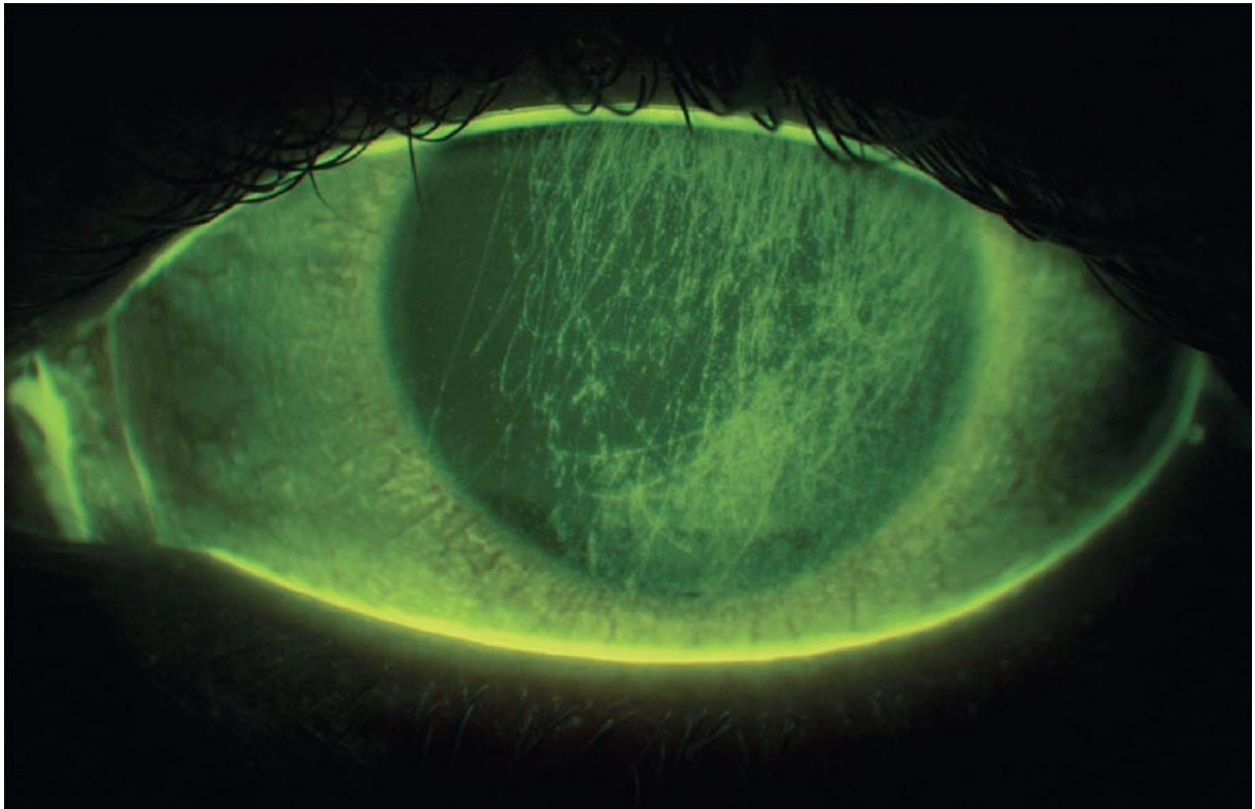


FIGURE 4.6 ■ “Ice-Rink Sign.” Multiple linear abrasions caused by upper lid foreign body. (Photo contributor: Aaron Sobol, MD.)

SUBCONJUNCTIVAL HEMORRHAGE

Clinical Summary

A subconjunctival hemorrhage or hematoma (SCH) may be a minor non–vision-threatening finding, or it could be associated with a major vision-threatening injury. SCH may occur with trivial events such as a cough, sneeze, Valsalva maneuver, or minor blunt trauma. The blood is usually bright red and sharply circumscribed and appears flat. It is limited to the bulbar conjunctiva and stops abruptly at the limbus. This appearance is important to differentiate from bloody chemosis, which can occur with scleral rupture or nontraumatic conditions. Uncomplicated SCH does not usually cause pain or diminution in visual acuity.

Management and Disposition

No treatment is required. Instruct the patient to expect the blood to resorb within 2 to 3 weeks.

Pearls

1. Elevated, dense, circumferential SCH should prompt evaluation for bleeding diathesis or globe rupture.
2. SCH involving the extreme lateral globe after blunt trauma is very suspicious for zygomatic arch fracture.
3. Evaluate patients with nontraumatic bloody chemosis for an underlying metabolic (coagulopathy) or structural (cavernous sinus thrombosis) disorder.



FIGURE 4.7 ■ Subconjunctival Hemorrhage. Subconjunctival hemorrhage from blunt trauma. The flat appearance of the hemorrhage suggests its benign nature. (Photo contributor: James K. Palma, MD, MPH.)

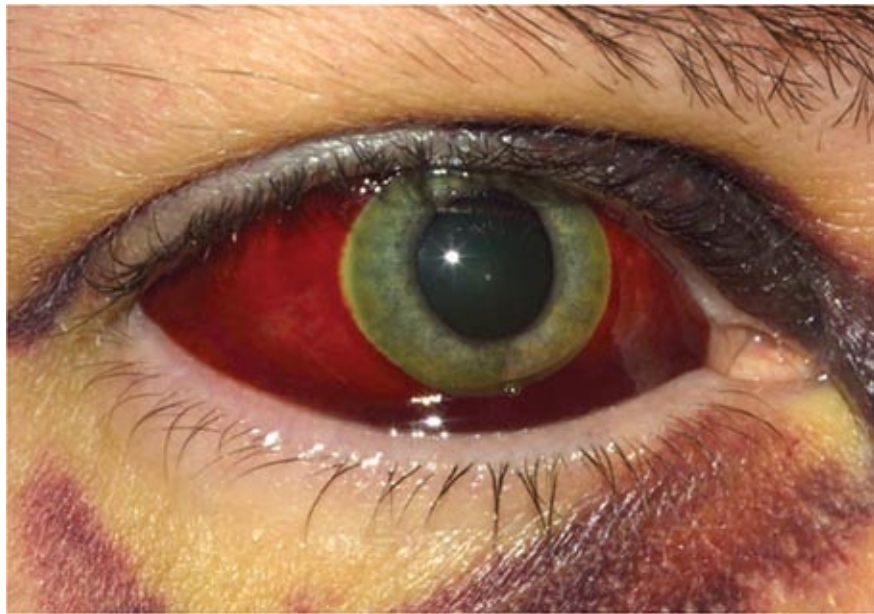


FIGURE 4.8 ■ Subconjunctival Hemorrhage. Subconjunctival hemorrhage that completely surrounds the eye but stops abruptly at the limbus in a patient with blunt trauma. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)



FIGURE 4.9 ■ Traumatic Bloody Chemosis. This anticoagulated patient presented after relatively minor trauma with normal visual acuity. He was able to close his eye and required only supportive care. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 4.10 ■ Traumatic Bloody Chemosis. Globe integrity should be assessed with CT and exam by an ophthalmologist. (Photo contributor: Alan B. Storrow, MD.)

CORNEAL FOREIGN BODY/RUST RING

Clinical Summary

Patients typically report getting something in the eye or complain of FB sensation. FBs that overlie the cornea may affect vision. Tearing, conjunctival injection, headache, and photo-phobia may also be present. The most important consideration is the possibility of a penetrating globe injury. One must elicit a meticulous history about the mechanism of injury, such as hammering metal on metal or using power lawn equipment.

Management and Disposition

Topical anesthetic drops (eg, 0.5% proparacaine or tetracaine) facilitate examination and removal. If superficial, removal with saline flush may be attempted before using a sterile eye spud or small (25-gauge) needle. Consider topical antibiotic drops or ointment for the residual corneal abrasion. Tetanus prophylaxis is indicated. A “short-acting” cycloplegic (cyclopentolate 1% or homatropine 5%) may benefit patients with headache or photophobia. FB or “rust ring” removal should be conducted using slit-lamp microscopy and *only* by a physician skilled in rust ring removal due to the risk of corneal perforation or scarring.

Pearls

1. Always evert the upper lid and search carefully for a FB. A FB adherent to the upper lid abrades the cornea, producing the “ice-rink” sign, caused by multiple linear abrasions.
2. Vigorous attempts to remove the entire rust ring are not warranted. This may await emergency department or ophthalmology follow-up in 24 hours.
3. The tip of an 18-gauge needle can be bent 90 degrees using sterile forceps or the needle cap to make a “scoop” for FB and rust ring removal.

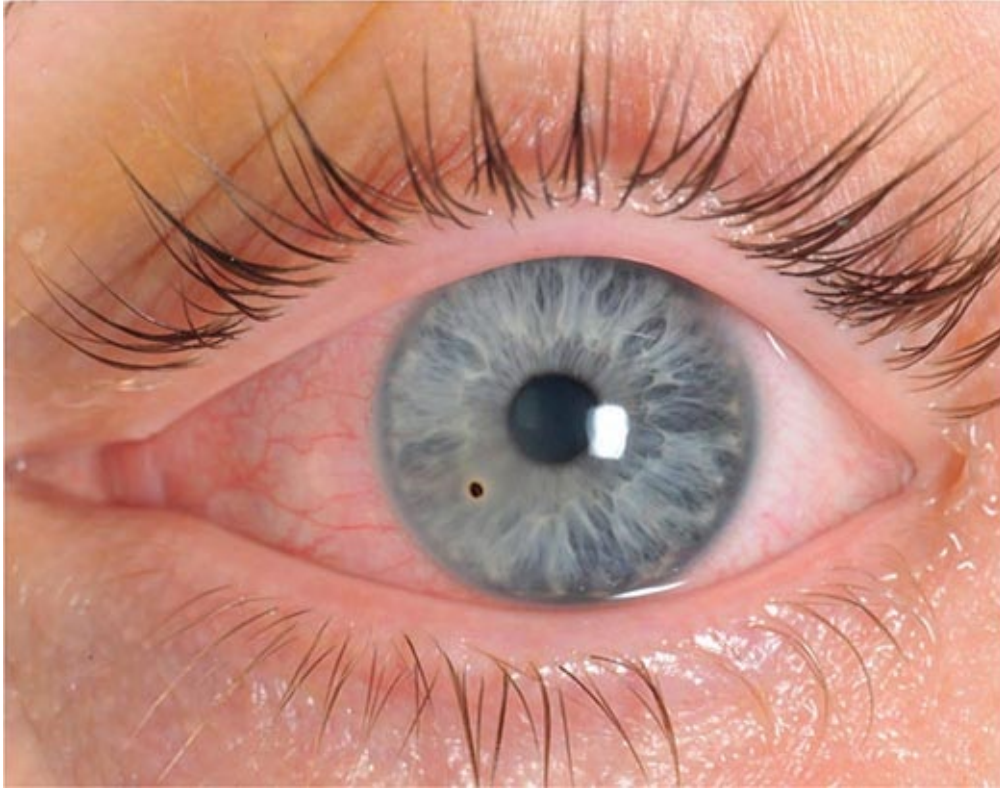


FIGURE 4.11 ■ Corneal Foreign Body. Metallic corneal foreign body with a rust ring and surrounding inflammation at 8 o'clock. Note corneal haziness extending out from the foreign body. (Photo contributor: Aubrey Mowery, MSN, MPH, CPNP.)

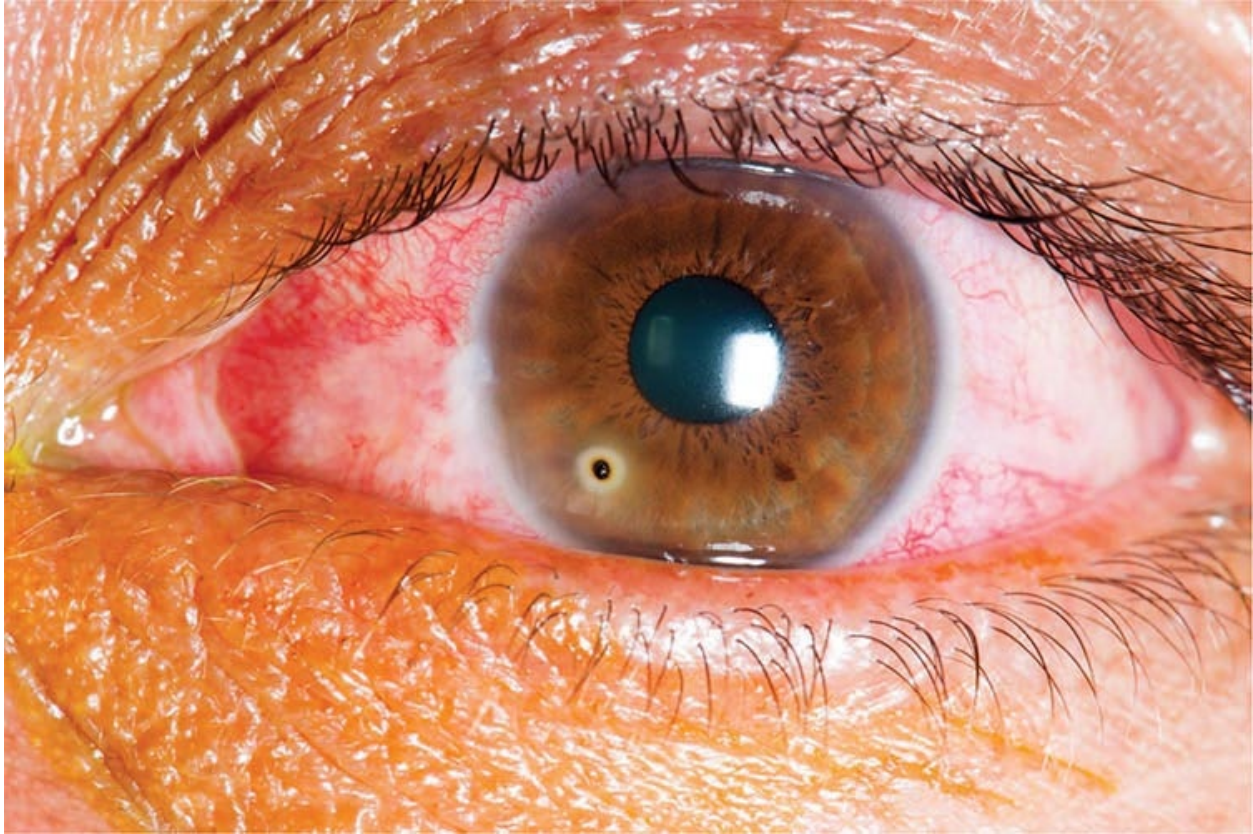


FIGURE 4.12 ■ Corneal Foreign Body. Metallic corneal foreign body with a rust ring and surrounding inflammation at 8 o'clock embedded for 2 days. (Photo contributor: Lawrence B. Stack, MD.)

HYPHEMA

Clinical Summary

Injury to the anterior chamber that disrupts the vasculature supporting the iris or ciliary body results in a hyphema. The blood tends to layer with time, and if left undisturbed, gravity will form a visible meniscus. Symptoms can include pain, photophobia, and possibly blurred vision secondary to obstructing blood cells. Nausea and vomiting may signal a rise in intraocular pressure (glaucoma) caused by blood cells clogging the trabecular meshwork.

Management and Disposition

Prevention of further hemorrhage is the foremost treatment goal. Most rebleeding occurs within the first 72 hours and is usually more extensive than the

initial event. Keep the patient at rest in the supine position with the head elevated slightly. Consider a hard eye shield to prevent further trauma from manipulation. Avoid medications with antiplatelet activity such as NSAIDs. Use antiemetics if the patient has nausea. Further treatment at the discretion of specialty consultants may include topical or oral steroids, antifibrinolytics such as aminocaproic acid or tranexamic acid, cycloplegics, and/or surgery. Measure intraocular pressure (IOP) unless there is a suspicion of penetrating injury to the globe. Treat elevated IOP with appropriate agents, including topical β -blockers, pilocarpine, and, if needed, osmotic agents (mannitol, sorbitol) and acetazolamide. Ophthalmologic consultation is warranted to determine local admission practices.

Pearls

1. Instruct patients specifically not to read or watch television, as these activities result in greater than usual ocular activity.
2. Rebleeding may occur in 10% to 20% of patients, most commonly in the first 2 to 5 days when the blood clots start to retract.
3. An “eight-ball” or total hyphema often leads to elevated IOP and corneal bloodstaining and typically requires surgical evacuation.
4. Patients with sickle cell and other hemoglobinopathies are at risk for sickling of blood inside the anterior chamber. This can cause a rise in IOP caused by obstruction of the trabecular meshwork even if only a small hyphema is present.
5. An abnormally low IOP should prompt consideration for presence of penetrating globe injury.
6. Evaluate supine trauma patients for slight differences in iris color to determine the presence of a hyphema.

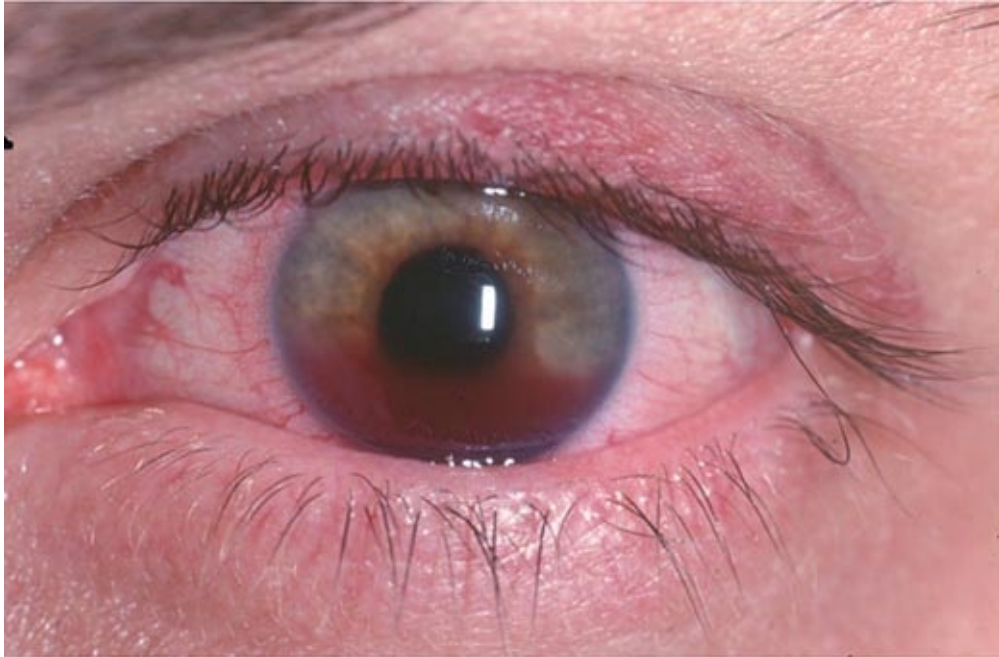


FIGURE 4.13 ■ Hyphema. This hyphema is just beginning to layer out, reflecting its acute nature. (Photo contributor: Lawrence B. Stack, MD.)

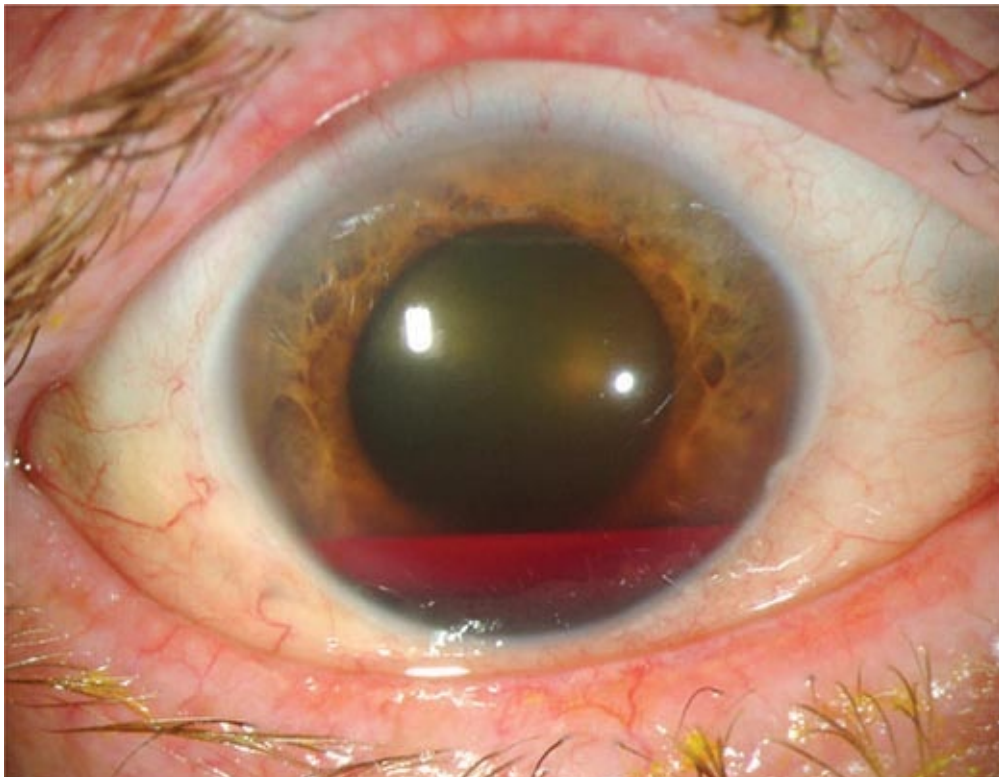


FIGURE 4.14 ■ Hyphema. This hyphema has completely layered out in the anterior chamber. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

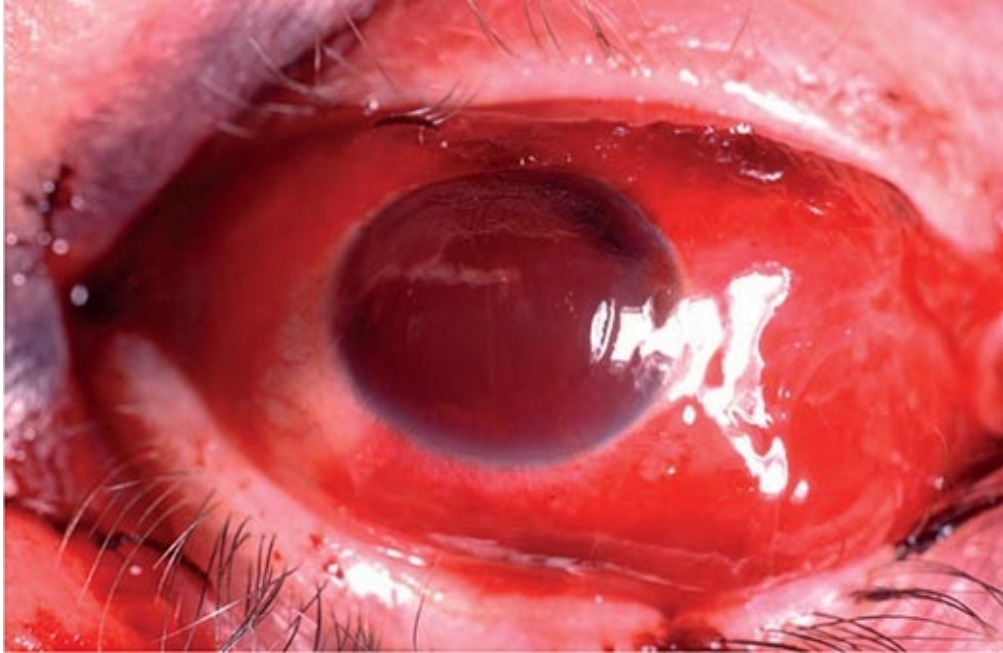


FIGURE 4.15 ■ “Eight-Ball” Hyphema. This hyphema completely fills the anterior chamber. (Photo contributor: Lawrence B. Stack, MD.)

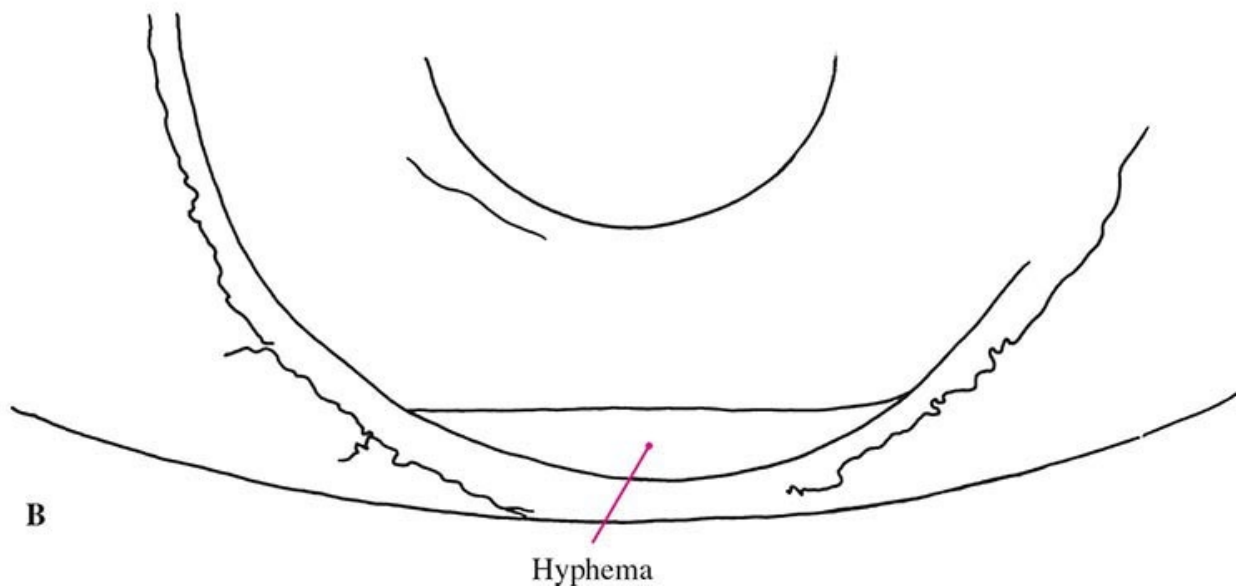
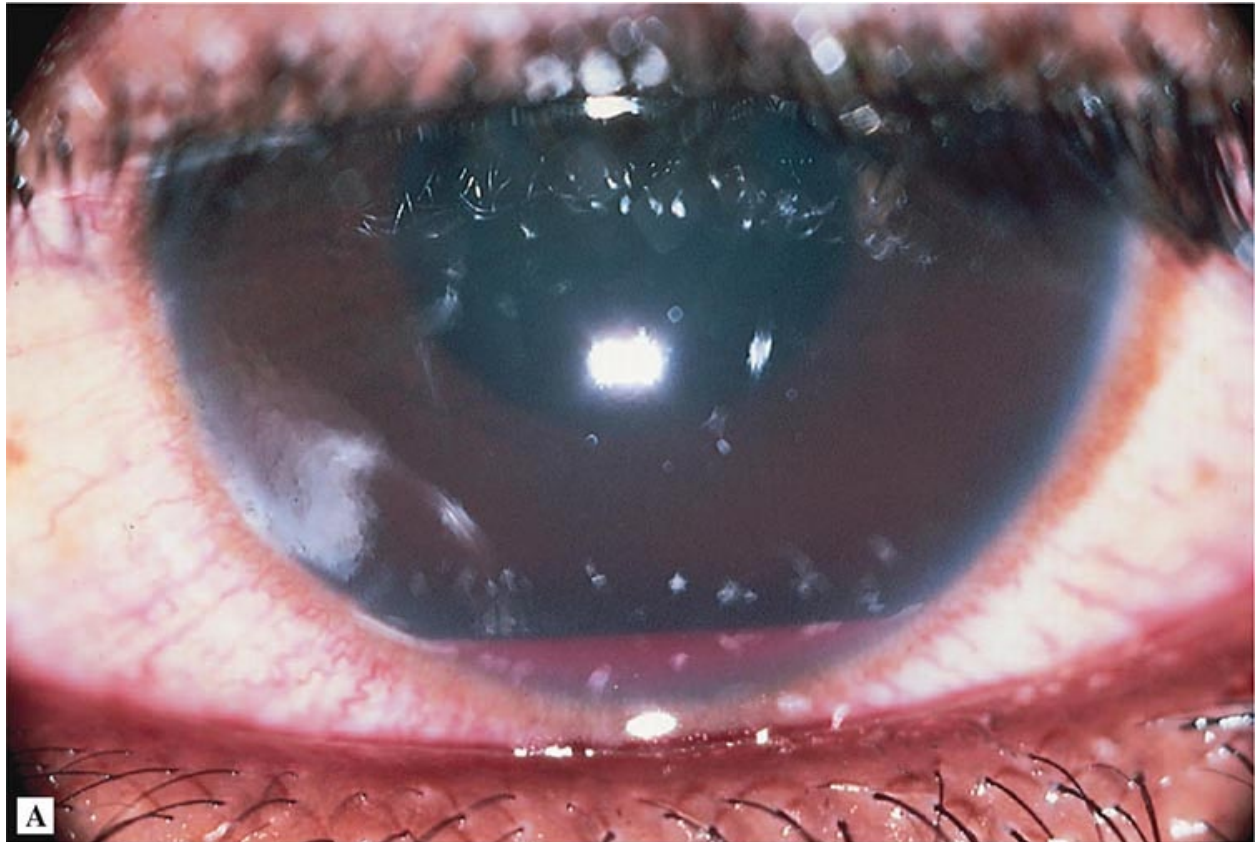


FIGURE 4.16 ■ Hyphema. A small hyphema (about 5%) in a patient with sickle cell disease. (Photo contributor: Dallas E. Peak, MD.)

INTRAOCULAR FOREIGN BODY

Clinical Summary

The most important consideration with any eye injury is the possibility of a penetrating globe injury with residual intraocular foreign body (IO FB). Patients may report FB sensation, but subtle presentations occur. Obtain a meticulous history about mechanism of injury (grinding or metal on metal).

Management and Disposition

For suspected subtle injury, a careful examination is required. Bedside ultrasound can be a useful adjunct and allows rapid identification of an IO FB. Care must be taken to avoid any pressure on the globe. A slit-lamp examination with Seidel test (copious amounts of fluorescein instilled and observed for streaming away from the site of perforation) may reveal a microperforation.

Pearls

1. Maintain a high index of suspicion for penetrating globe injury, especially in mechanisms involving use of “metal on metal” such as grinding or hammering. A positive Seidel test demonstrates corneal microperforation.
2. If ocular penetration is suspected, a diligent search for a retained FB is indicated. Careful bedside ultrasound using a high-frequency transducer with copious gel (to avoid any pressure on the globe) can be used initially, but computed tomography (CT) is the diagnostic study of choice. Avoid magnetic resonance imaging (MRI), which should be saved for indeterminate results or when confirmation is required.
3. If using x-ray, obtain in both up and down gaze to assist localization of the metallic FB.

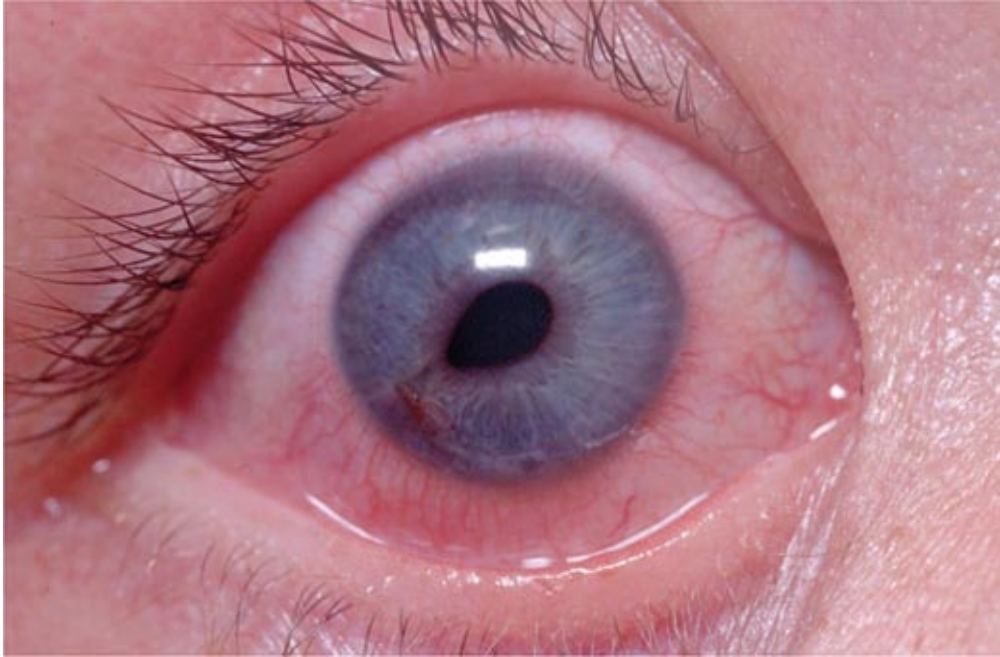


FIGURE 4.17 ■ Anterior Chamber Foreign Body. A shard from a nail is seen embedded in the anterior chamber. A “teardrop” pupil is present, indicating perforation. (Photo contributor: Lawrence B. Stack, MD.)

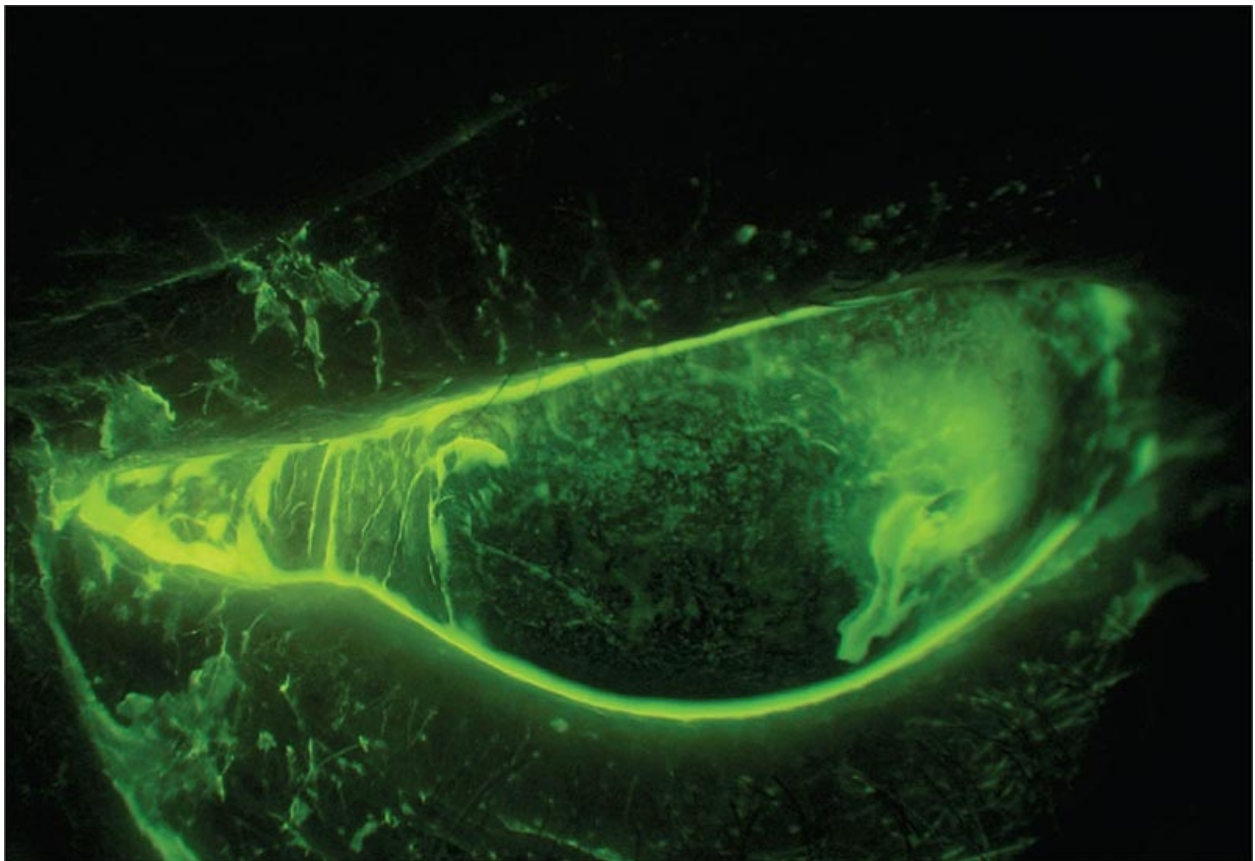


FIGURE 4.18 ■ Seidel Test. A positive Seidel test shows aqueous leaking through a corneal perforation while being observed with the slit lamp. (Photo contributor: Aaron Sobol, MD.)

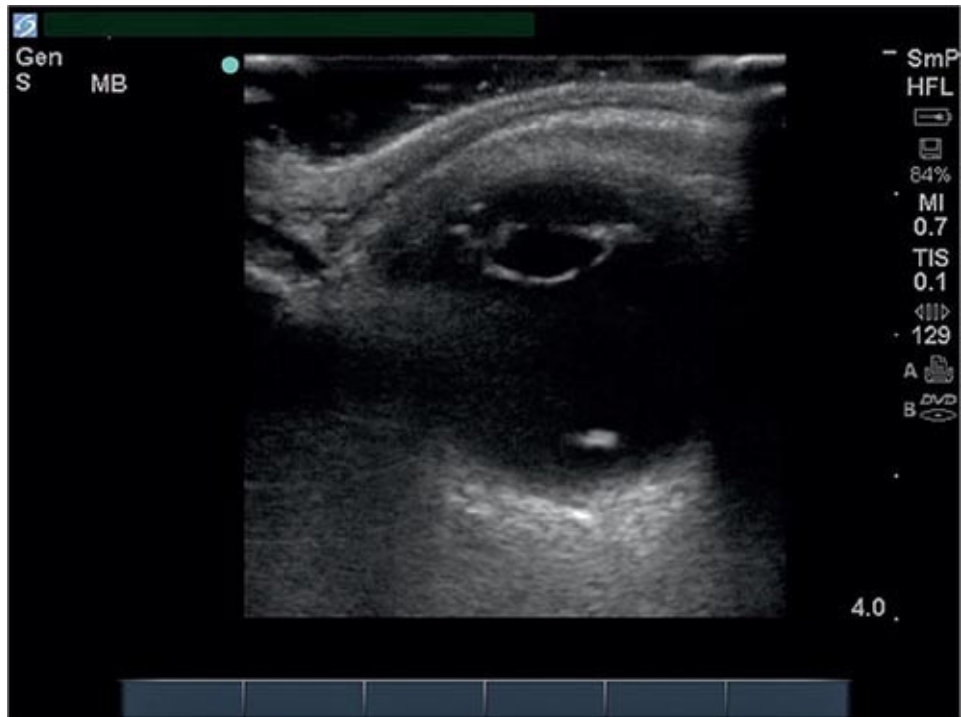


FIGURE 4.19 ■ Intraocular Foreign Body. Hyperechoic foreign body clearly seen in the posterior vitreous. Note the layer of gel overlying the eyelid indicating “no pressure” technique. (Photo contributor: James K. Palma, MD.)

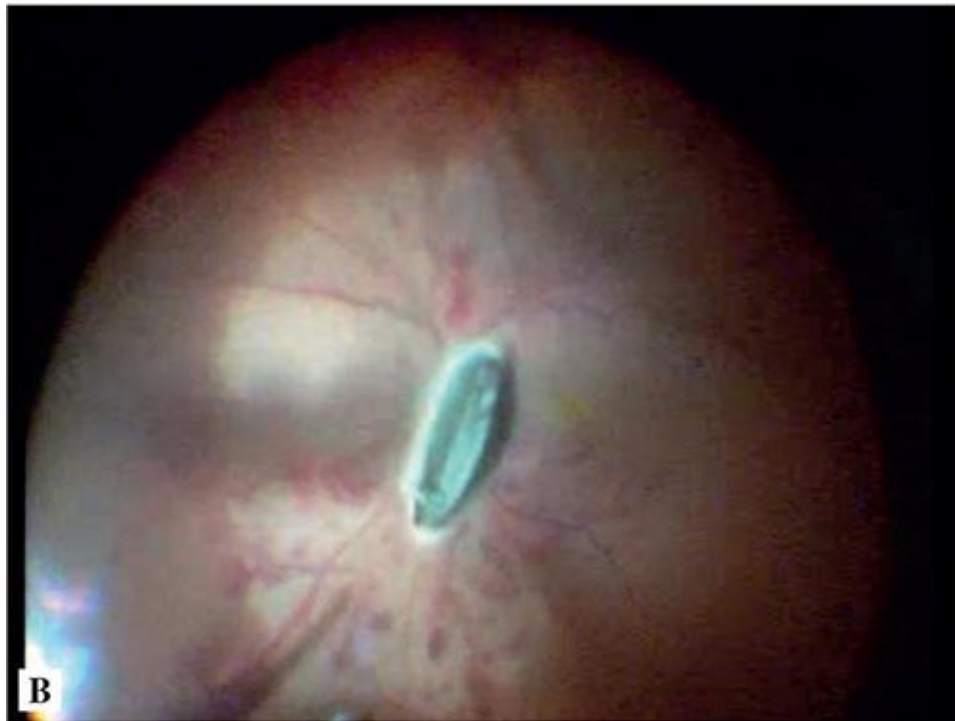
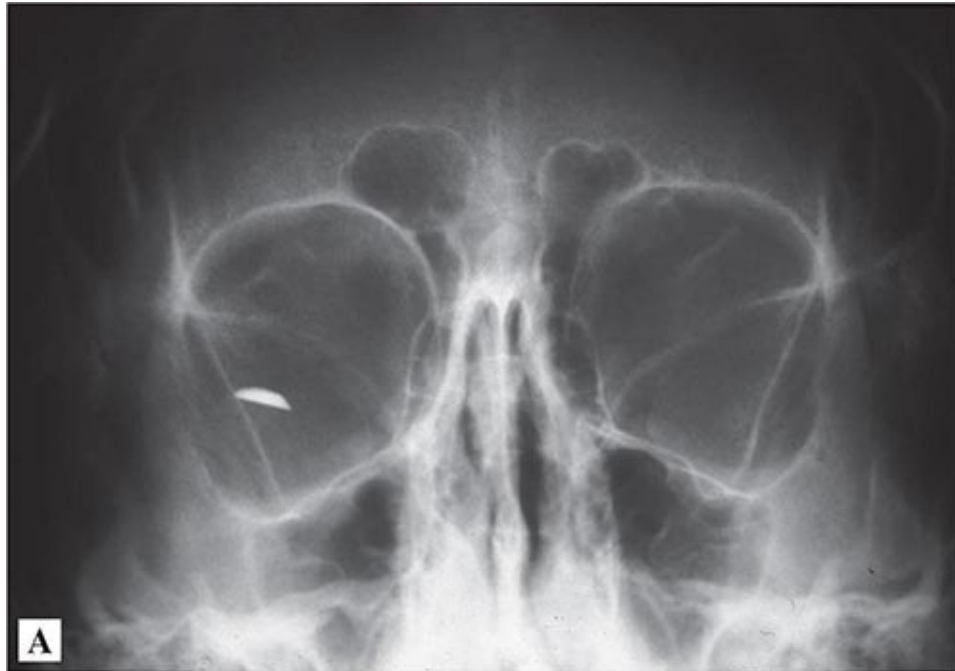


FIGURE 4.20 ■ Intraocular Foreign Body (FB). Skull x-ray with intraocular metallic FB (**A**) and corresponding fundoscopic exam with FB near optic nerve (**B**). This occurred following hammering of metal. (Photo contributor: Lawrence Lee, MD.)

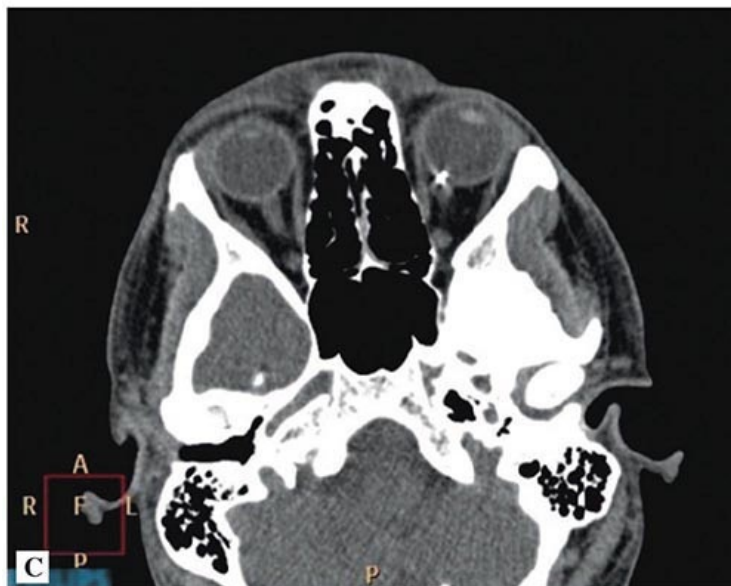
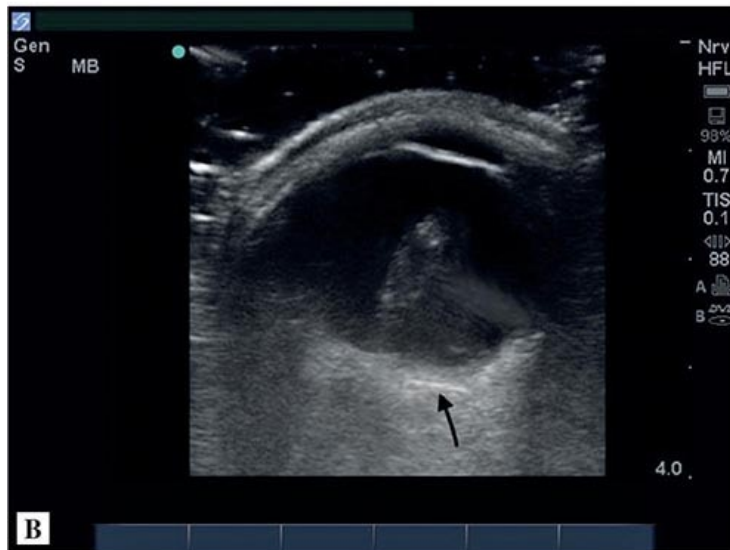


FIGURE 4.21 ■ Penetrating Globe Injury/Intraocular Foreign Body. (A) This fragmentation wound has a deformed pupil, iridodialysis, extrusion of ocular contents laterally, and mucopurulent discharge. (B) Ocular ultrasound demonstrates a hyperechoic stripe in the vitreous along the path of the foreign body and an irregular posterior retinal border. A careful ultrasound exam ensures that no pressure is placed on the globe. (C) CT demonstrates a posterior metallic ocular foreign body. (Photo contributor: James V. Ritchie, MD.)

IRIDODIALYSIS

Clinical Summary

Traumatic iridodialysis is the result of an injury, typically blunt trauma that pulls the iris away from the ciliary body. The resulting deformity appears as a lens-shaped defect at the outer margin of the iris. Patients may present complaining of a “2nd pupil.” As the iris pulls away from the ciliary body, a small amount of bleeding may result. Look closely for associated traumatic hyphema.

Consider etiologies such as penetrating injury to the globe, scleral rupture, IO FB, and lens dislocation causing billowing of the iris.

Management and Disposition

A remote traumatic iridodialysis requires no specific treatment in the emergency department. Recent history of ocular trauma should prompt a diligent slit-lamp examination for associated hyphema or lens dislocation. If hyphema is present, treat it as discussed (see “Hyphema”). Refer cases of iridodialysis for specialty consultation to exclude other injuries; if the defect is large enough to result in monocular diplopia, surgical repair may be necessary.

Pearls

1. The examination should carefully exclude posterior chamber pathology and hyphema. Consider bedside ultrasonography to rule out posterior pole injuries (retinal detachment, vitreous hemorrhage, lens dislocation, or FB).
2. Carefully review the history to exclude penetrating trauma. If the history is unclear, CT scan may be used to exclude the presence of IO FB.
3. A careful examination includes searching for associated lens dislocation.

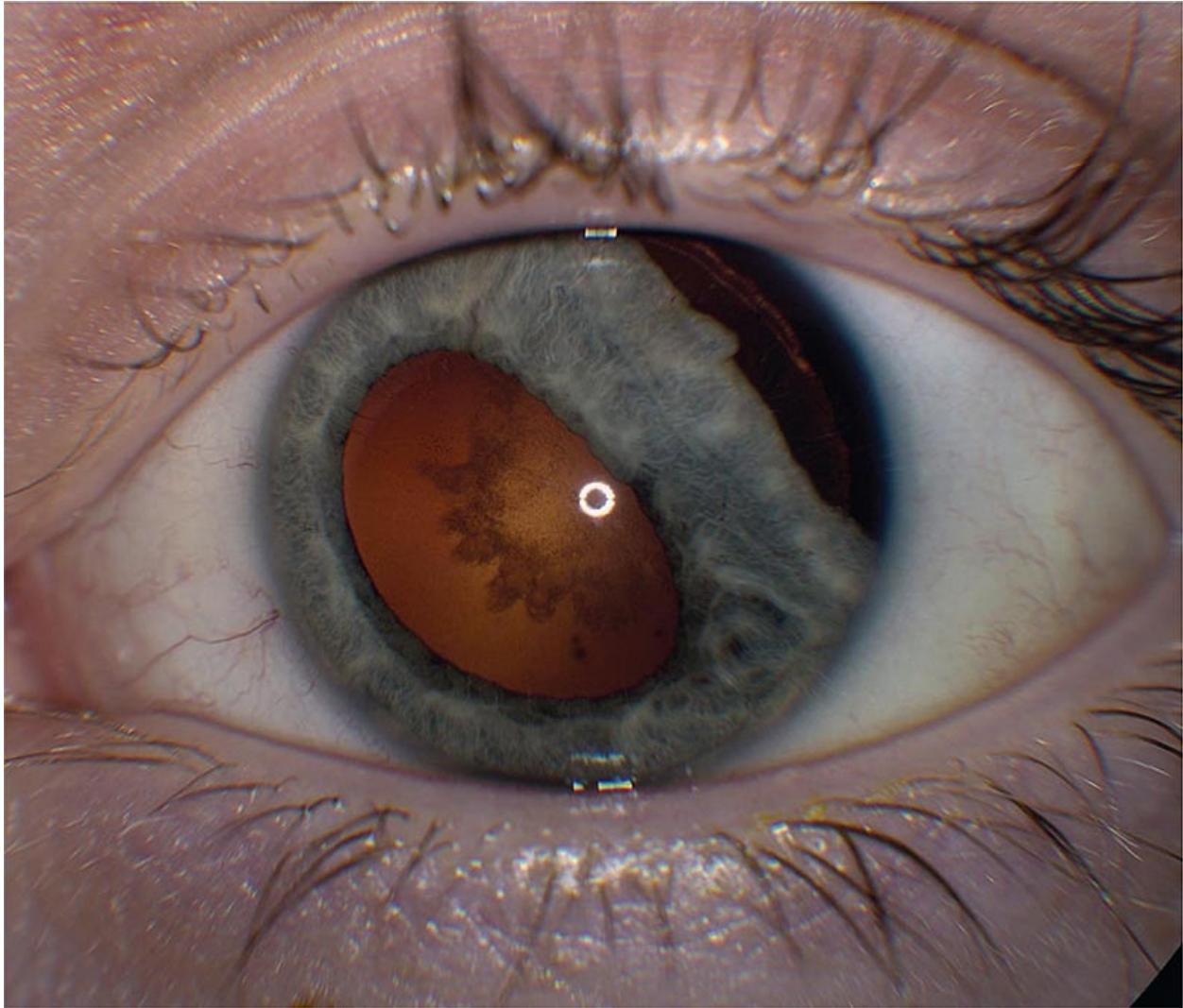


FIGURE 4.22 ■ Traumatic Iridodialysis. The iris has pulled away from the ciliary body as a result of blunt trauma. A traumatic cataract is also seen. The rosette pattern is classically seen after contusion injuries. It is due to separation of lens fibers around lens sutures. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

LENS DISLOCATION

Clinical Summary

Lens dislocation may result from blunt trauma to the globe. As the anterior surface of the eye is struck, there is compression in the anteroposterior dimension with resultant stretching of the globe along its equator in the medial-lateral plane. As this occurs, it stretches the zonule fibers, which suspend the

lens in place, and they, along with the lens capsule, may become disrupted. The patient may experience symptoms of monocular diplopia or gross blurring of images, depending on the severity of the injury. Occasionally there can be dramatic visual fluctuations caused by the lens changing position with resultant phakic and aphakic vision. There is generally a lack of pain except if secondary angle closure glaucoma occurs from the lens causing pupillary block. On slit-lamp examination, the displaced crystalline lens appears as a crescent shape along its edge against a backdrop of the red reflex from the fundus. The edge of the subluxed lens may be visible only with pupillary dilatation. Use caution in dilating the pupil, as this may cause the lens to sublux into the anterior chamber, which occurs if all the zonule fibers are torn. Chronically, the lens may lodge in either the anterior chamber or the vitreous. Marfan syndrome, tertiary syphilis, and homocystinuria may be present and should be considered in patients presenting with lens dislocation.

Management and Disposition

A subluxed lens does not always require surgery; partial subluxations may require only a change in refraction. Surgery is required if anterior dislocation of the lens results in pupillary block and angle closure glaucoma results.

Pearls

1. Patients may experience lens dislocation with seemingly trivial trauma if they have an underlying coloboma of the lens, Marfan syndrome, homocystinuria, or syphilis.
2. Iridodonesis is a trembling movement of the iris noted after rapid eye movements and is a sign of occult posterior lens dislocation.
3. Phacodonesis is a tremulousness of the lens itself caused by disruption of the zonule fibers.
4. Lens dislocation or subluxation is commonly associated with traumatic cataract formation.
5. Other associated injuries include hyphema, vitreous hemorrhage, and globe rupture.

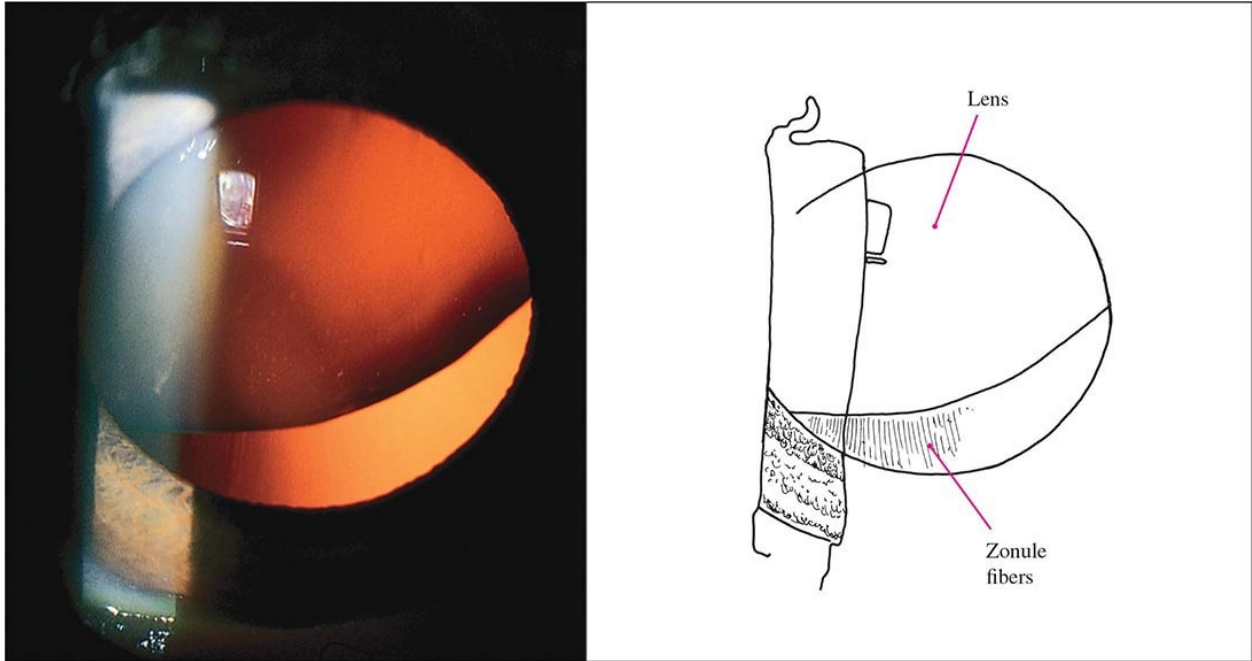


FIGURE 4.23 ■ Lens Dislocation. Lens dislocation revealed during slit-lamp examination. Note the zonule fibers, which normally hold the lens in place. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

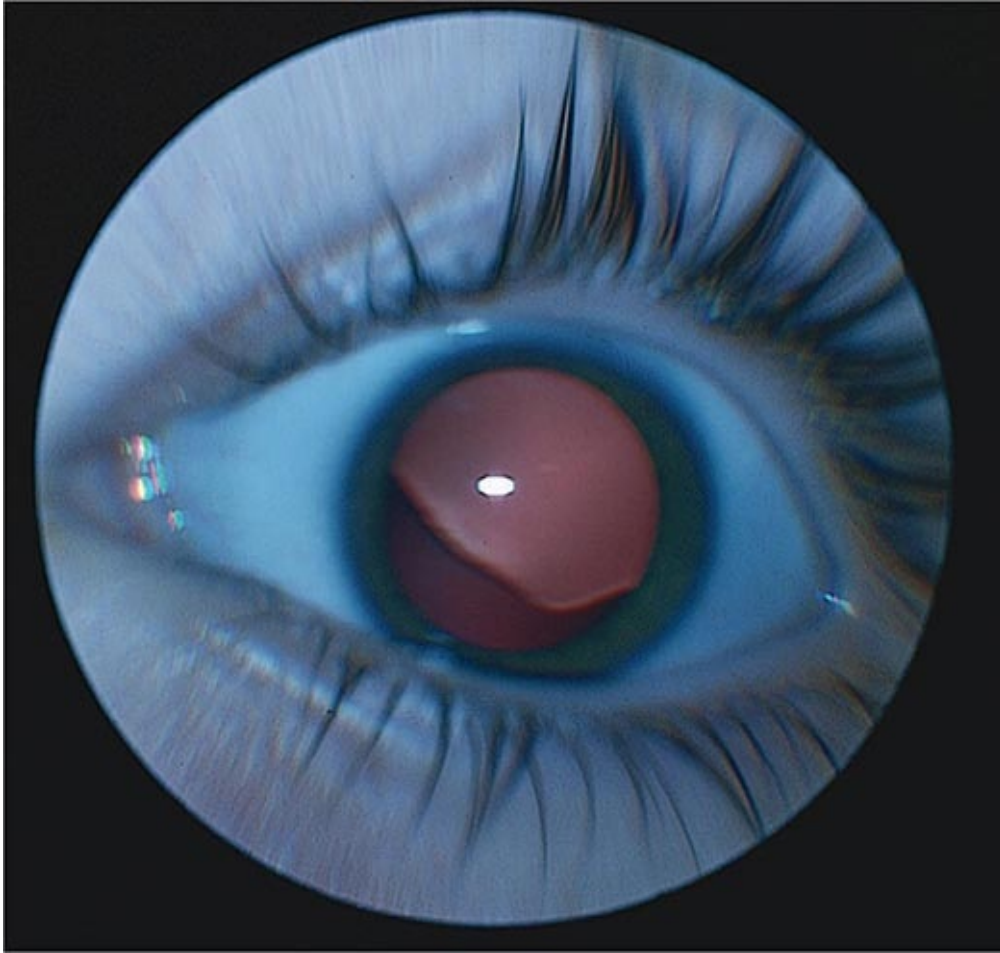


FIGURE 4.24 ■ Lens Dislocation. The edge of this dislocated lens is visible with the pupil dilated as an altered red reflex. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

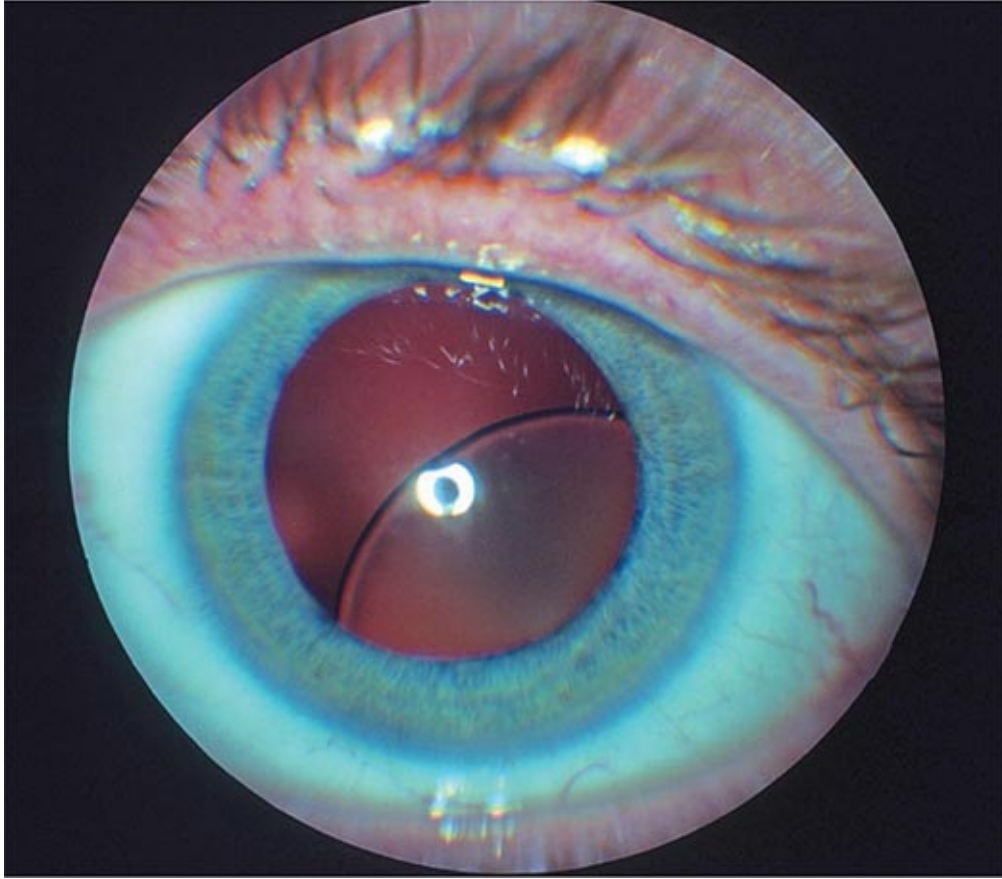


FIGURE 4.25 ■ Lens Dislocation. The crescentic edge of this dislocated lens is visible. Red reflex is abnormal. (Photo contributor: Thomas Egnatz, CRA.)

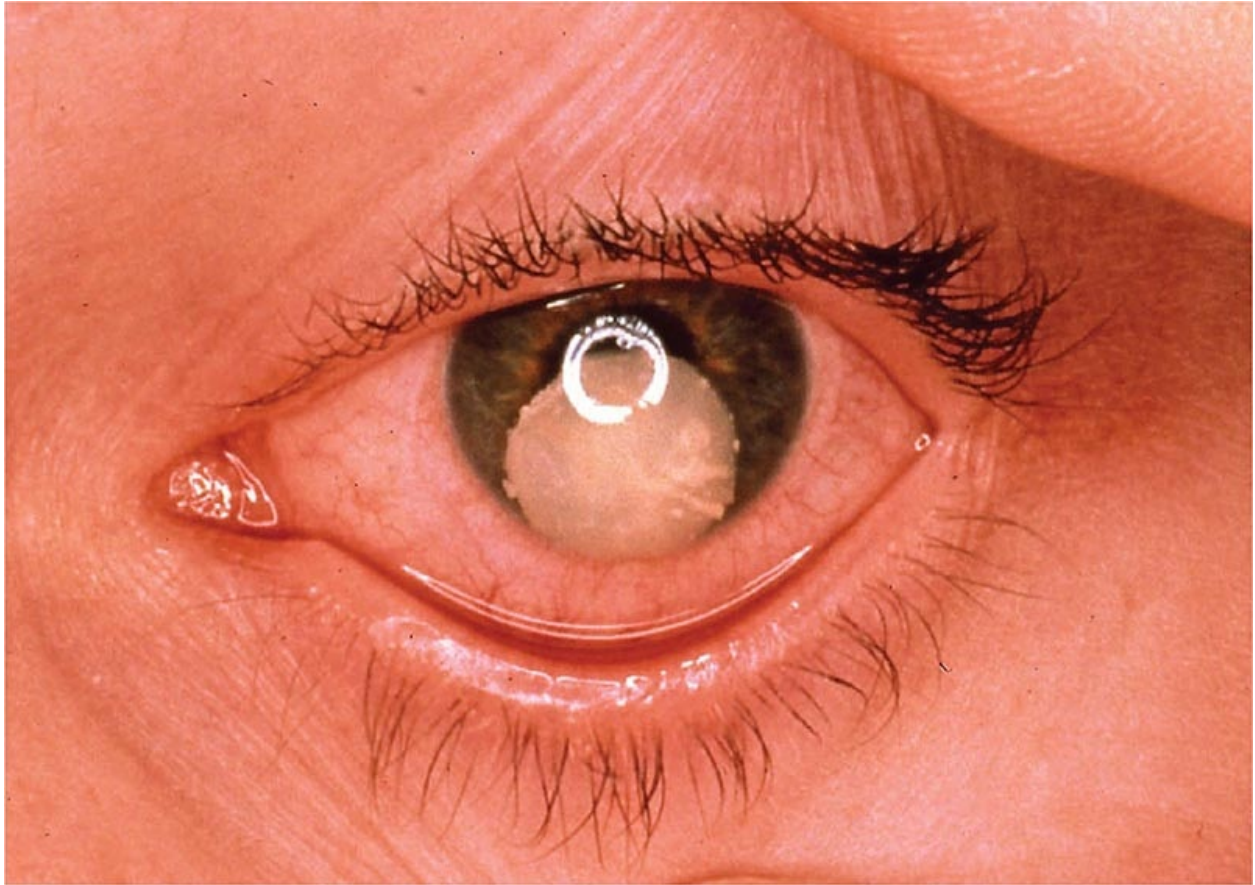


FIGURE 4.26 ■ Lens Dislocation. Complete lens dislocation prolapsed into the anterior chamber. (Photo contributor: David Effron, MD.)

PENETRATING GLOBE INJURY

Clinical Summary

Penetrating globe injuries can be subtle and easily overlooked. All are serious injuries. Signs to look for are loss of anterior chamber depth caused by leakage of aqueous humor, a tear-drop-shaped (“peaked”) pupil, a dark area on the bulbar conjunctiva, or prolapse of choroid through the wound.

Management and Disposition

Open globe injuries require urgent specialty consultation. Immediately protect the affected eye with a rigid eye shield. If a specifically designed shield is

unavailable, the bottom of a Styrofoam cup may be used. Do not use a pressure patch. Tonometry to measure pressures is strictly contraindicated. It is imperative to avoid inadvertent pressure on the globe with resulting irreversible expulsion of choroid through the wound. Intravenous antibiotics appropriate to cover gram-positive organisms are indicated. Consider adding gram-negative coverage for injuries that involve organic FBs. Prophylactic anti-emetics, sedation, and aggressive pain management are crucial to prevent or decrease expulsion of intraocular contents caused by crying, activity, or vomiting. Update tetanus status. Other significant blunt trauma may accompany penetrating globe. Always consider the possibility that FBs may have penetrated through the globe into the posterior orbit and possibly extend into the cranial vault.



FIGURE 4.27 ■ Open Globe. This injury is not subtle; extruded ocular contents (vitreous) can be seen; a teardrop pupil is also present. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 4.28 ■ Open Globe. An obvious penetrating globe injury is seen once lids are carefully retracted. The lower lid laceration makes this a high risk for globe penetration. Intraocular foreign body should be suspected. (Photo contributor: David Effron, MD.)

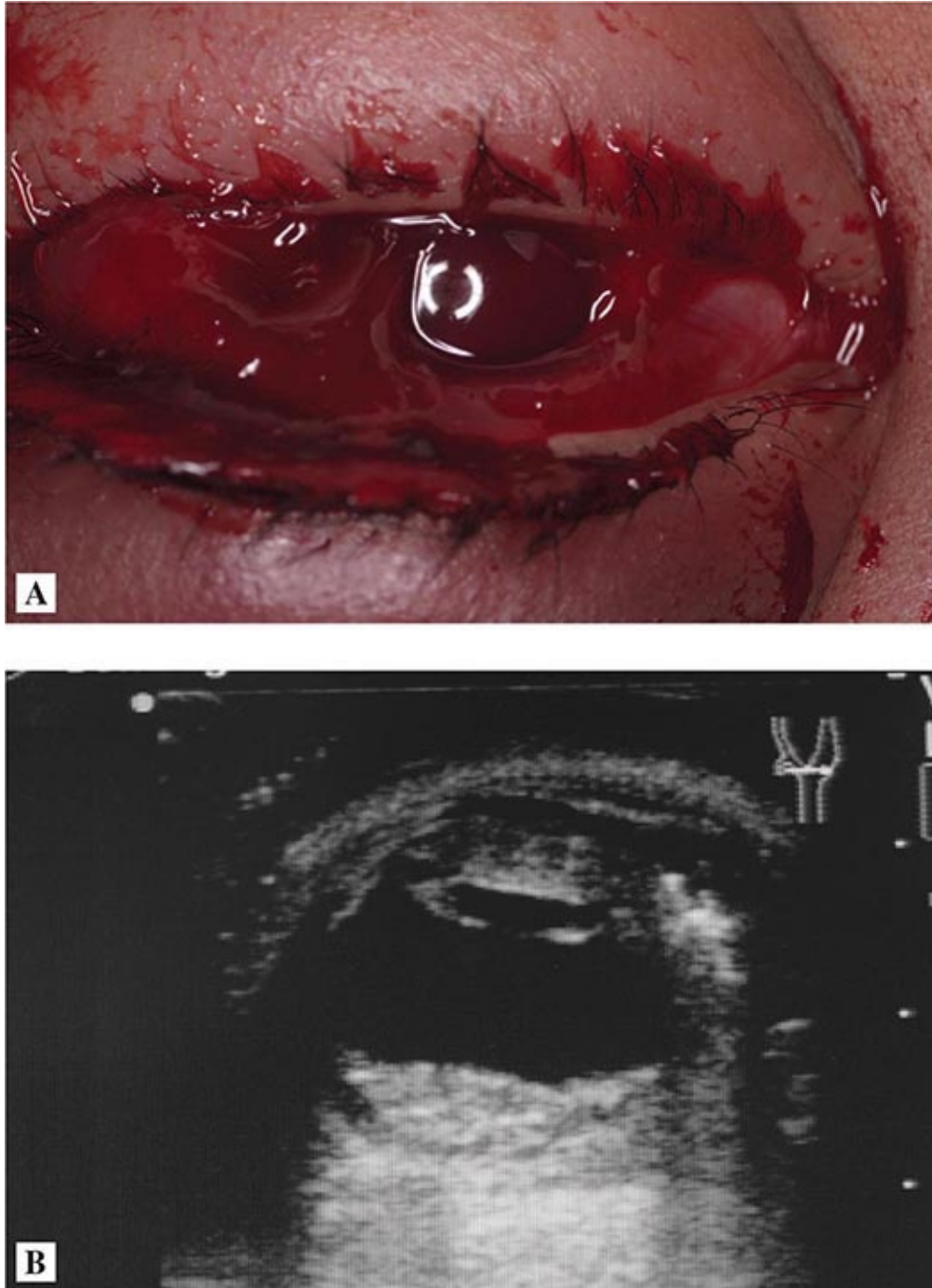


FIGURE 4.29 ■ Penetrating Globe. (A) This penetrating injury shows an “eight-ball” hyphema. (B) Bedside ultrasound shows both intraocular and anterior chamber hemorrhage. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Stabilize protruding FBs without manipulation until definitively treated in the

operating room.

2. Control of pain, activity, and nausea may be sight saving and requires proactive use of appropriate medications.
3. Use of lid retractors is preferred to open the eyelids of trauma victims with blepharospasm or massive swelling when such examination is indicated. Attempts to use fingers can inadvertently increase the pressure on the globe.
4. Emergency department ultrasound with a high-frequency probe using no pressure technique can be a useful adjunct to detect lens displacement, retinal detachment, and intraocular FB at the bedside. It is not useful to diagnose commotio retina, globe rupture, or retrobulbar hematoma.
5. Penetrating globe injuries are a relative contraindication to the sole use of depolarizing neuromuscular blockade (eg, succinylcholine).



FIGURE 4.30 ■ Eyelid Retractors. Retractors fashioned from paper clips can safely be used when standard retractors are not available. A lateral subconjunctival hemorrhage and associated lateral orbital wall fracture were seen in this patient. (Photo contributor: James K. Palma, MD.)



FIGURE 4.31 ■ Open Globe. A tear in the sclera and exposed vitreous is seen. A dark area on the sclera in the setting of trauma is an open globe injury until proven otherwise. (Photo contributor: Naval Medical Center, Portsmouth, VA.)

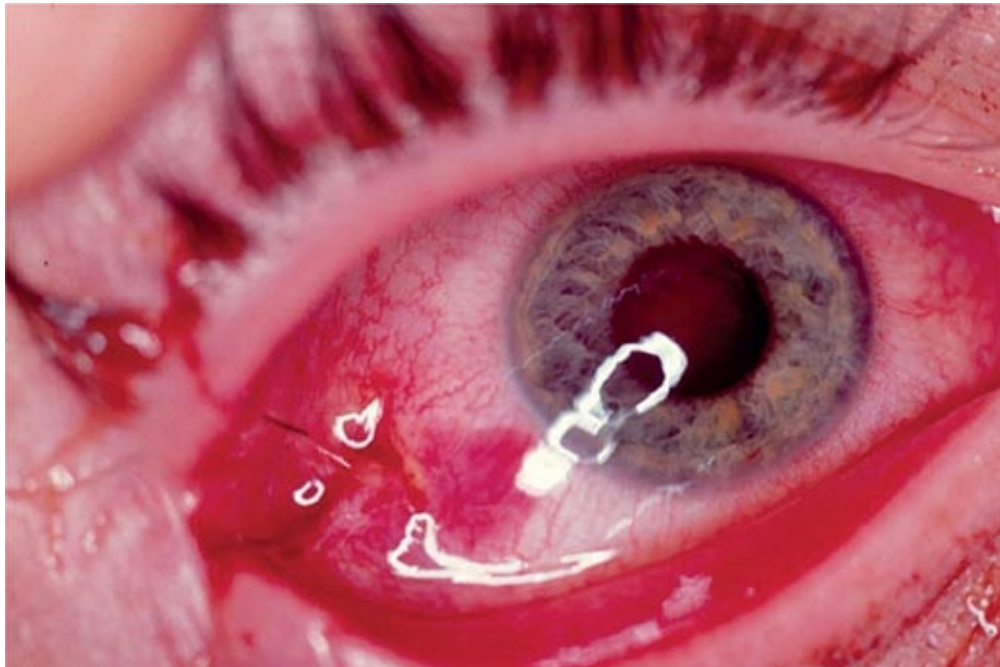


FIGURE 4.32 ■ Temporal Scleral Laceration. This was the entry site for a large intraocular foreign body. The pupil is round, and the anterior chamber appears normal, but the laceration was on the temporal sclera. (Photo contributor: Lawrence Lee, MD.)

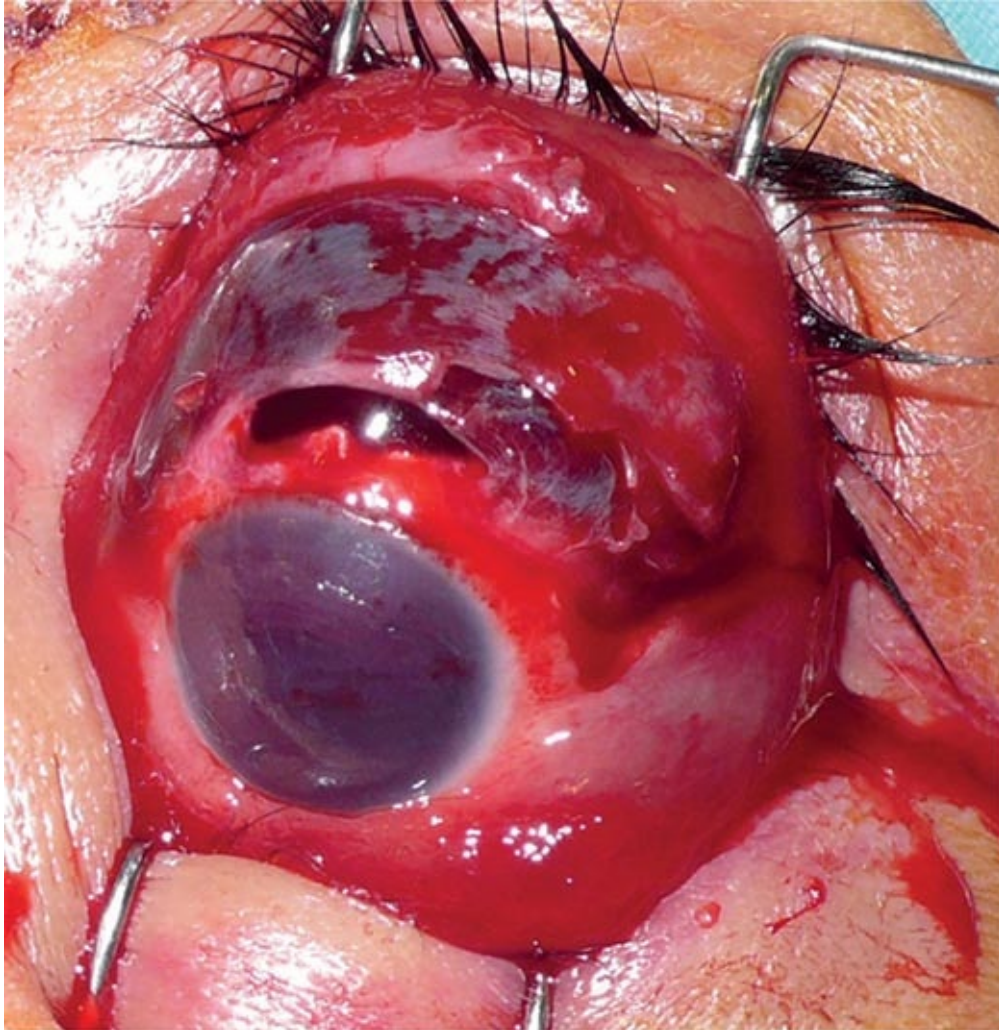


FIGURE 4.33 ■ Dense Subconjunctival Hemorrhage with Open Globe Injury. This patient was hit by a golf ball, causing a globe rupture. An elevated, dense, circumferential subconjunctival hemorrhage can hide an open globe injury. (Photo contributor: Hayyam Kiratli, MD.)

6. CT scanning is the most preferred modality to evaluate penetrating injuries; however, sensitivity is not sufficient for *ruling out* an open globe injury. Ophthalmology evaluation and possible surgical exploration are recommended.
7. Some nonmetallic objects such as wood, glass, or plastic may be difficult to visualize with CT. MRI is preferable in these cases and is excellent at demonstrating associated soft-tissue injuries to the globe and orbit. MRI is contraindicated when a metallic FB is suspected.



FIGURE 4.34 ■ Corneal Perforation and Rust Ring. A compressed air wire wheel brush lodged in the cornea when a wire came loose. The wire was removed in the operating room where it was found to penetrate fully through the cornea and required one corneal suture. The rust ring had formed around the wire only about 3 hours after this injury occurred. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)



FIGURE 4.35 ■ Protective Metal (Fox) Shield. A protective shield is used in the setting of a suspected or confirmed perforating injury. (Photo contributor: Kevin J. Knoop, MD, MS.)

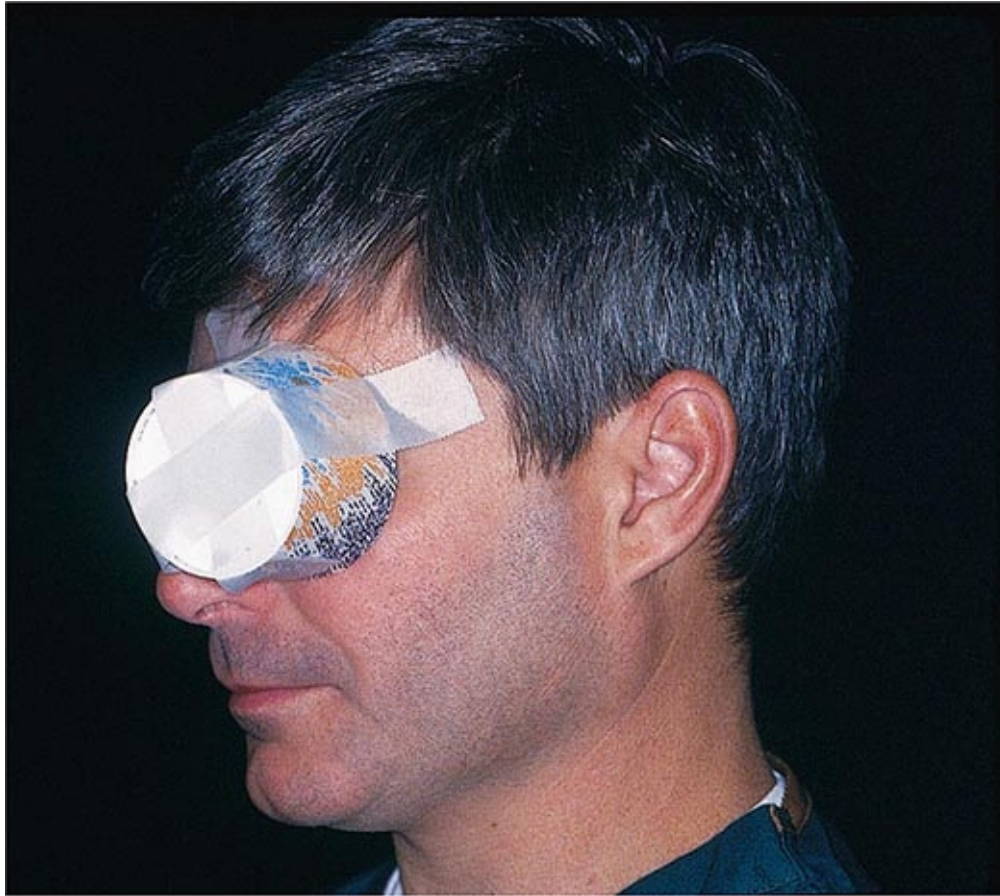


FIGURE 4.36 ■ Protective Shield. A protective shield is readily fashioned from a paper cup if a metal shield is not available. (Photo contributor: Kevin J. Knoop, MD, MS.)

GLOBE RUPTURE

Clinical Summary

Blunt or penetrating trauma to the eye may result in a ruptured globe. The diagnosis is obvious when orbital contents spill from the globe itself. Occult presentations manifest as a tiny rent in the sclera. Ruptures at the limbus, the margin between the cornea and sclera, may cause a small amount of iris to herniate, resulting in an irregularly shaped “teardrop” or “peaked” pupil. Choroidal rupture may also occur. A coloboma of the iris may appear similar to a teardrop pupil.

Management and Disposition

Obtain urgent specialty consultation. Protect the eye with a rigid eye shield, and defer all further examination and manipulation of the eye. Administer antibiotics and adequate sedation (parenteral analgesics are appropriate). Address tetanus status. Since vomiting may result in further prolapse of intraocular contents, treat with antiemetics proactively. Consider CT scanning if the presence of a FB is suspected.

Pearls

1. Suspect a rupture with severe conjunctival hemorrhage following trauma.
2. Rupture usually occurs where the sclera is thinnest, at the attachment of extraocular muscles and the limbus.
3. A teardrop pupil may easily be overlooked in the triage process or in the setting of multiple traumatic injuries.



FIGURE 4.37 ■ Prolapsed Iris. A teardrop pupil is present, with a small amount of iris herniating from a rupture at 8-o'clock position on the limbus. (Photo contributor: Lawrence B. Stack, MD.)

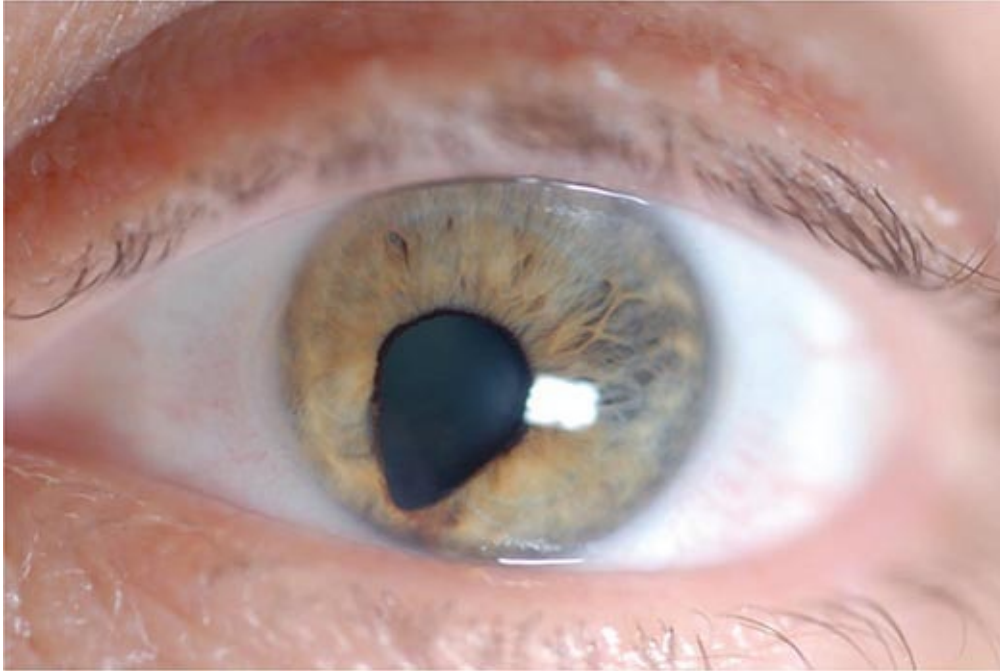


FIGURE 4.38 ■ Iris Coloboma. Iris coloboma is a congenital finding resulting from incomplete closure of the fetal ocular cleft. It appears as a teardrop pupil and may be mistaken for a sign of scleral rupture. (Photo contributor: R. Jason Thurman, MD.)

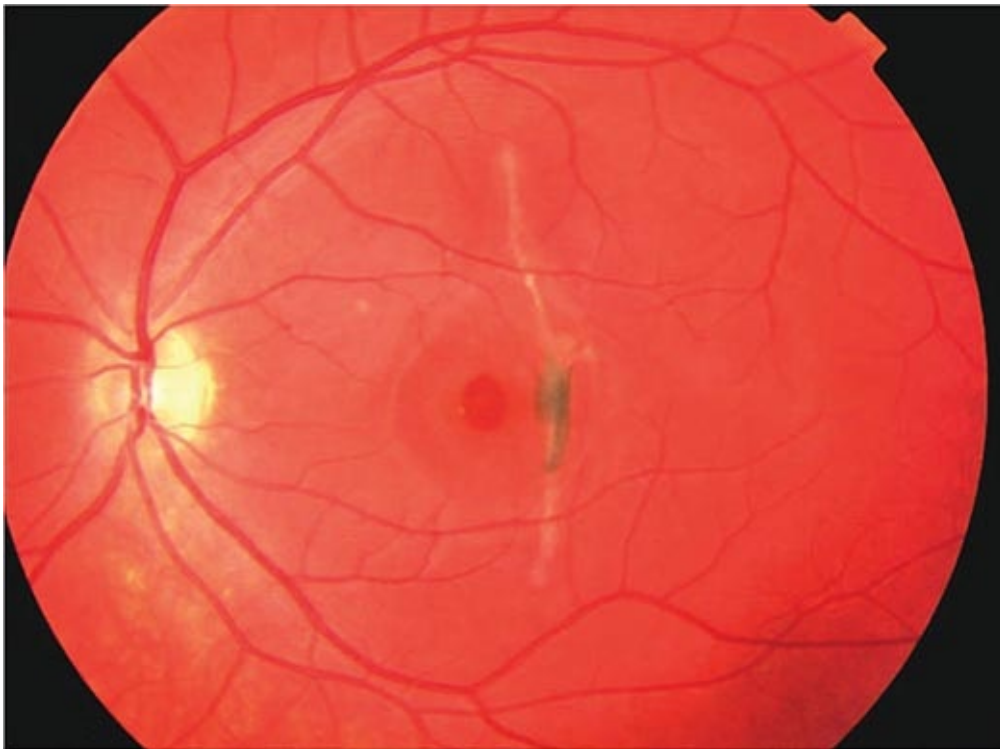


FIGURE 4.39 ■ Choroidal Rupture. A traumatic choroidal rupture appears as a vertical linear line on the temporal aspect of the macula. There is pigmentation within the choroidal rupture and an adjacent traumatic macular hole. (Photo contributor: Lawrence Lee, MD.)

TRAUMATIC CATARACT

Clinical Summary

Any trauma to the eye that disrupts the normal architecture of the lens may result in the development of a traumatic cataract—a lens opacity. The mechanism behind cataract formation involves fluid infiltration into the normally avascular and acellular lens stroma. The lens may be observed to swell with fluid and become cloudy and opacified. The time course is usually weeks to months following the original insult. Cataracts that are large enough may be observed by the naked eye. Those that are within the central visual field may cause blurring of vision or distortion of light around objects (eg, halos).

Management and Disposition

No specific treatment is rendered in the emergency department for cases of delayed traumatic cataract. Most cases require routine ophthalmologic referral.

Pearls

1. Traumatic cataracts are frequent sequelae of lightning injury. Advise all victims of lightning strike of this possibility.
2. Cataracts may also occur as a result of electric current injury to the vicinity of the cranial vault.
3. Leukocoria results from a dense cataract, which causes loss of the red reflex.
4. If a cataract develops sufficient size and “swells” the lens, the trabecular meshwork may become blocked, producing glaucoma.

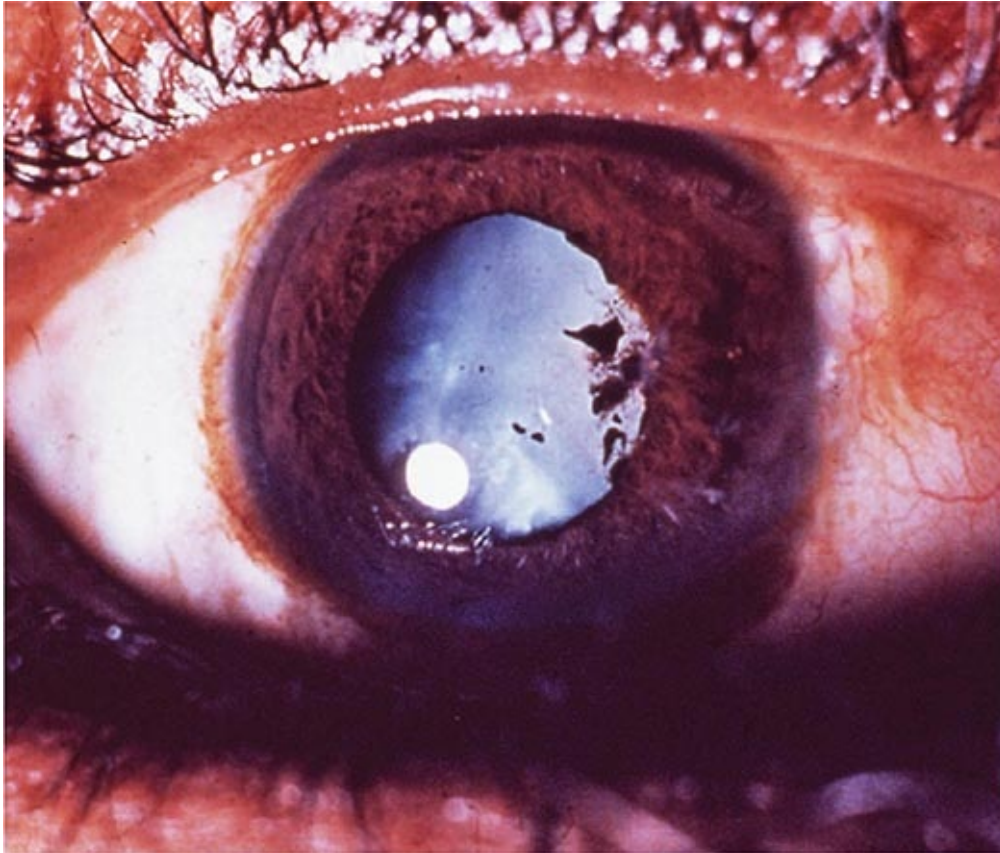


FIGURE 4.40 ■ Traumatic Cataract. This mature traumatic cataract is seen as a large lens opacity overlying the visual axis. A traumatic iridodialysis is also present. (Photo contributor: David Effron, MD.)

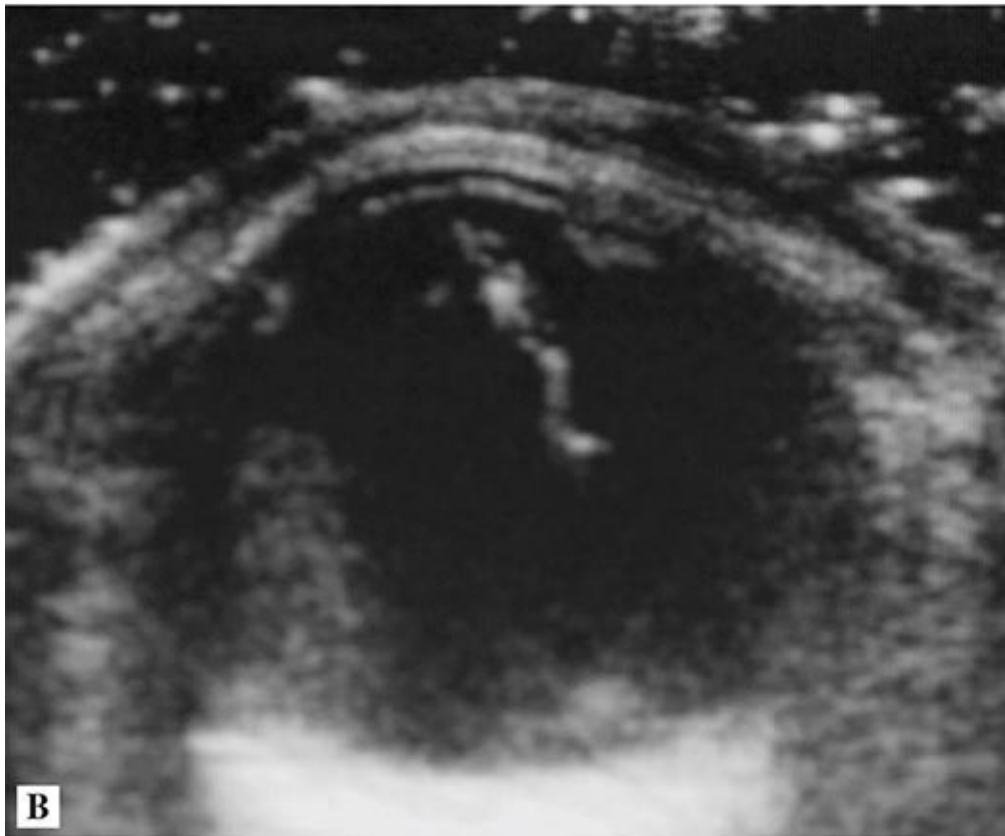


FIGURE 4.41 ■ Traumatic Cataract. (A) This acute traumatic cataract is seen as a milky cornea at the time of injury. (B) Bedside ultra-sound shows a collapsed anterior chamber and intraocular foreign body. (Photo contributor: Kevin J. Knoop, MD, MS.)

EYELID LACERATION

Clinical Summary

Eyelid lacerations should always prompt a thorough search for associated injury to the globe, penetration of the orbit, or involvement of other adnexal structures (eg, lacrimal glands, canaliculi, puncta). Depending on the mechanism of injury, a careful exclusion of FB is important.

Management and Disposition

Repair eyelid lacerations involving superficial skin with 6-0 nonabsorbable interrupted sutures, which should remain in place for 3 to 5 days. Lacerations through an anatomic structure called the gray line, situated on the palpebral edge, require diligent reapproximation, making referral prudent. Other injuries that require specialty consultation for repair include:

- Lacerations through the lid margins: these require exact realignment to avoid entropion or ectropion.
- Deep lacerations through the upper lid that divide the levator palpebrae muscles or their tendinous attachments: these require repair with fine absorbable suture to avoid ptosis.
- Lacrimal duct injuries: these are repaired by stenting of the duct to avoid permanent epiphora.
- Medial canthal ligaments: these require repair to avoid drooping of the lids and telecanthus.

The most important objectives are to rule out injury to the globe and to search diligently for FBs.

Pearls

1. Lacerations of the medial one-third of the lid or epiphora (tearing) raises suspicion for injury to the lacrimal system or the medial canthal ligament.
2. A small amount of adipose tissue seen within a laceration is a sign that perforation of the orbital septum has occurred (there is no subcutaneous fat in the eyelids).

3. Laceration of the levator palpebrae musculature or tendinous attachments may result in traumatic ptosis.

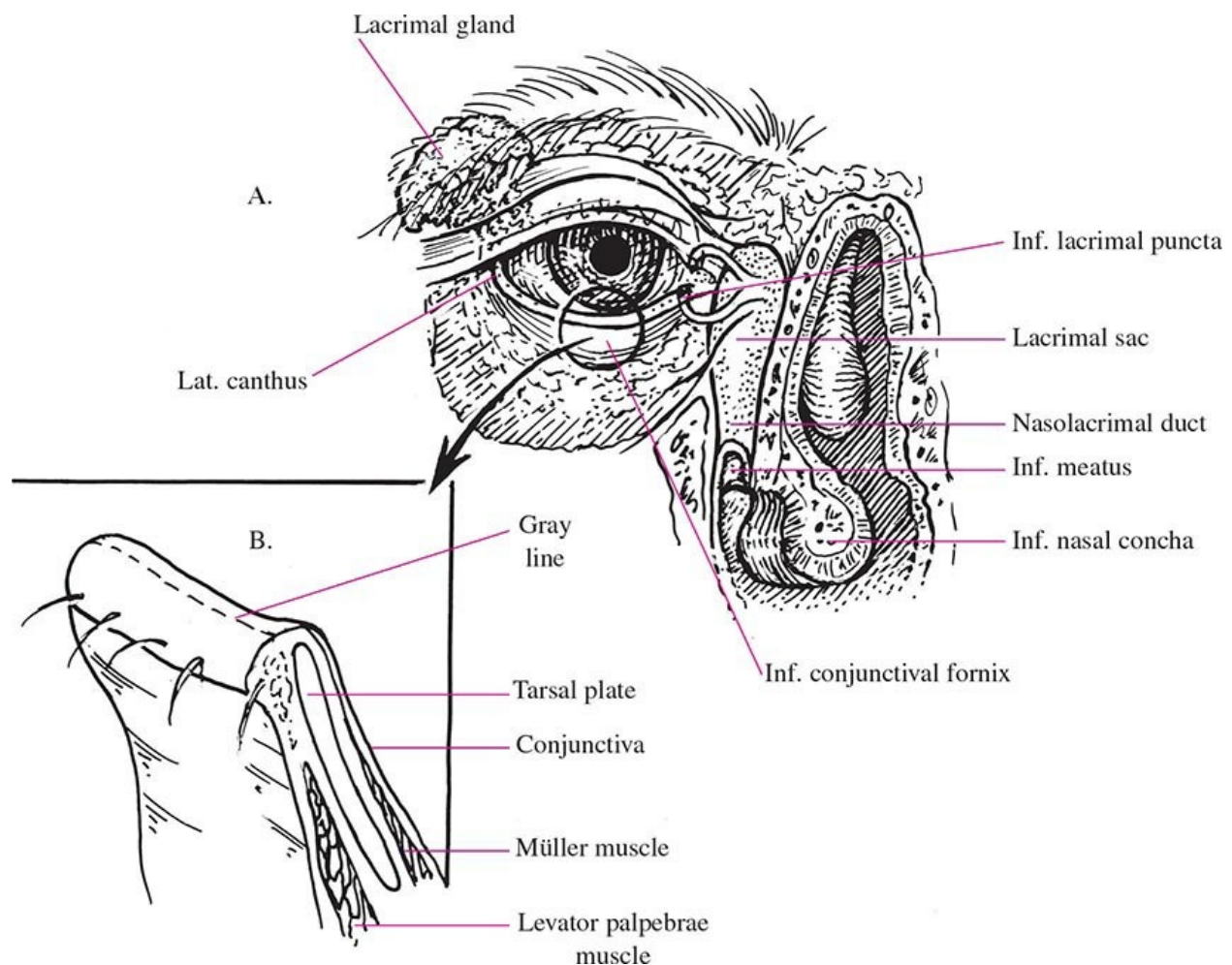


FIGURE 4.42 ■ Eyelid and Adnexa Anatomy. Ocular trauma should prompt examination of surrounding anatomic structures for associated injuries.

4. Laceration of the canthal ligamentous support is suggested when there is rounding of the lid margins or telecanthus (widening of the distance between the medial canthi).
5. Anesthesia of the forehead may result from supraorbital nerve injury and should be sought prior to instilling local anesthetics.



FIGURE 4.43 ■ Eyelid Laceration. This complex laceration showing the displaced inferior punctum clearly violates the canalicular structures. (Photo contributor: Harold Lee, MD.)

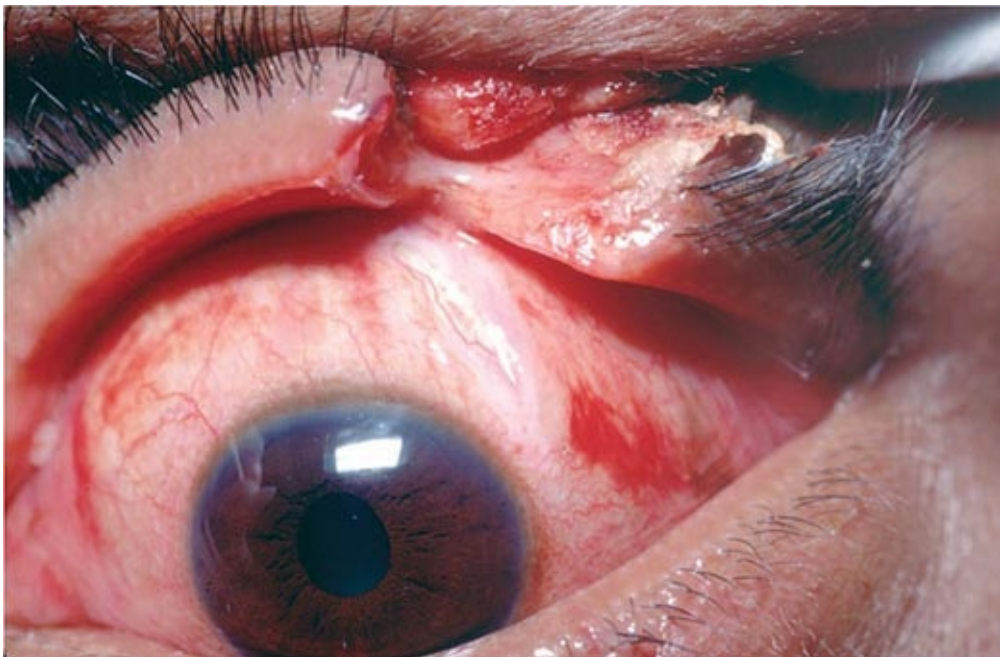


FIGURE 4.44 ■ Eyelid Laceration. Laceration to upper lid involving the tarsal plate. The assailant was wearing a ring. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 4.45 ■ Eyelid Laceration. Laceration involving the lid margin requires anatomic closure, ideally by a specialist. Careful approximation of the lid margins is required for adequate function. (Photo contributor: Kevin J. Knoop, MD, MS.)

IMPALED FOREIGN BODY

Clinical Summary

Impaled FB around the eye has a special apprehension because of the potential for loss of sight. The FB should be stabilized to prevent further damage. Nausea, retching, pain, coughing, and other symptoms should be aggressively controlled to prevent movement of the FB. Empiric broad-spectrum antibiotics should be administered. Tetanus immunization status should be addressed.

Plain x-rays are an acceptable starting point and screening tool, but typically, advanced imaging such as CT is indicated to thoroughly evaluate the depth of penetration, fragmentation of FB, degree of associated injuries, and proximity to sensitive structures. The possibility of intracranial involvement must always be considered. If organic materials are involved, CT may not fully evaluate the FB,

so alternate imaging such as MRI and/or ultrasound should be used. It is essential to collect as much information as possible regarding the location of the object and surrounding anatomy to determine the most appropriate surgical approach.

Management and Disposition

Head-to-toe trauma evaluation and resuscitation are completed in the emergency department. A multidisciplinary team may be required for management of impaled facial FB: anesthesiology for preoperative airway management to minimize movement of the FB, ophthalmology for eye injuries, and maxillofacial or otorhinolaryngology surgery for associated head and neck injuries. If there is intracranial involvement, neurosurgery will typically take first priority.

Pearls

1. Detailed ophthalmic and neurologic examinations are required upon presentation and serially to guide treatment decisions and to document deficits.
2. The face is highly vascular. The FB may be tamponading damaged blood vessels; movement or dislodgement can lead to torrential bleeding.



FIGURE 4.46 ■ Impaled Stick. A stick is impaled 7 cm into the medial aspect of the eye. Globe penetration was ruled out in the operating room. (Photo contributor: Elena Geraymovych, MD.)

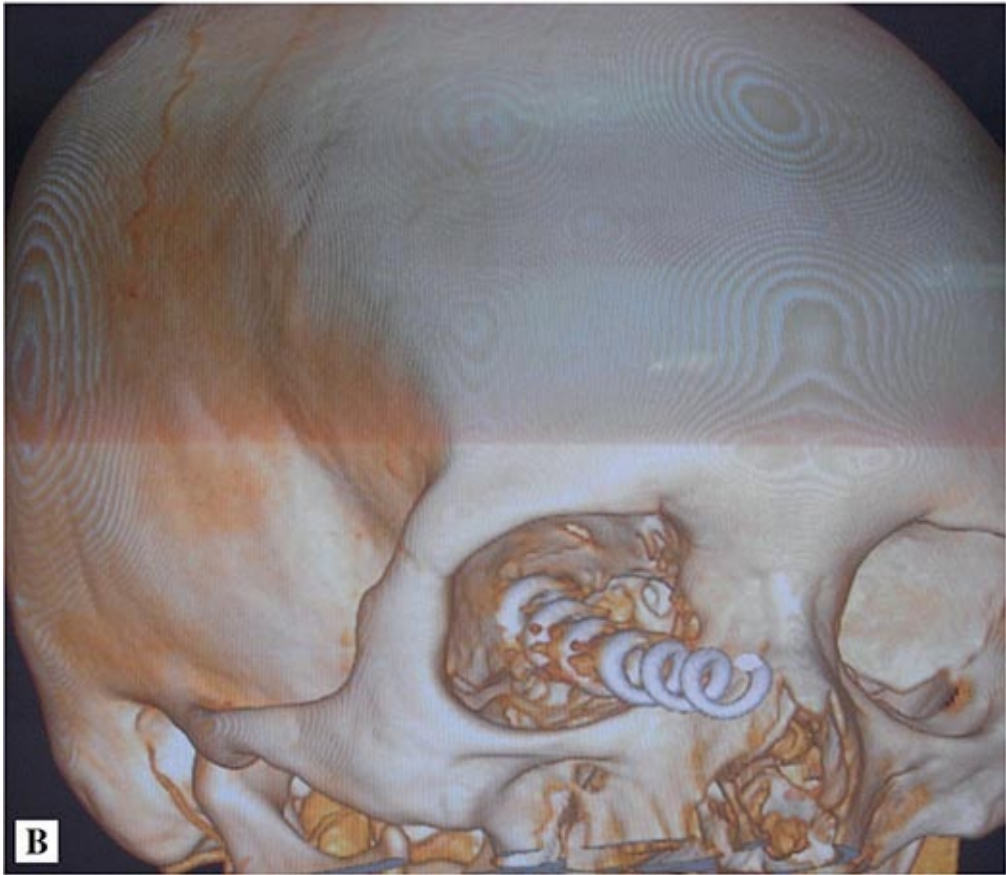
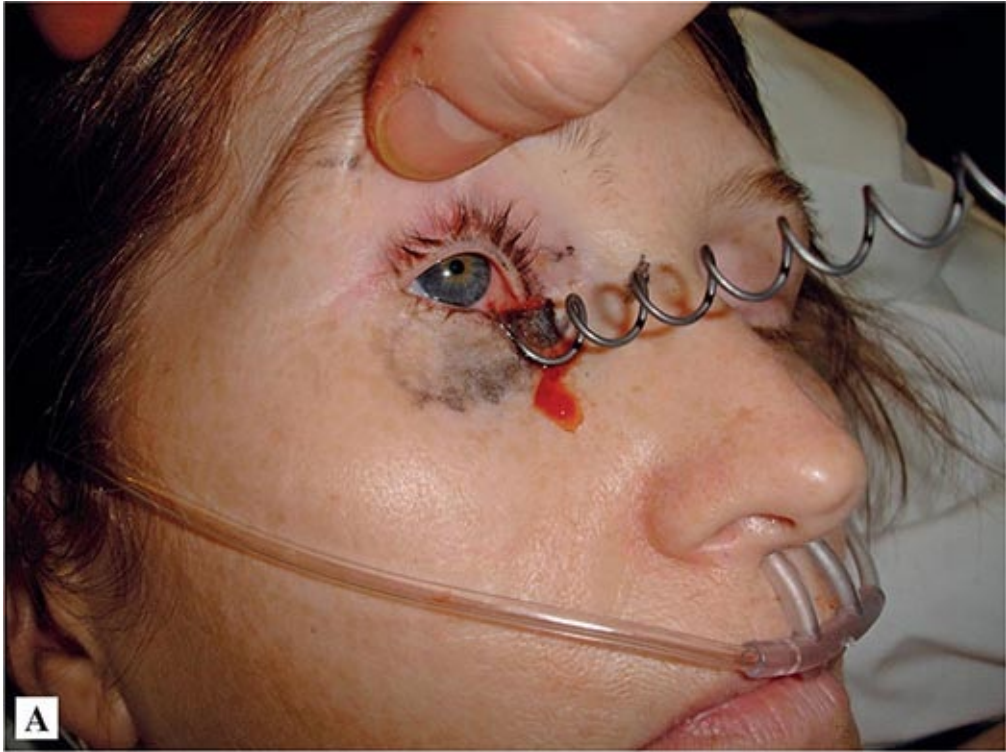


FIGURE 4.47 ■ Impaled Spring. (A) A spring impaled into the periocular structures is seen. The orbit was not injured despite the appearance of the injury. (B) Three-dimensional reconstruction showing spring in relation to the orbit. (Photo contributor: Dallas E. Peak, MD.)

CHEMICAL EXPOSURE

Clinical Summary

Symptomatic ocular exposures involve either immediate or delayed onset of eye discomfort accompanied by itching, tearing, redness, photophobia, blurred vision, and/or FB sensation. Conjunctival injection or chemosis may be seen. Exposure to defensive sprays or riot-control agents (eg, Mace or tear gas) causes immediate onset of severe burning, intense tearing, blepharospasm, and nasal and oropharynx irritation.

Management and Disposition

Begin copious irrigation immediately at the scene. Ubiquitously available, tap water is appropriate initially; switch to lactated Ringer's or normal saline when available to limit corneal edema from hypotonic water. Acute caustic exposures are tri-aged to immediate treatment. Determine the conjunctival sac pH with a broad-range pH paper without delaying treatment. Continue irrigation until achieving a tear film pH of 7.4. Topical anesthetic drops permit examination and facilitate irrigation. Evert upper lid conjunctiva to examine for concretions. Alkali exposures penetrate deeper into tissues, causing more severe injury (liquefaction necrosis). Acids are less damaging (coagulation necrosis), which creates a barrier to further penetration. The exception is hydrofluoric acid, which acts as an alkali exposure.

Irrigate liberally after all exposures. Many chemicals merely cause irritative symptoms; however, some may also denude the corneal epithelium and inflame the anterior chamber. Perform slit-lamp examination to document corneal epithelial defects or anterior chamber inflammation. Cycloplegics may reduce ciliary spasm and pain. Address tetanus status.



FIGURE 4.48 ■ Alkali Burn. Diffuse opacification of the cornea occurred from a “lye” burn to the face. (Photo contributor: Stephen W. Corbett, MD.)

Pearls

1. Immediate onset of severe symptoms calls for immediate treatment and should prompt consideration of alkali or acid exposure.
2. Prolonged (up to 24 hours) irrigation may be needed for alkaline exposures.
3. Concretions from the exposure agent may form deep in the conjunctival fornices. Removal is critical to prevent ongoing injury.
4. A Morgan lens or other eye irrigation system is ideal for effective treatment as blepharospasm severely limits effectiveness of intravenous tubing alone.



FIGURE 4.49 ■ Alkali Burn. Diffuse fluorescein uptake of the entire cornea is seen from an alkali exposure. (Photo contributor: Sarah M. Escott, MD.)



FIGURE 4.50 ■ Caustic Burn Adhesions (Symblepharon). Scar-ring of both palpebral and bulbar conjunctivae results in severe adhesions between the lids and the globe. (Photo contributor: Arden H. Wander, MD.)

The authors acknowledge the special contributions of Christopher S. Weaver, Dallas E. Peak, and Carey D. Chisholm for contributions to prior editions.

Chapter 5

EAR, NOSE, AND THROAT CONDITIONS

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Mastoiditis. Postauricular redness, swelling, and proptosis in a young child with acute mastoiditis. (Photo contributor: Lawrence B. Stack, MD.)

OTITIS MEDIA

Clinical Summary

Children between the ages of 6 months and 2 years are at highest risk of developing acute otitis media (AOM). Children at increased risk of recurrent AOM contract their 1st episode prior to 12 months, have a sibling with a history of recurrent AOM, are in day care, or have parents who smoke.

AOM is an acute inflammation and effusion of the middle ear. Viral, bacterial, and fungal pathogens may cause AOM. The pathogenesis of bacterial AOM is eustachian tube dysfunction, typically following a viral infection, allowing retention of secretions (serous otitis) and seeding of bacteria. The most common bacterial isolates are *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*, and *Streptococcus pyogenes*. There is an increased prevalence of antimicrobial resistance for *S pneumoniae* and β -lactamase-producing strains of *H influenzae*. Vaccination for strains of *S pneumoniae* has resulted in a modest decrease in incidence of AOM.

Patient presentations and complaints vary with age. Infants with AOM have vague, nonspecific symptoms (irritability, lethargy, and decreased oral intake). Young children can be irritable, often febrile, and frequently pull at their ears, but they may also be completely asymptomatic. Older children and adults note ear pain, impaired hearing, and occasionally otorrhea.

Otoscopy should focus on color, position, translucency, and mobility of the tympanic membrane (TM). Compared with the TM of a normal ear, AOM causes the TM to appear dull, erythematous or injected, bulging, and less mobile. The light reflex, normal TM landmarks, and malleus become obscured. Pneumatic otoscopy and tympanometry enhance accuracy in diagnosing AOM.

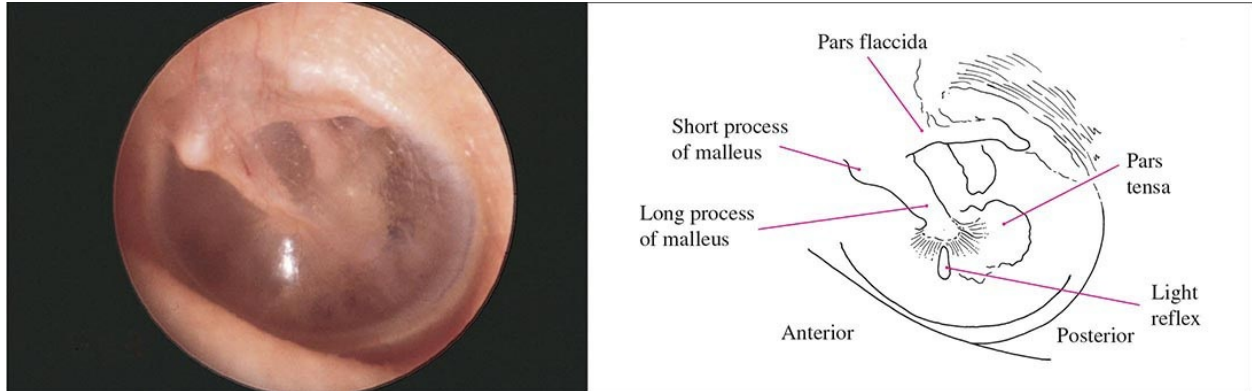


FIGURE 5.1 ■ Normal Left Tympanic Membrane. Normal tympanic membrane anatomy and landmarks. (Photo contributor: Richard A. Chole, MD, PhD.)



FIGURE 5.2 ■ Upper Respiratory Tract Infection Tympanic Membrane. Pink tympanic membrane often seen with fever or upper respiratory tract infections. Antibiotics are not indicated. (Photo contributor: Dr. Mike Starr. Used with permission, from resources at The Royal Children's Hospital, Melbourne, Australia; <https://www.rch.org.au/clinicalguide>.)

Management and Disposition

Although AOM generally resolves spontaneously, most patients are treated with

antibiotics and analgesics. Decongestants and antihistamines do not alter the course in AOM but may improve upper respiratory tract symptoms.

Follow-up in 10 to 14 days or return if symptoms persist or worsen after 48 hours is appropriate, and patients should be referred to an otolaryngologist for further evaluation, an audiogram, and possible tympanostomy tubes if they have significant hearing loss, failure of two complete courses of outpatient antibiotics during a single event, chronic otitis media (COM) with or without acute exacerbations, or failure of prophylactic antibiotics.

Pearls

1. AOM is a clinical diagnosis based on the presence of distinct fullness or bulging of the TM.
2. In children, recurrent otitis media (OM) may be due to food allergies.
3. Only 4% of children less than 2 years old with AOM develop temperatures greater than 104°F. Those with fever higher than 104°F or with signs of systemic toxicity should be closely evaluated for other causes before attributing the fever to AOM.
4. Most children over 2 years qualify for observation for 48 hours prior to initiation of antibiotics.
5. Management of AOM typically requires enteral analgesia; topical Auralgan may decrease pain.
6. TM perforation is common in patients with AOM but does not alter primary treatment.

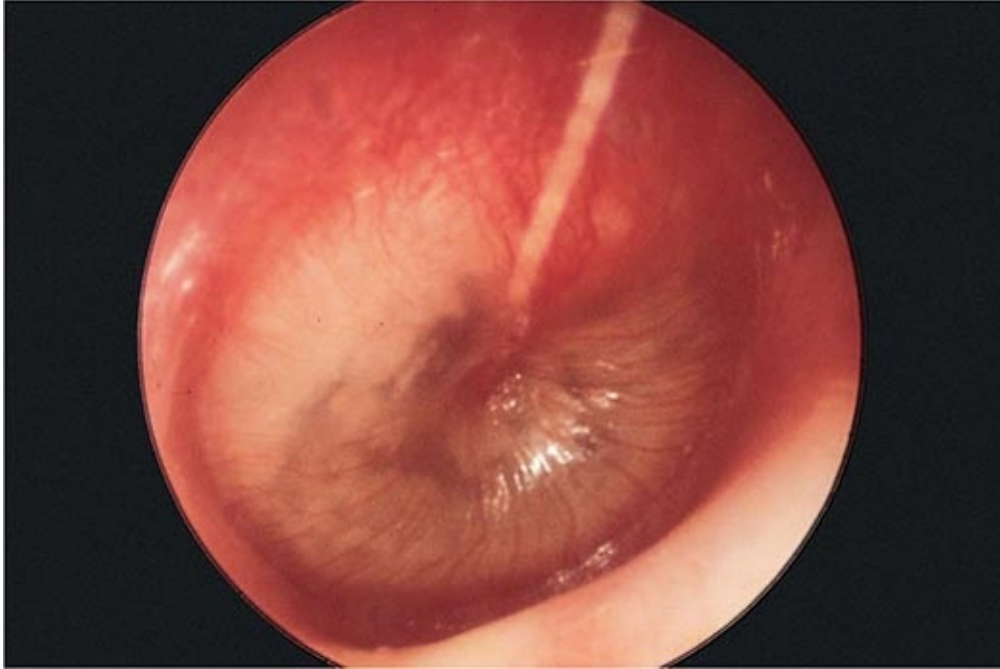


FIGURE 5.3 ■ Acute Otitis Media. The middle ear is filled with purulent material behind an erythematous, bulging tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)



FIGURE 5.4 ■ Serous Otitis Media with Effusion (OME). Serous OME is commonly seen after AOM, but it is also common without this history. Any process that leads to obstruction of a eustachian tube may cause OME. A clear, amber-colored effusion with a single air-fluid level is seen in the middle ear behind a normal tympanic membrane. (Photo contributor: C. Bruce MacDonald, MD.)



FIGURE 5.5 ■ Serous OME. A clear, amber-colored effusion with multiple air-fluid levels is seen in the middle ear behind a normal tympanic membrane. (Photo contributor: C. Bruce MacDonald, MD.)



FIGURE 5.6 ■ Tympanostomy Tube. Typical appearance of a tympanostomy tube in the tympanic membrane. These tubes will migrate to the periphery and eventually drop out. Occasionally, they will be found in the external ear canal. (Photo contributor: C. Bruce MacDonald, MD.)

BULLOUS MYRINGITIS

Clinical Summary

Bullous myringitis is a direct inflammation and infection of the TM secondary to a viral or bacterial agent. The hallmarks of bullous myringitis are vesicles or bullae filled with blood or serosanguinous fluid on an erythematous TM. Frequently, a concomitant OM with effusion is noted. Typical pathogens are the same as seen in AOM.

The onset of bullous myringitis is preceded by an upper respiratory tract infection and is heralded by sudden onset of severe ear pain, scant serosanguinous drainage from the ear canal, and frequently some degree of hearing loss. Otoscopy reveals bullae on either the inner or outer surface of the TM. Patients presenting with fever, hearing loss, and purulent drainage are more likely to have concomitant infections, such as OM and otitis externa.

Management and Disposition

Differentiation between viral and bacterial etiologies for TM bullae is not necessary. Although most episodes have a viral etiology and resolve spontaneously, many physicians prescribe antibiotics to cover presumptive *Mycoplasma pneumoniae*. Warm compresses, topical or strong analgesics, and oral decongestants provide symptomatic relief. Referral is not necessary in most cases unless rupture of the bullae is required for pain relief.

Pearls

1. Instruct parents that TM rupture may occur with sudden resolution of the pain and drainage from the ear canal.
2. Carefully differentiate TM bullae from cholesteatomas or herpetic vesicles.
3. Facial nerve paralysis associated with clear, fluid-filled TM vesicles is characteristic of herpes zoster oticus.



FIGURE 5.7 ■ Bullous Myringitis. A large fluid-filled bulla is seen distorting the surface of the tympanic membrane. (Photo contributor: Michael Hawke, MD, and The Hawke Library.)

CHOLESTEATOMA

Clinical Summary

Despite their name, cholesteatomas are not composed of cholesterol, nor are they a form of a malignancy. Cholesteatomas are collections of desquamating stratified squamous epithelium found in the middle ear or mastoid air cells. Congenital cholesteatomas occur most frequently in children and young adults. Acquired cholesteatomas originate from perforation or retraction of the TM allowing migration of epithelium into the middle ear.

Injury may occur to middle ear ossicles through the production of collagenases and may erode into the temporal bone, inner ear structures, mastoid

sinus, or posterior fossa dura. Treatment delays can lead to permanent conductive hearing loss or infectious complications.

Many cholesteatomas have an insidious progression without associated pain or symptoms. Computed tomography (CT) scans may reveal bony destruction.

Management and Disposition

Refer to otorhinolaryngology (ENT) upon initial diagnosis. No emergent medical or surgical management is required unless they become symptomatic.

Pearls

1. Persistent pain associated with headache, facial motor weakness, nystagmus, or vertigo suggests inner ear or intracranial involvement.
2. Polyps found on the TM may indicate the presence of a cholesteatoma and require further evaluation to exclude its presence.

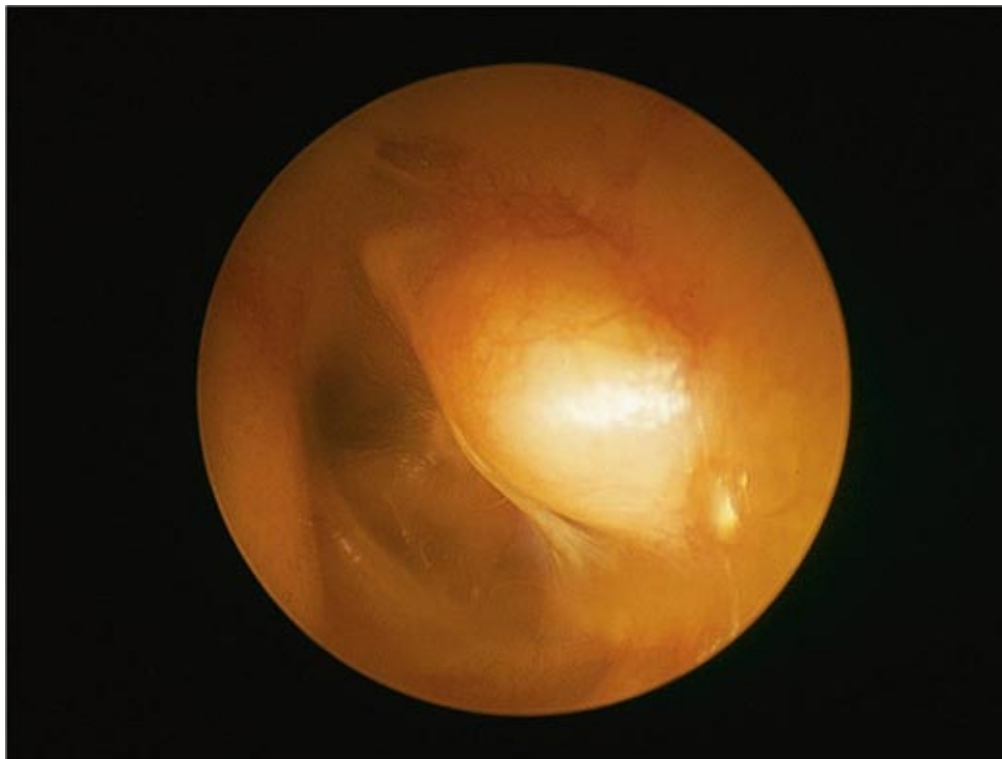


FIGURE 5.8 ■ Congenital Cholesteatoma. A congenital cholesteatoma is seen behind an intact tympanic membrane. (Photo contributor: C. Bruce MacDonald, MD.)



FIGURE 5.9 ■ Acquired Cholesteatoma. A large cholesteatoma is noted with significant distortion of the TM. Note the yellow epithelial debris from the cholesteatoma in the area of the pars flaccida. Often there is an effusion and debris, which can distort the anatomy on otoscopy. (Photo contributor: C. Bruce MacDonald, MD.)



FIGURE 5.10 ■ Acquired Cholesteatoma. This is a left ear with an inferior perforation and granulation tissue present just below the tip of the malleus. A white mass is seen behind the eardrum in the posterosuperior quadrant, roughly 12-o'clock to 3-o'clock position. This patient had a history of pressure equalizer (PE) tubes placed several years ago with a resultant perforation allowing squamous epithelium access to the middle ear. (Photo contributor: David R. White, MD.)

EXOSTOSIS

Clinical Summary

Exostoses are seen on otoscopy as discreet single or multiple round shiny swellings deep in the bony external auditory canal (EAC). They are usually asymptomatic, an incidental finding on exam, and extremely slow growing. The inferior canal walls anteriorly and posteriorly are hypertrophied, with a resultant “V” shape. Rarely they enlarge enough to occlude the EAC. They are usually bilateral and are thought to develop in response to repeated cold water exposure, such as frequent swimmer or divers. Hearing loss, ear infection, pain, and tinnitus may arise when they enlarge sufficiently to interfere with the normal self-cleansing of cerumen and desquamated keratin, leading to external otitis or conductive hearing loss from impacted cerumen.

Management and Disposition

Exostoses require no medical or surgical management unless they become symptomatic. Treatment then involves removal of impacted cerumen and/or treating otitis externa if present. For severe or recurrent symptoms, ENT referral for possible surgery is indicated.

Pearls

1. Exostosis is the most common tumor of the EAC.
2. Use of a Q-tip or examination with an otoscope speculum may cause trauma to the overlying skin in patients with exostosis.
3. In coastal areas, this condition is often referred to as “surfer’s ear.”



FIGURE 5.11 ■ Exostosis. Two exostoses are present: a large sessile anterior exostosis and a smaller pedunculated posterior exostosis. The tympanic membrane, malleus handle, and fibrous annulus are visible behind the exostoses. (Photo contributor: Michael Hawke, MD, and The Hawke Library.)



FIGURE 5.12 ■ Exostosis. A small sessile exostosis and a pedunculated exostosis are seen. (Photo contributor: Michael Hawke, MD, and The Hawke Library.)

TYMPANIC MEMBRANE PERFORATION

Clinical Summary

Acute tympanic membrane (TM) perforations may be caused by direct penetrating trauma, barotrauma, OM, corrosives, thermal injuries, or iatrogenic causes (foreign-body removal, tympanostomy tubes). TM perforations are occasionally accompanied by injuries to the ossicular chain and temporal bone.

Patients with an acute TM perforation complain of a sudden onset of ear pain, vertigo, tinnitus, and altered hearing after a specific event. Physical examination of the TM reveals a slit-shaped tear or a larger perforation with an irregular

border, often associated with blood along the margins. Subacute or chronic perforations have smooth margins and a round or ovoid shape.

Management and Disposition

Treatment of acute TM perforations is tailored to the mechanism of injury. All easily removable foreign bodies should be extracted. Corrosive exposures require face, eye, and ear decontamination. Antibiotics and irrigation do not improve the rate or completeness of healing unless the injury is associated with OM. Systemic antibiotics should be reserved for perforations associated with OM, penetrating injury, and possibly water-sport injuries (see “Otitis Media” earlier). Topical steroids impede perforation healing and should not be used.

Patients are instructed to avoid water in the ear while the perforation is healing and to return if symptoms of infection appear. While nearly 80% of all TM perforations heal spontaneously, all TM perforations require referral to an otolaryngologist for follow-up and for possible myringoplasty.

Pearls

1. Corticosteroid eardrops of any formulation retard spontaneous healing and should be avoided.
2. Topical ototoxic medications (eg, gentamycin, neomycin sulfate, tobramycin) should be avoided.
3. Traumatic TM perforation associated with cranial nerve (CN) deficits or persistent vertigo requires immediate otolaryngology consultation due to possible temporal bone fractures or injury to the round or oval window.

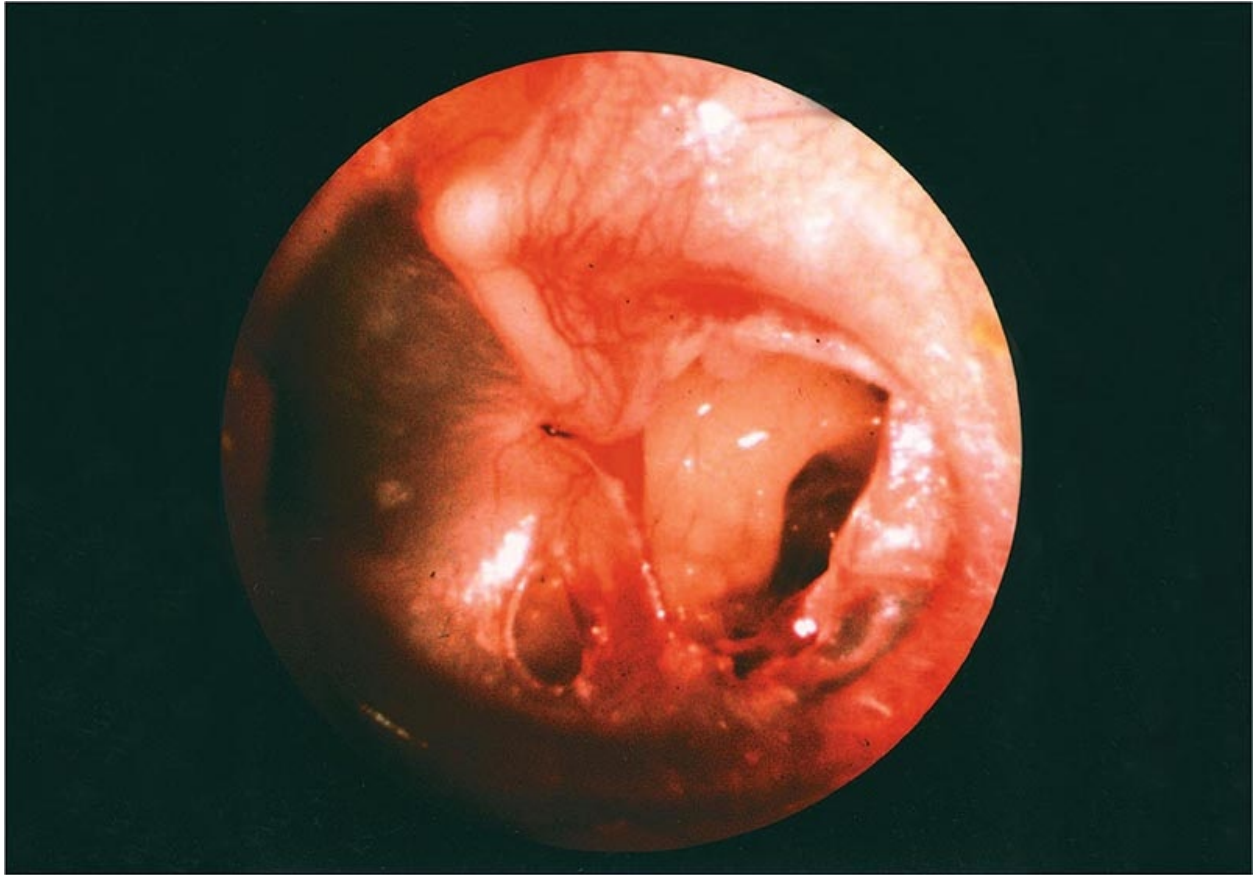


FIGURE 5.13 ■ Acute Tympanic Membrane Perforation. An acute tympanic membrane perforation is seen. Note the sharp edges of the ruptured tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)

OTITIS EXTERNA

Clinical Summary

Otitis externa (OE), or “swimmer’s ear,” is an inflammatory condition of the auricle and EAC, often with an accompanying infection (bacterial or fungal). Typical symptoms include otalgia, pruritus, otorrhea, and hearing loss. Physical examination reveals EAC hyperemia and edema, otorrhea, occlusion from debris and swelling, pain with manipulation of the tragus, and periauricular lymphadenopathy.

Several factors predispose the EAC to infection: increased humidity and heat, repeated water immersion, foreign bodies, in-ear headphone, trauma, hearing aids, and cerumen impaction. Bacterial OE is primarily an infection due to

Pseudomonas species or *Staphylococcus aureus*. Patients with diabetes are particularly prone to infections by *Pseudomonas*, *Candida albicans*, and, less commonly, *Aspergillus niger*.

Management and Disposition

EAC irrigation and suctioning are recommended to thoroughly evaluate the EAC. Topical antibiotic *suspensions* containing polymyxin, neomycin, and hydrocortisone or ciprofloxacin with ear wicks are effective. Topical *solutions* are not pH balanced and thus are irritating and may cause inflammation in the middle ear if a perforation is present. Topical fluoroquinolones may be less irritating and are only given twice a day. Systemic antibiotics are not indicated unless extension into the periauricular tissues is noted. Patients should avoid swimming and should prevent water from entering the ear while bathing. Dry heat aids in resolution, and analgesics provide symptomatic relief. Follow-up should be arranged in 10 days for routine cases.

Pearls

1. Resistant cases may have an allergic or eczematous component. These typically present with a dry, scaly, itchy EAC and are chronic in nature.



FIGURE 5.14 ■ Otitis Externa. A discharge is seen coming from the external auditory canal, which is swollen and almost completely occluded. An ear wick placed in the EAC facilitates delivery of topical antibiotic suspension and drainage of debris. (Photo contributor: Frank Birinyi, MD.)

2. Drying the EAC after water exposure with a 50:50 mixture of isopropyl alcohol and water or with acetic acid (white vinegar) minimizes recurrence. If the TM is possibly perforated, isopropyl alcohol should be avoided.
3. If a TM perforation is suspected and antibiotic drops are indicated, a suspension is recommended.
4. Consider malignant OE, typically caused by *Pseudomonas aeruginosa*, in elderly, diabetics, or immunocompromised patients. Exposed bone, ulceration of the EAC, and facial nerve weakness are hallmarks. Treatment for malignant OE is more intensive and focused on the presumptive organism.



FIGURE 5.15 ■ *Aspergillus Otitis Externa*. Chronic otitis externa with copious debris, including black spores from *A niger*, cottony fungal elements, and wet debris. This patient had been treated with topical and systemic antibiotics. (Photo contributor: C. Bruce MacDonald, MD.)

PREAURICULAR SINUS ABSCESS

Clinical Summary

A preauricular sinus is a common congenital malformation of the preauricular soft tissues. It is a sinus located near the front of the ear and is lined with squamous epithelium and thus may produce epithelial-lined subcutaneous cysts, which may become infected, leading to cellulitis or abscess. Patients may have other congenital anomalies such as hearing loss or renal disease. Patients may present with recurrent ear discharge, pain, swelling, redness, itching, headache, and fever. The diagnosis is often overlooked in early stages.

Management and Disposition

Initiate oral antibiotics to cover for *S aureus* (most common). Incision and

drainage should be avoided in the emergency department (ED). Needle aspiration may be attempted and may give some relief. Discharge patients with antibiotics and instructions for using a warm compress and oral analgesics. Follow-up with an otolaryngologist for complete excision of the sinus track is indicated.

Pearls

1. Preauricular sinus abscesses may be confused with 1st branchial cleft cysts.
2. Because of their close proximity to the facial nerve, referral to a specialist who is familiar with the underlying anatomy is recommended.



FIGURE 5.16 ■ Preauricular Sinus Abscess. Recurrent preauricular swelling and redness in the area of a “pit” suggests sinus abscess and was confirmed on contrast-enhanced CT scan. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.17 ■ Preauricular Sinus Abscess. Painful preauricular swelling and redness and a visible “pit” are seen. A loculated sinus abscess was confirmed on contrast-enhanced CT scan. Facial and periorbital edema were present. (Photo contributor: Kevin J. Knoop, MD, MS.)

MASTOIDITIS

Clinical Summary

Mastoiditis is an infection or inflammation of the mastoid air cells often resulting from extension of purulent OM with progressive destruction and coalescence of air cells. Medial sinus wall erosion can cause cavernous sinus thrombosis, facial nerve palsy, meningitis, brain abscess, and sepsis. With the use of antibiotics for AOM, the incidence of mastoiditis has fallen sharply.

Patients present with fever, chills, postauricular ear pain, and otorrhea. Patients may have tenderness, erythema, swelling, and fluctuance over the

mastoid process; proptosis of the pinna; erythema of the posterior-superior EAC wall; and otorrhea.

Management and Disposition

Initial evaluation includes a thorough head, neck, and cranial nerve examination. A complete blood count and sedimentation rate may be obtained to establish a baseline for treatment efficacy assessment. Contrasted CT of the head or mastoid sinus may reveal bone erosion and intracranial involvement.

While oral penicillinase-resistant penicillins, amoxicillin-clavulanic acid, 3rd-generation cephalosporins, and the newer macrolides are effective in mild cases of mastoiditis, severe cases require parenteral antibiotics. Mastoiditis requires prompt consultation and close follow-up.

Pearls

1. Most patients require admission for parenteral antibiotics to cover *S pneumoniae*, *H influenzae*, *M catarrhalis*, streptococcal species, and *S aureus*.
2. Surgical incision and debridement, and possibly mastoidectomy, are reserved for refractory cases.
3. Chronic mastoiditis describes chronic otorrhea of at least 2 months in duration. It is often associated with craniofacial anomalies.
4. Consider a branchial cleft cyst in the differential diagnosis with periauricular swelling.



FIGURE 5.18 ■ Acute Mastoiditis. Postauricular swelling, redness, and proptosis in a young girl with acute mastoiditis and sinusitis. (Photo contributor: Lawrence B. Stack, MD.)

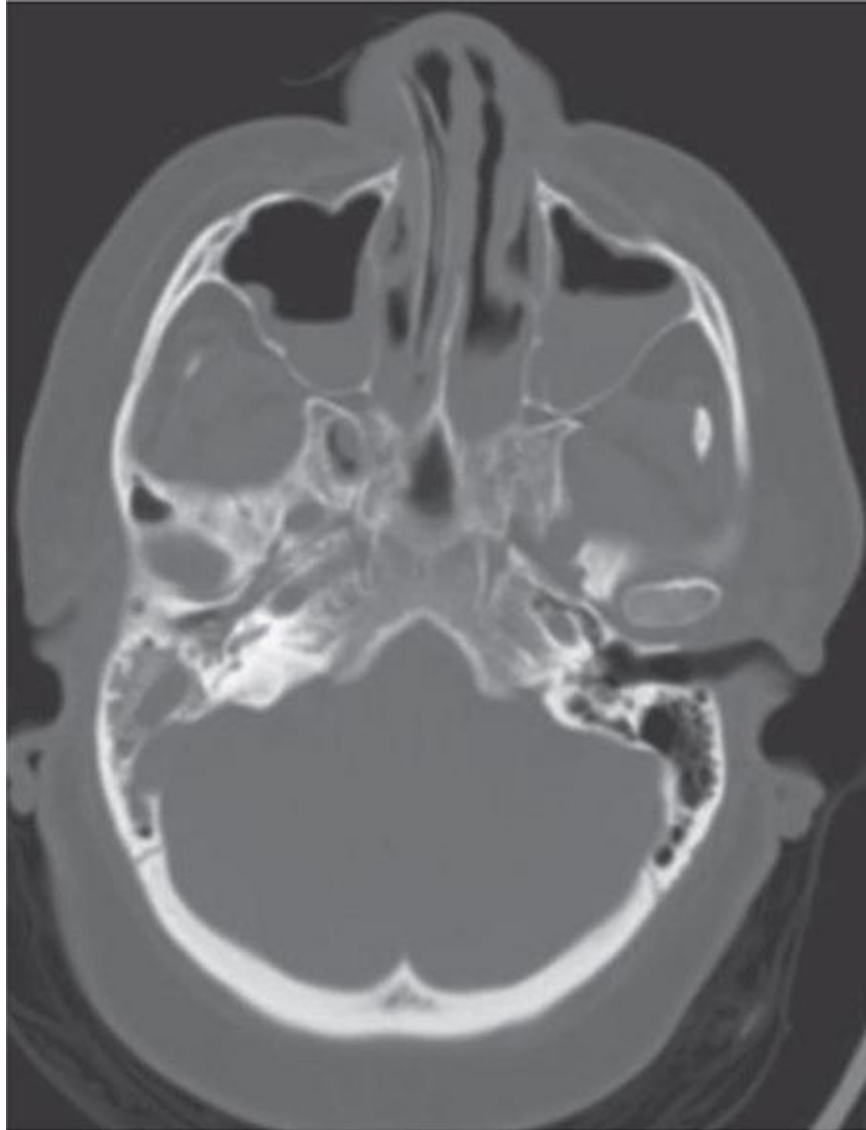


FIGURE 5.19 ■ Acute Mastoiditis. Bone window of a head CT scan which demonstrates fluid in the right mastoid air cells. Maxillary sinus disease is also present. (Photo contributor: Lawrence B. Stack, MD.)

AURICULAR PERICHONDritis

Clinical Summary

Auricular perichondritis is a bacterial infection of the overlying skin and perichondrium of the ear, by definition sparing the auricular cartilage. It may be an extension of OE. Causative organisms include *P aeruginosa*, *S aureus*, and *S pyogenes*. Pre-disposing factors include surgery, ear piercing, burns, frostbite,

insect bites, and contact sports. Clinical findings include a swollen, tender, erythematous, and warm auricle, which may involve the ear lobule, and often a fever; the TM is unaffected. Infectious perichondritis may be confused with relapsing polychondritis, an autoimmune condition involving the cartilage of the ears, nose, and trachea.

Management and Disposition

Institute oral antibiotics with *Pseudomonas* coverage and follow up in 48 hours; however, hospitalization is often required for parenteral antibiotics in children and immunocompromised or diabetic patients. Topical antibiotic otic drops should be used if OE is present.

Pearls

1. *P aeruginosa* is the most common bacteria causing auricular perichondritis.
2. Ear piercing is the most common activity resulting in auricular perichondritis.
3. Fluctuance and auricular deformity suggest auricular chondritis, a frequent complication of perichondritis.
4. Consult ENT for abscess drainage to limit permanent ear disfigurement.



FIGURE 5.20 ■ Perichondritis. Swollen and erythematous pinna, excluding the earlobe with no concomitant otitis externa, mastoiditis, or furuncle. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.21 ■ Perichondritis. Erythema, swelling, and tenderness seen in the pinna with multiple ear piercings. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.22 ■ Auricular Chondritis. Deformity of the auricular cartilage is seen. Ear piercing caused the initial insult to this pinna. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.23 ■ Relapsing Polychondritis. Erythema of the right pinna that spares the lobe. A month prior, the left pinna was involved and was treated as infectious perichondritis. (Photo contributor: Lawrence B. Stack, MD.)

HERPES ZOSTER OTICUS (RAMSAY HUNT SYNDROME)

Clinical Summary

Herpes zoster oticus (HZO), or Ramsay Hunt syndrome, is the second most common cause of facial paralysis, representing 3% to 12% of cases. The syndrome consists of facial and neck pain, auditory symptoms, and facial palsy associated with the reactivation of latent varicella-zoster virus in the facial nerve

and geniculate ganglion. Patients first note pruritus, followed by pain out of proportion to the physical examination over the face and ear. Patients may also experience vertigo, hearing loss from involvement of the 8th cranial nerve, tinnitus, rapid onset of facial paralysis, decrease in salivation, loss of taste sensation over the posterolateral tongue, and vesicles on the ear, EAC, and face.

Management and Disposition

The diagnosis of HZO is based largely on history and physical examination. Tzanck preparations may be difficult because of the vesicles' location. Magnetic resonance imaging (MRI) with contrast, if performed, may show enhancement of the geniculate ganglion and facial nerve.

Oral antivirals and steroids are mainstays of treatment; earlier initiation is associated with the highest rates of improvement. As with Bell palsy, it is important to protect the involved eye from corneal abrasions and ulcerations by using lubricating drops. Referral to a specialist should be made for follow-up care.

Pearls

1. The prognosis for facial paralysis due to HZO is worse than that for Bell palsy. Approximately 10% and 66% of patients with full and partial facial paralysis, respectively, recover fully. The prognosis improves if the symptoms of HZO are preceded by the vesicular eruption.
2. The combination of antivirals with steroids produces better outcomes than with either agent alone.

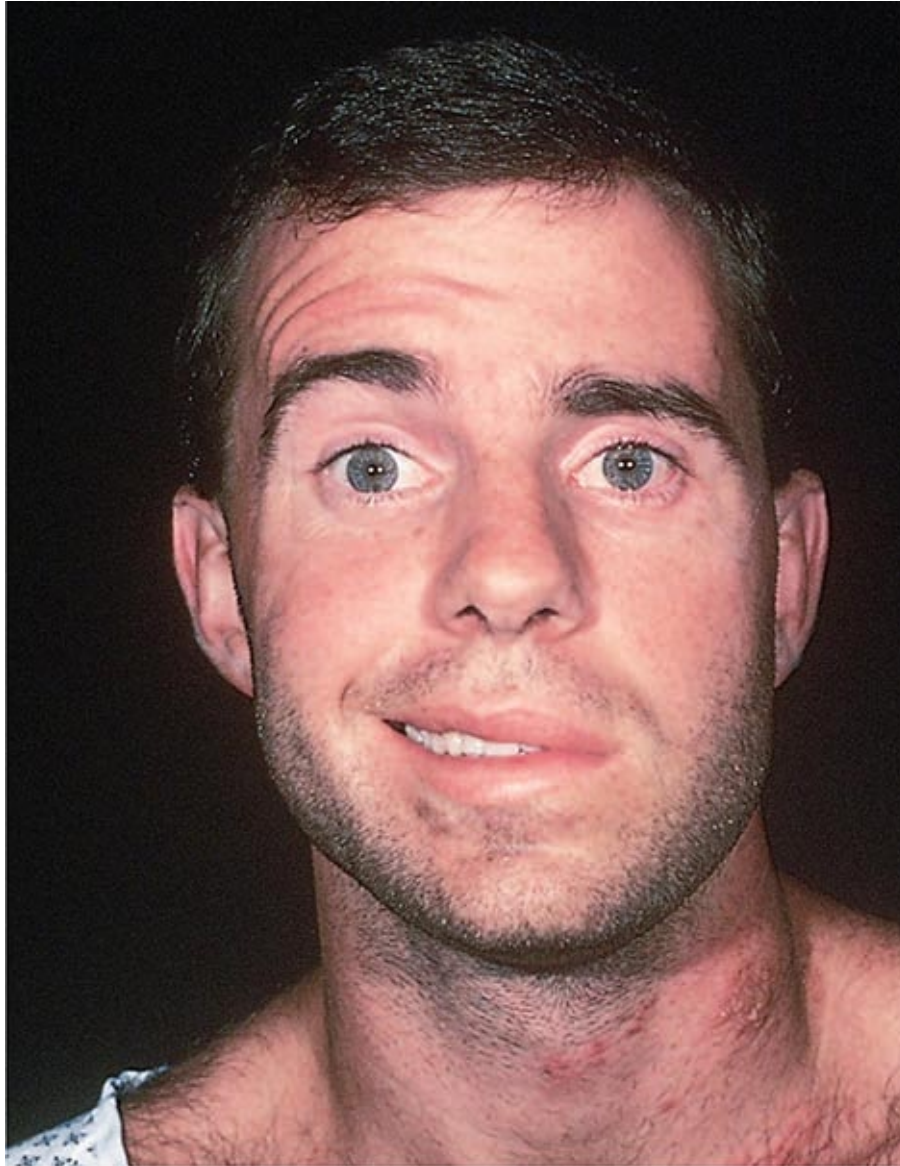


FIGURE 5.24 ■ Herpes Zoster Oticus. Facial palsy in a young adult. Note the vesicular eruptions on the neck. (Photo contributor: Frank Birinyi, MD.)



FIGURE 5.25 ■ Herpes Zoster Oticus. On closer examination, the vesicles extend up the neck to the external auditory canal. (Photo contributor: Frank Birinyi, MD.)



FIGURE 5.26 ■ Herpes Zoster Oticus. Erythema and drainage coming from the EAC are seen in this patient with herpes zoster oticus. Otitis externa can have a similar appearance but does not have vesicles, as seen in this patient. (Photo contributor: David Effron, MD.)



FIGURE 5.27 ■ Herpes Zoster Oticus. Vesicles of zoster are clearly seen in this patient. (Photo contributor: Lawrence B. Stack, MD.)

FACIAL NERVE PALSY

Clinical Summary

CN VII innervates the facial muscles via the five branches of the motor root, the submandibular, sublingual, and lacrimal glands, and the taste organs on the anterior two-thirds of the tongue; and it provides sensation to the pinna of the

ear. Facial palsies are either central or peripheral. Central lesions occur proximal to the CN VII nucleus in the pons. Lesions distal to the nucleus are classified as peripheral lesions. The ipsilateral frontalis muscle is functional or “spared” in central lesions since it receives innervation in the nucleus from both ipsilateral and contralateral motor cortices. Peripheral injuries involve the entire side of the face, including the forehead; thus, the forehead is not “spared.”

Most commonly, 7th-nerve dysfunction is idiopathic (Bell palsy). One percent of patients have bilateral involvement; 60% have a viral prodrome. There is no age, sex, or racial predilection. Patients note an acute onset over a few hours to days of facial weakness and may have numbness or pain on the ipsilateral face, ear, tongue, and neck, as well as a decrease or loss of ipsilateral tearing and saliva flow. Hearing is most commonly preserved.

Prognosis is variable. Facial weakness has a better prognosis for full recovery than complete paralysis. Palsies due to herpes zoster have a protracted course, and many do not fully resolve. In comparison, 80% of patients with Bell palsy completely recover within 3 months.

Management and Disposition

Initial examination should include a thorough examination of the ear (including sensorineural or conductive hearing loss), the eye (including lacrimation), and the CNs—especially extraocular muscles. Motor function of the 7th CN is evaluated by having the patient raise their eyebrows, smile, pucker, and frown. No single laboratory test is diagnostic but may be required for excluding other disorders. Screening CT or MRI of the head is of little value in the absence of additional findings on physical examination.

Antivirals combined with corticosteroids are associated with higher rates of motor recovery. Eye lubricants and taping the eye shut at night help prevent keratitis and ulceration. Referral to a neurologist should be made for follow-up care.

Pearls

1. Facial nerve paralysis is a symptom, not a diagnosis.
2. If a provisional diagnosis of Bell palsy is made and no resolution of symptoms occurs, the diagnosis must be reconsidered. In patients misdiagnosed with Bell palsy, tumors are the most common missed etiology. MRI of the brainstem and internal auditory canal is indicated in these circumstances. Also

consider Lyme disease in areas of high risk.

3. The finding of CN VI (lateral rectus) palsy along with CN VII palsy is suggestive of a brainstem stroke, which involves the ipsilateral CN VII as it partially surrounds the CN VI nucleus. Hence, always evaluate for CN VI palsy when evaluating CN VII palsy.
4. Further ED evaluation is required for facial palsy presenting with sparing of the ipsilateral frontalis muscle as this represents a central lesion that may be caused by neoplasm or stroke.

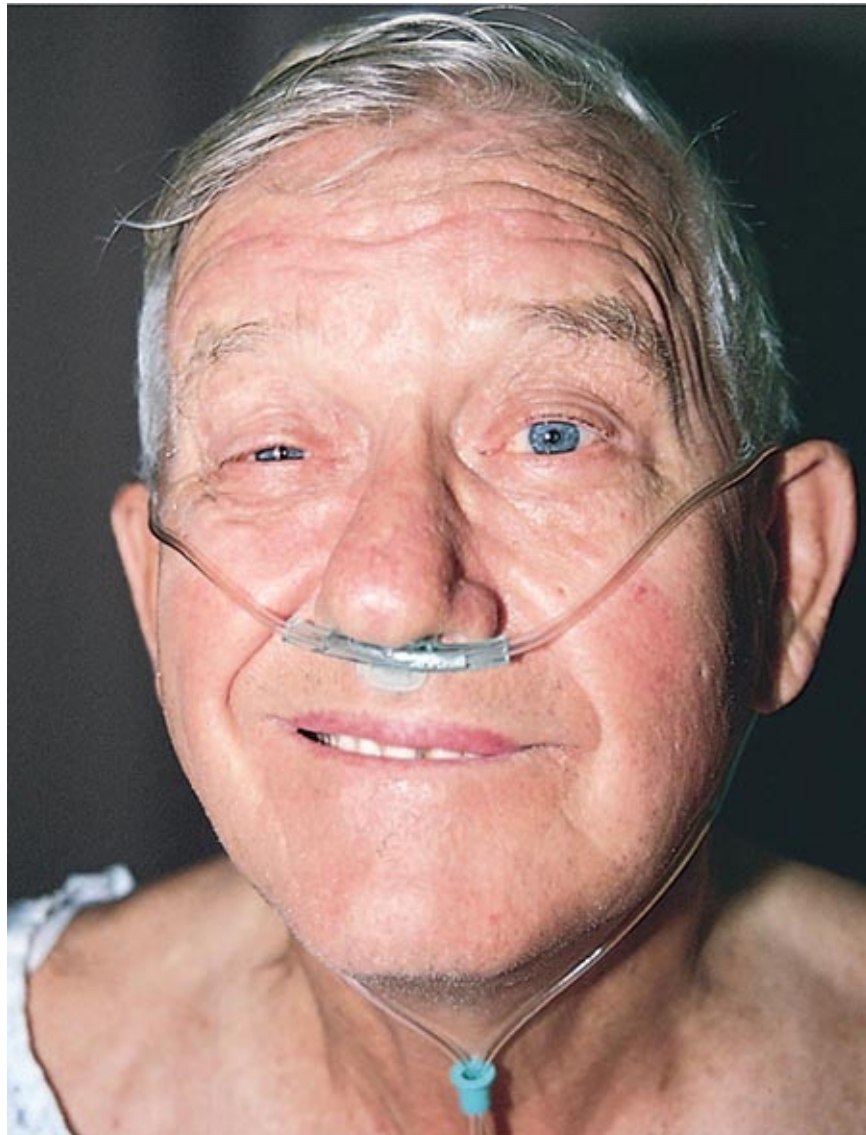


FIGURE 5.28 ■ Central Seventh-Nerve Palsy. Central facial nerve paralysis with forehead sparing. (Photo contributor: Frank Birinyi, MD.)



FIGURE 5.29 ■ Peripheral Seventh-Nerve Palsy. A peripheral nerve paralysis involving the entire ipsilateral face, including the forehead, is seen in this patient with Bell palsy. (Photo contributor: Lawrence B. Stack, MD.)

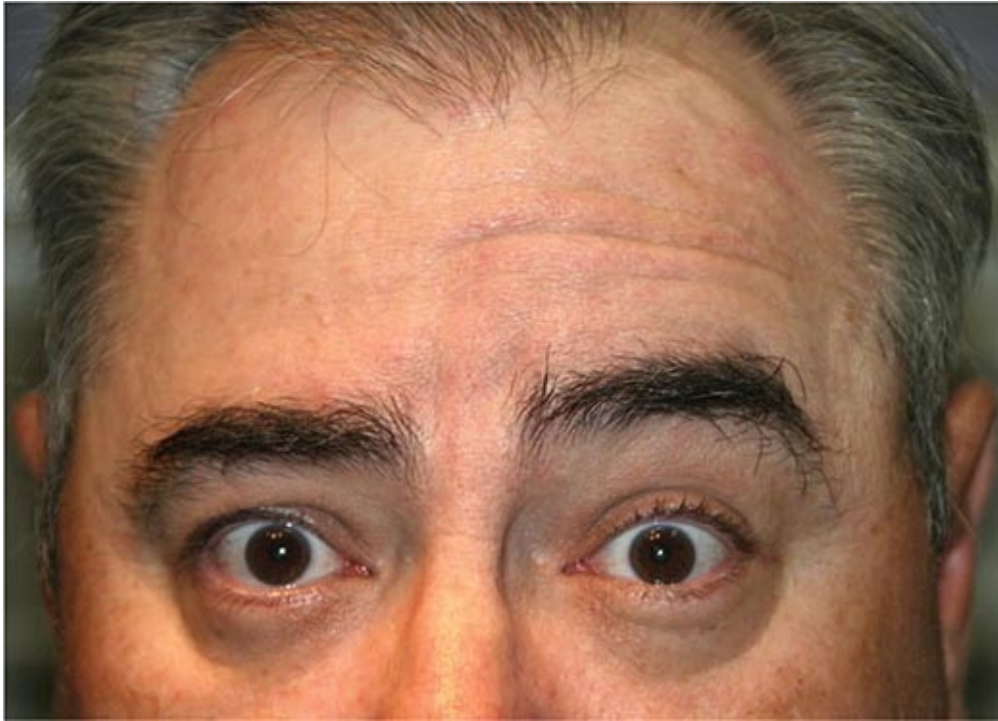


FIGURE 5.30 ■ Peripheral Seventh-Nerve Palsy. Forehead muscle paralysis is clearly apparent in this patient with Bell palsy. (Photo contributor: Suzanne Dooley-Hash, MD.)

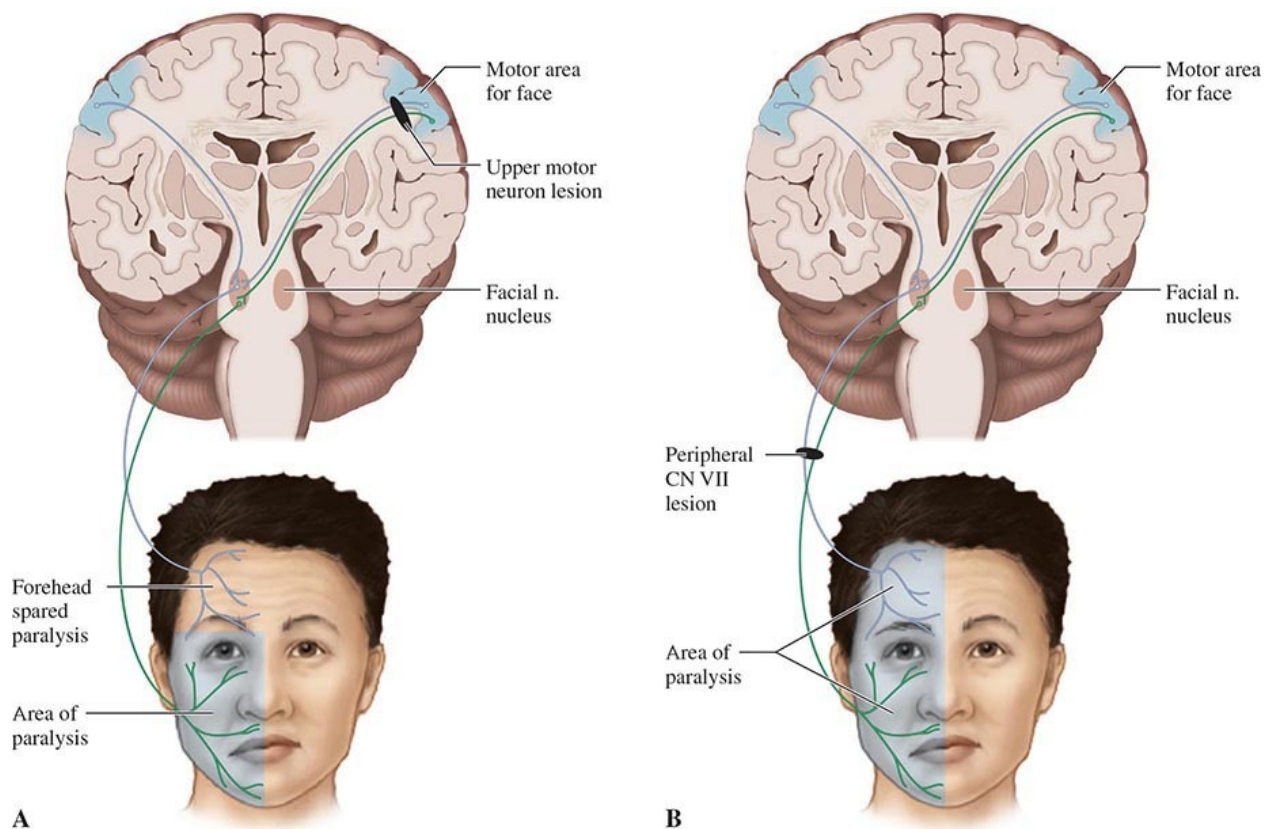


FIGURE 5.31 ■ Cranial Nerve VII Neuropathways. (A) Central CN VII lesion. Crossed fibers allow

forehead sparing in an upper motor neuron lesion identifying this presentation as a cerebrovascular accident versus a Bell palsy. (B) Peripheral CN VII nerve lesion causes paresis of both forehead and lower facial muscles.

ANGIOEDEMA

Clinical Summary

Angioedema is clinically characterized by acute onset of well-demarcated cutaneous swelling of the face, lips, and tongue; edema of the mucous membranes of the mouth, throat, or abdominal viscera; or nonpitting edema of the hands and feet (often asymmetric). It is hereditary, allergic, acquired, or idiopathic. Hereditary angioedema (HAE) is an autosomal dominant trait associated with a deficiency of serum inhibitor of the activated first component of complement (C1). Acquired angioedema has been described in patients with lymphoproliferative disorders and patients who possess autoantibodies against C1 esterase inhibitor. Often, the occurrence of angioedema is the first suggestion of an underlying illness. Allergic angioedema can result from medications or contrast agents, environmental antigens, or local trauma. Complications range from dysphagia and dysphonia to respiratory distress, airway obstruction, and death. Angiotensin-converting enzyme (ACE) inhibitor–induced angioedema has a predilection for involvement of the lips, face, tongue, and glottis, whereas alteplase-induced angioedema most commonly involves the tongue, and both, like HAE, are often refractory to medical therapy.

Management and Disposition

Airway protection remains the primary focus of emergency treatment. Frequent reassessment and early airway management are mandatory as deterioration due to edema formation can be rapid.

Medical therapy includes steroids, H₁ and H₂ histamine blockers, and subcutaneous or intramuscular epinephrine. Chronic angioedema responds better to corticosteroids and H₂ blockers. HAE may be treated with C1 esterase inhibitors (eg, Cinryze), a kallikrein inhibitor (eg, ecallantide), or a selective bradykinin B2 receptor antagonist (eg, icatibant). Further promising therapies for the treatment and prevention of HAE are in development.



FIGURE 5.32 ■ Angioedema. Severe angioedema of the face and tongue requiring emergent cricothyroidotomy. (Photo contributor: W. Brian Gibler, MD.)

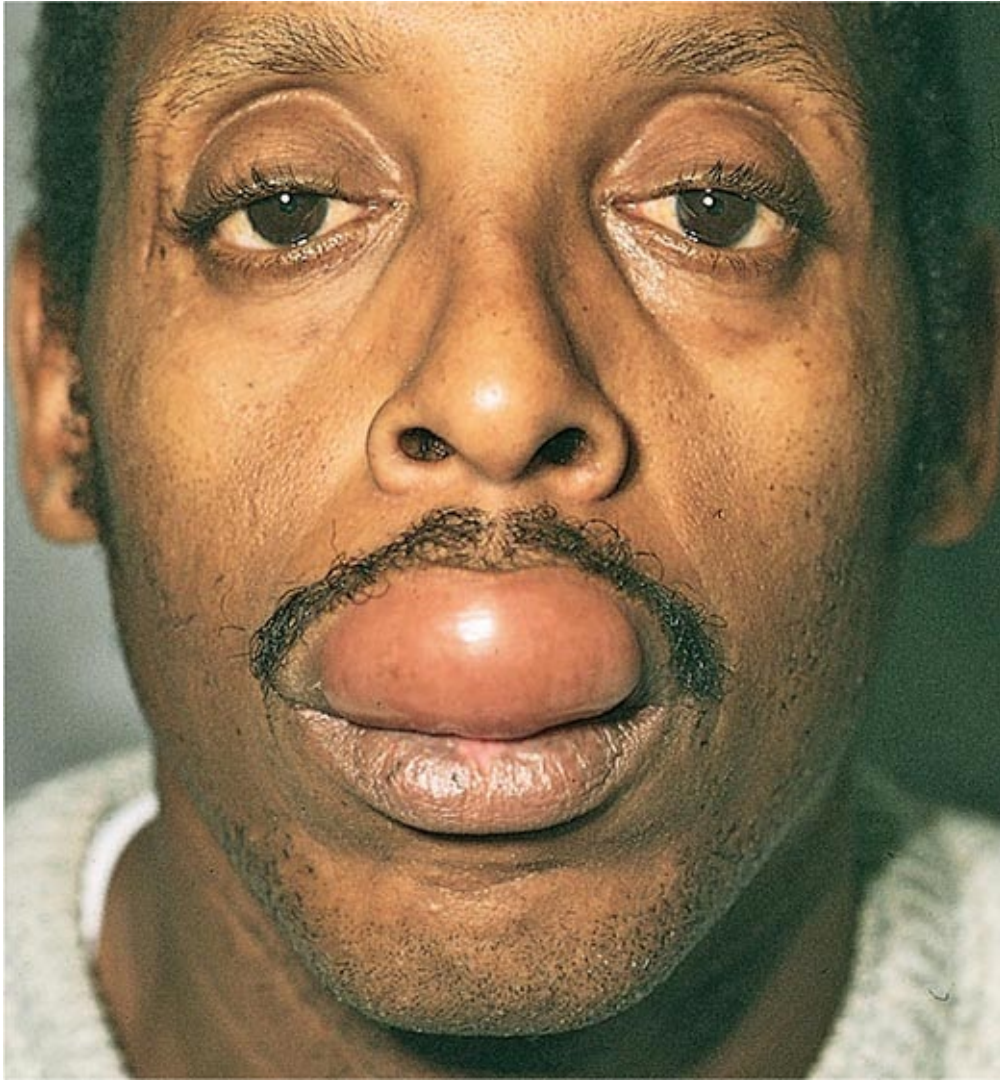


FIGURE 5.33 ■ ACE Inhibitor–Induced Angioedema. Angioedema of the upper lip in a man who had been taking an ACE inhibitor for 2 years. The patient had no previous episodes. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 5.34 ■ ACE Inhibitor–Induced Angioedema. Unilateral tongue swelling in a man who had been taking ACE inhibitors for 5 years. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.35 ■ Angioedema. Severe lip swelling in a patient with hereditary angioedema. (Photo contributor: Clay B. Smith, MD.)



FIGURE 5.36 ■ **Angioedema.** Severe lip swelling from an unknown cause. (Photo contributor: Clay B. Smith, MD.)



FIGURE 5.37 ■ Angioedema. Severe tongue swelling unresponsive to epinephrine in a patient with idiopathic hereditary angioedema. (Photo contributor: Kevin J. Knoop, MD, MS.)

Disposition depends on the severity and resolution of symptoms. Patients with symptomatic improvement or showing no worsening after several hours of observation may be discharged home. Discontinue suspect medications. Patients with any airway involvement require admission to a monitored environment with surgical airway equipment at the bedside.

Pearls

1. Do not underestimate the degree of airway involvement; act early to preserve airway patency.
2. Angioedema can also cause gastrointestinal and neurologic involvement.
3. Early response to medical intervention does not preclude rebound of

symptoms to a greater extent than at presentation.

4. Patients who have been using ACE inhibitors for months or years can still develop angioedema from these agents.

PHARYNGITIS

Clinical Summary

Pharyngitis is an inflammation and commonly an infection of the pharynx and its lymphoid tissues. Viral causes account for 90% of all cases. Group A β -hemolytic streptococci (GABHS) are responsible for up to 50% of bacterial infections. Other bacterial causes include other streptococci, *M pneumoniae*, *Neisseria gonorrhoeae*, and *Corynebacterium diphtheriae*. In immunocompromised patients and patients on antibiotics, *Candida* species can cause thrush.

Patients with bacterial pharyngitis present with an acute onset of sore throat and fever, frequently accompanied by nausea, vomiting, headache, and abdominal cramping. They may have an erythematous posterior pharynx and palatine tonsils, tender cervical lymphadenopathy, and palatal petechiae. Classically, the tonsils have a white or yellow exudate with debris in the crypts; however, many patients do not have exudate on examination. Viral pharyngitis is typically more benign, with a gradual onset, less fever, and less impressive erythema and swelling of the pharynx. Infectious mononucleosis can take weeks to resolve, whereas most cases of viral pharyngitis are self-limited, with spontaneous resolution in a matter of days. Lingual and adenoid tonsillitis may also be present.

Management and Disposition

Treatment is largely supportive except for antibiotics and rehydration. Analgesics, antipyretics, and throat sprays or gargles can provide symptomatic relief. Patients with known or suspected GABHS require antibiotics primarily to prevent rheumatic fever and suppurative complications. Centor criteria clinical decision rules developed to guide physicians in testing and prescribing of antibiotics include: (1) tonsillar exudates, (2) tender anterior cervical adenopathy, (3) fever by history, and (4) absence of cough. Neither antibiotic

treatment nor diagnostic testing is recommended with fewer than two criteria. Most authorities now favor evaluation using a sensitive rapid streptococcal antigen test (RSAT) for GABHS, without throat culture for negative results in adult patients with two or more Centor criteria. In children, it is recommended that all negative RSAT be followed up with a throat culture. Current first-line antibiotic therapies remain a single dose of intramuscular benzathine penicillin or oral penicillin for 10 days.



FIGURE 5.38 ■ Palatal Petechiae. Palatal petechiae and erythema of the tonsillar pillars in a patient with streptococcal pharyngitis. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 5.39 ■ Streptococcal Pharyngitis–Palatal Petechiae. Test-proven streptococcal pharyngitis in a patient with fever, sore throat, and cervical adenopathy. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.40 ■ Exudative Pharyngitis. Intense erythema with scant exudates is seen in this early (< 24 hours) case of GABHS pharyngitis. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 5.41 ■ Tonsillar Exudate. White and yellow cryptic exudates are seen in this patient with rapid strep test proven streptococcal pharyngitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.42 ■ Infectious Mononucleosis. Nearly touching exudative tonsils in a patient with elevated liver transaminases and hepatosplenomegaly. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.43 ■ Viral Pharyngitis. Intense erythema of the uvula and tonsillar pillars and ulcerations are typical of viral pharyngitis. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Other manifestations of pharyngitis should be sought in patients with sore throat. Sandpaper rash, Pastia lines, and desquamation are suggestive of scarlet fever. Hepatomegaly and/or splenomegaly should raise suspicion for infectious mononucleosis.
2. Sore throat or pharyngitis that lasts more than 2 weeks must be referred for further evaluation to rule out possible neoplastic or neurologic causes, especially in patients over 50 years old who have a smoking or chewing tobacco history.



FIGURE 5.44 ■ Inflammatory Pharyngitis. Inflammation and ulcerations of the uvula and soft palate seen in this adolescent patient with a Crohn disease flare-up. (Photo contributor: Lawrence B. Stack, MD.)

3. Amoxicillin should be avoided if infectious mononucleosis is a possibility, as a morbilliform eruption will occur in up to 80% of patients.
4. Pharyngitis itself may be a prodrome for other pathologic conditions, such as measles, scarlet fever, and influenza.



FIGURE 5.45 ■ Hemorrhagic Pharyngitis. Hemorrhagic tonsils, palatal petechiae, and pinch purpura of the buccal mucosa are seen in this patient with infectious mononucleosis–induced immunologic thrombocytopenic purpura. (Photo contributor: Sheila McMorrow-Jones, MD.)

NASAL SEPTAL CONDITIONS

Clinical Summary

Septal hematoma, with or without abscess formation, is an uncommon complication of septal surgery or direct nasal trauma. Bleeding from submucosal blood vessels leads to an accumulation of blood between the mucoperichondrium and the septal cartilage, which may lead to ischemic avascular necrosis of the underlying cartilage, destruction of the cartilage, and saddle nose deformity (see Fig. 1. 30). The hematoma and any necrotic cartilage may then serve as a nidus for infection, resulting in a septal abscess. Chronic cocaine use results in vasoconstriction and eventual necrosis of the septal cartilage, leading to perforation.

Septal injuries may lead to cosmetic nasal deformity, chronic sinus infections, recurrent epistaxis, and sleep disturbances. Rarely, it can result in more serious complications such as cavernous sinus thrombosis and meningitis. Since the

original trauma is often minor, patients may present days to weeks after the injury. Young children and infants may present with poor feeding, fever, and rhinorrhea. Older children and adults may note bleeding, headache, and more focal pain.

Nasal examination reveals a large, red, round swelling originating off the septum and occluding most of the nasal cavity. The mass is tender to palpation and may cause the outer aspects of the nose to be tender as well. Septal abscesses tend to be more painful and larger than uncomplicated hematomas. Fever is frequently present. *S aureus*, GABHS, *H influenzae*, and *S pneumoniae* are the organisms most commonly isolated in septal abscesses. A septal perforation is easily seen with pen-light examination.

Management and Disposition

Suspicion and recognition are essential in diagnosing septal injuries. Prompt referral to an otolaryngologist is mandatory for incision and drainage of the hematoma or abscess.



FIGURE 5.46 ■ Septal Hematoma. Fluctuant grapelike structure in the right naris after blunt facial trauma consistent with a septal hematoma. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Intranasal examination for septal hematoma in all patients with a history of

nasal trauma regardless of severity is crucial.

2. Antibiotics are required in septal hematomas with a clinical suspicion for a secondary infection or abscess.
3. The physician must explore the possibility of child abuse in young children and infants with a septal hematoma or abscess.



FIGURE 5.47 ■ Septal Abscess. A septal abscess is seen in both nares 1 week after blunt nasal trauma. (Photo contributor: Lawrence B. Stack, MD.)

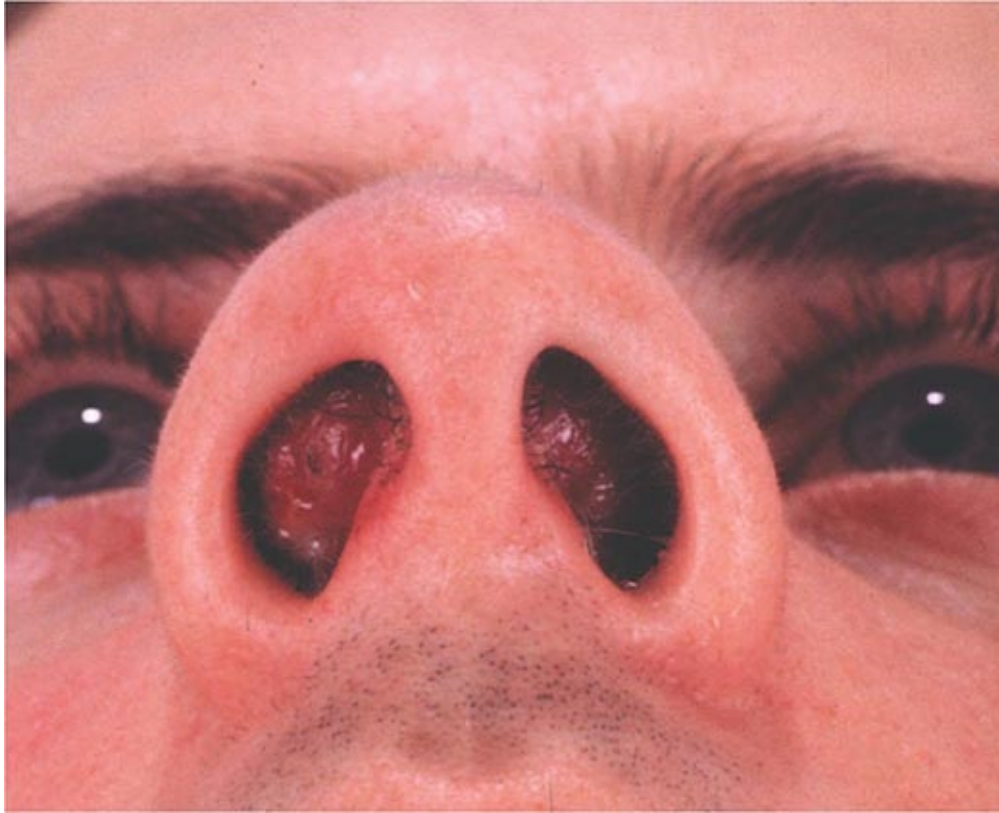


FIGURE 5.48 ■ Septal Abscess. A postoperative septal abscess is seen in both nares. (Photo contributor: Naval Medical Center Portsmouth).

NASAL CELLULITIS

Clinical Summary

Nasal cellulitis is an acute infection of the skin and subcutaneous tissues but does not involve the nasal cartilage. It is most common at the extremes of age. Bacterial invasion by *S pyo-genes* and *S aureus* due to disruption of the skin is the usual cause. Risk factors include nasal surgery, instrumentation, diabetes, immunodeficiency, and nasal piercing. Clinical features include pain, redness, and swelling of the nasal tissues. Headache, fever, and malaise suggest complicated disease. Complications of nasal cellulitis include abscess, cavernous sinus thrombosis, chondritis of the nasal cartilage, bacteremia, and sepsis.

Management and Disposition

The diagnosis of nasal cellulitis is clinical. However, evaluation may include complete blood count, blood and tissue cultures, contrasted CT if abscess is suspected, and CT venography if a cavernous sinus thrombosis is suspected. Remove any foreign body that might be a nidus of infection.

Amoxicillin-clavulanate or amoxicillin-sulbactam are first-line antibiotics. Clindamycin, vancomycin, trimethoprim-sulfamethoxazole, and 1st-generation cephalosporins are other options. Patients should be hospitalized if they have systemic symptoms, diabetes, a suspected abscess, or retained foreign bodies, or are immunocompromised. Patients at extremes of age should be strongly considered for admission.



FIGURE 5.49 ■ Nasal Cellulitis. Swelling and erythema of the nose in a middle-aged man. A contrasted CT was done to exclude abscess because of the marked swelling of the nasal septum. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.50 ■ Nasal Cellulitis. Swelling and erythema of the nose of a 2-year-old child, suggesting nasal cellulitis. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Threshold for hospital admission for patients with nasal cellulitis should be low.
2. In children, consider *H influenzae* and *S pneumoniae* as causative organisms of facial cellulitis.
3. Patients with pain at the nasal entrance but no overt physical findings of cellulitis may have vestibulitis, responsive to topical antibiotics (mupirocin).



FIGURE 5.51 ■ Nasal Cellulitis. Painful swelling and erythema of the nose suggestive of cellulitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

DIPHTHERIA

Clinical Summary

Diphtheria is a rare but highly contagious disease caused by the exotoxin-producing bacterium *C diphtheriae*. It is transmitted either by direct contact or through respiratory aerosolization. Many adults are now susceptible to diphtheria because their vaccine-induced immunity decreased over time or owing to decreased opportunity for naturally acquired immunity.

Diphtheria may involve any mucous membrane, but most commonly it affects the mucosa of the upper respiratory tract. It typically produces an ulcerated

pharyngeal mucosa with a white-to-gray inflammatory pseudomembrane, classically with a “wet mouse” odor. Patients present with symptoms, in order of frequency, of fever, sore throat, weakness, pain with swallowing, change in voice, loss of appetite, neck swelling, difficulty breathing, and nasal discharge.

While the organism remains localized to the mucosa, hematogenous spread of the exotoxin typically produces myocarditis or peripheral neuropathies. Case fatality rates from diphtheria range from 5% to 20% and are due to either tracheobronchial obstruction by the pseudomembrane acutely or cardiac complications several weeks after the primary infection.

Management and Disposition

The diagnosis is initially made clinically and confirmed by successful isolation and toxigenicity testing of *C diphtheriae*.

Antitoxin, available from the Centers for Disease Control and Prevention (CDC), is the mainstay of therapy and must be given even before laboratory confirmation. Erythromycin or penicillin given promptly when diphtheria is suspected has been shown to decrease both exotoxin production and spread of the bacterium.

Patients require hospital admission for observation of airway obstruction, pulmonary support, and intravenous hydration and antibiotics. Strict isolation is essential.

Pearls

1. Outcome is improved with early treatment; thus, the diagnosis of diphtheria must be made clinically and treatment begun empirically before bacteriologic confirmation.
2. Patients with a membranous pharyngitis need to be questioned regarding immunization, exposures, and recent travel.
3. All contacts should have a booster dose of vaccine (TD or Td, depending on age), while nonimmune contacts should also be given prophylactic antibiotics after a throat swab.



FIGURE 5.52 ■ Diphtheria Pharyngitis. An exudative pharyngitis with a gray pseudomembrane is seen in this patient with diphtheria. (Photo contributors: Dileep C. Unnikrishnan, MD, and Nadeem Kocheri, MD.)

PERITONSILLAR ABSCESS

Clinical Summary

Peritonsillar abscess (PTA), or quinsy, is the most common deep head and neck

infection. Although most occur in young adults, immunocompromised and diabetic patients are at increased risk. Most PTAs develop as a complication of tonsillitis or pharyngitis, but may also result from odontogenic spread, recent dental procedures, and local mucosal trauma.

The pathogens involved are similar to those causing tonsillitis, especially streptococcal species, but many infections are polymicrobial and involve anaerobic bacteria (*Fusobacterium*). Patients present with a fever, severe sore throat that is often out of proportion to physical findings, localization of symptoms to one side of the throat, trismus, drooling, dysphagia, dysphonia, fetid breath, and ipsilateral ear pain.

During the early stages, the tonsil and anterior pillar are erythematous, appear full, and may be shifted medially. Later, the uvula and soft palate are shifted to the contralateral side. The tonsillar pillar may feel fluctuant and tender.

Management and Disposition

In most patients, needle aspiration performed as the sole surgical drainage procedure produces a satisfactory outcome. Alternative surgical drainage procedures—including incision and drainage and abscess tonsillectomy—can be performed by an otolaryngologist or oral surgeon. Most PTAs are managed in the outpatient setting with oral antibiotics following drainage. Patients who are immunocompromised, have airway involvement, appear toxic, or cannot tolerate oral intake require admission for rehydration, parenteral antibiotics, and specialty consultation.



FIGURE 5.53 ■ Early Peritonsillar Abscess. Edema and marked erythema of the left tonsillar pillar in early peritonsillar abscess. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 5.54 ■ Peritonsillar Abscess. Acute peritonsillar abscess showing medial displacement of the uvula, palatine tonsil, and anterior pillar. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. The value of culturing aspirates is questionable, with a review of several studies showing no clinical benefit from the cultures unless the patient is

immunocompromised.

2. ED ultrasound along with palpation of the mass is a valuable adjunct in confirming diagnosis and verifying anatomical landmarks prior to aspiration.
3. A contrasted CT scan of the neck will confirm the presence of a PTA if the diagnosis is uncertain.



FIGURE 5.55 ■ Peritonsillar Abscess—Ultrasound. Ultrasound demonstrates size and location of abscess, in addition to confirming relationship to vascular structures. Over 2 mL of pus was aspirated from this abscess. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 5.56 ■ Peritonsillar Phlegmon. Marked erythema of the tonsillar pillars is seen in this patient currently on oral penicillin. No swelling or fluctuance is present. (Photo contributor: Lawrence B. Stack, MD.)

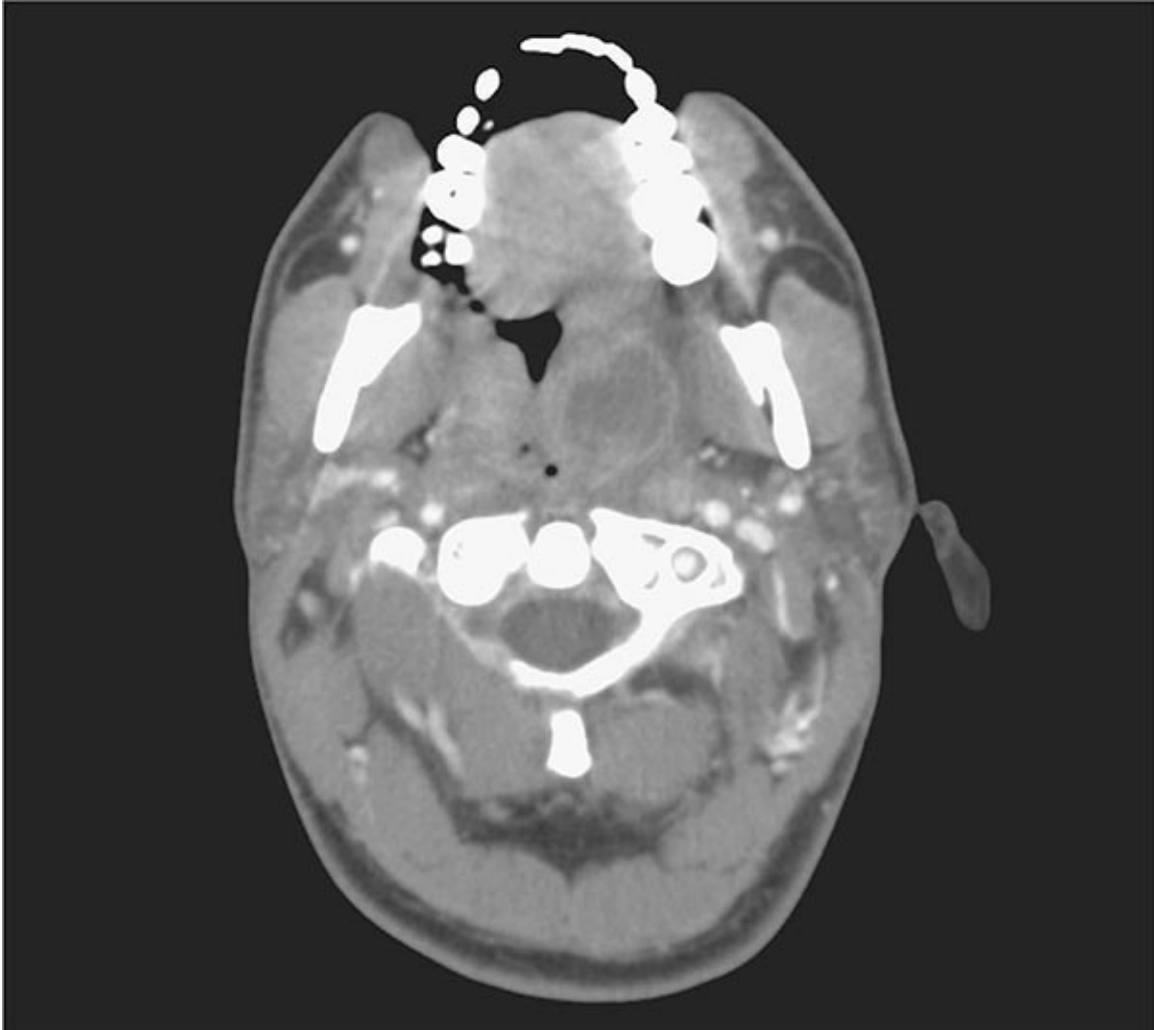


FIGURE 5.57 ■ Peritonsillar Abscess—CT. Ring-enhancing lesion with a hypodense core consistent with a left peritonsillar abscess. (Photo contributor: Lawrence B. Stack, MD.)

EPIGLOTTITIS

Clinical Summary

Epiglottitis or supraglottitis is an infection of the epiglottis and adjacent tissues. Bacterial epiglottitis, a rare but potentially fatal infection, is caused primarily by *H influenzae*, but *S pneumoniae*, *S aureus*, and β -hemolytic *Streptococcus* have also been isolated. The advent of the *H influenzae* B vaccination for infants has changed what used to be a disease primarily of children, with a peak age range from 2 to 6 years, to one occurring predominantly in adults. Bacterial epiglottitis

occurs most commonly in the winter and spring.

Patients, especially children, with acute epiglottitis appear quite ill. They present with sore throat, fever, drooling, severe dysphagia, dyspnea, muffled or hoarse voice, and occasionally inspiratory stridor. Patients with severe respiratory distress assume the “tripod” position: sitting upright with the neck extended, arms supporting the trunk, and the jaw thrust forward. This position maximizes airway patency and caliber. Adults typically have an indolent course with a prodromal viral illness, but many children have a sudden onset and rapid progression to respiratory distress.

Management and Disposition

Airway management is paramount. Even prior to diagnosis, children should be calmed, comforted by a parent, and allowed to assume whatever position they feel is most comfortable. Anesthesiology and ENT should be consulted immediately. Indications for intubation are clinical, but severe stridor and respiratory distress are clear reasons to intervene. Nasotracheal intubation over a flexible endoscope is preferred. Needle cricothyrotomy can provide temporary oxygenation until a surgical airway is provided.

Plain radiographs of the neck may reveal the classic “thumb” sign, a thickened epiglottis on the lateral soft-tissue neck radio-graph. Visualization of the epiglottis is possible in the stable adult patient via direct and indirect laryngoscopy and fiberoptic nasopharyngoscopy. In children, the top of the swollen epiglottis may be visualized on careful oral examination, whereas pharyngoscopy is typically reserved for an experienced anesthesiologist or otolaryngologist in a controlled setting.

The mainstay of epiglottitis treatment is antibiotic therapy. Third-generation parenteral cephalosporins, ampicillin with sulbactam or trimethoprim-sulfamethoxazole, have proven efficacy in treating epiglottitis. Steroids or epinephrine, either nebulized or subcutaneous, may provide some improvement in edema.

In addition to airway compromise, complications of epiglottitis include epiglottic abscess, meningitis, pulmonary edema, pneumonia, and empyema (associated with *H influenzae*).

Pearls

1. Transport of patients with suspected epiglottitis must be done by an

experienced transport team. The airway must be secured before transport of all but the most stable patients.

2. During intubation, pushing on the patient's chest may cause a bubble to form at the airway orifice, guiding placement of the tube.
3. In areas with a significant prevalence of infection with community-associated methicillin-resistant *S aureus* (MRSA), clindamycin should be considered as part of the empiric choice for gram-positive coverage.
4. Failure to intervene prior to loss of the airway carries a sixfold increase in mortality.

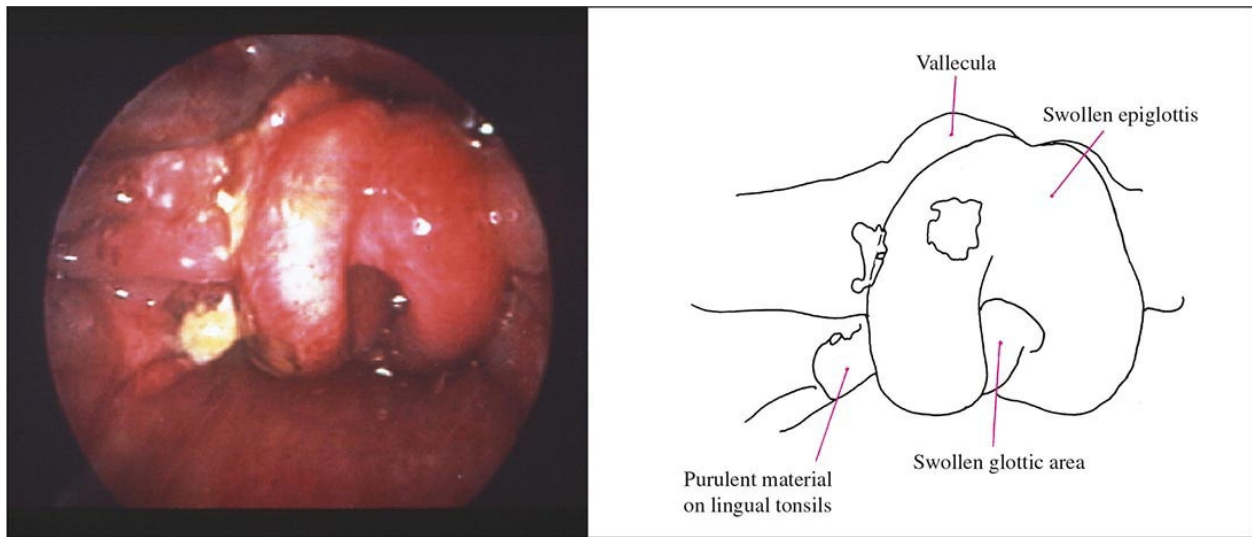


FIGURE 5.58 ■ Adult Epiglottitis. Fiberoptic laryngoscopy showing an edematous epiglottis and glottic area with marked airway compromise in an adult with epiglottitis. (Photo contributor: Timothy L. Smith, MD.)



FIGURE 5.59 ■ Adult Epiglottitis. Soft-tissue lateral neck radiograph of an adult with epiglottitis demonstrating the classic “thumb” sign of a swollen epiglottis. (Photo contributor: Kevin J. Knoop, MD, MS.)

UVULITIS

Clinical Summary

Idiopathic uvulitis is the most common cause of uvulitis, followed by infections and angioedema. Most patients complain of a sore throat, a gagging sensation, or a foreign-body sensation in the back of the mouth.

The infectious etiologies of uvulitis include bacterial, including *H influenzae* and streptococci; fungal, such as *C albicans*; and viral. Uvular infections are typically extensions from adjacent infections, such as epiglottitis, tonsillitis,

PTAs, and pharyngitis. Patients note fever, odynophagia, trismus, facial pain, hoarseness, neck pain, and headache. On examination, the uvula is red, firm, swollen, and very tender to palpation.

Angioedema of the uvula, known as the Quincke disease, can be hereditary, acquired, or idiopathic. Medications, allergens, thermal stimuli, pressure, and iatrogenic or accidental trauma can initiate angioedema. In addition to the swollen uvula, patients may note pruritus, urticaria, and wheezing. With uvular edema, the angioedema may also involve the face, tongue, and oropharynx. Airway compromise is more common in angioedema of the uvula, which appears pale, boggy, and edematous, resembling a large white grape (uvular hydrops).

Management and Disposition

Most cases of uvulitis are benign and self-limited. Angioedematous uvulitis is treated with steroids, antihistamines, and epinephrine in severe cases, either subcutaneously or nebulized. For infectious uvulitis, antibiotic coverage is dictated by the primary source of infection. Admission is based on severity of airway compromise and accompanying infections.



FIGURE 5.60 ■ Uvulitis. Isolated edema of the uvula in a patient who presents with a foreign-body sensation of the throat. (Photo contributor: R. Jason Thurman, MD.)

Pearl

1. Any airway symptom prompts an evaluation of the hypopharynx, by either radiographic imaging, fiberoptic nasopharyngoscopy, or direct laryngoscopy.

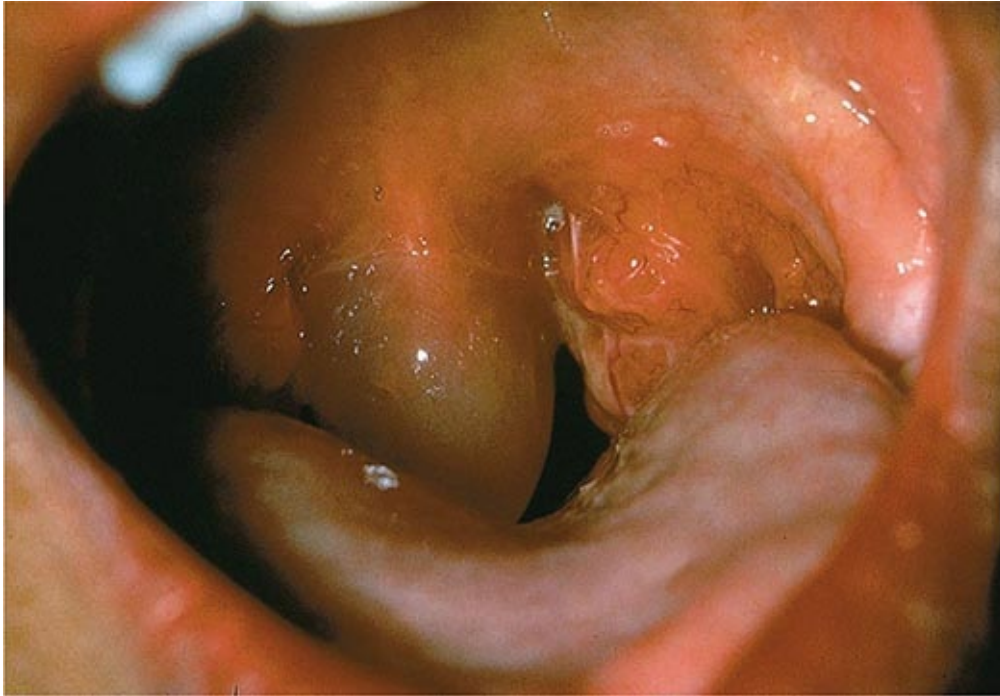


FIGURE 5.61 ■ Uvular Hydrops. Angioedema of the uvula, known as the Quincke disease. A pale, boggy, and edematous, uvula resembling a large white grape is seen. (Photo contributor: Robin T. Cotton, MD.)



FIGURE 5.62 ■ Uvulitis. Isolated edema of the uvula showing a globular structure that presents with foreign-body sensation. (Photo contributor: Lawrence B. Stack, MD.)

SIALOADENITIS

Clinical Summary

Sialoadenitis is a general term describing inflammation of any salivary gland. The three major salivary gland pairs are the parotid, submandibular, and sublingual. There are also numerous smaller salivary glands that empty into the oral cavity and all are capable of becoming inflamed. Salivary gland disorders have numerous causes, including acute and chronic infections; metabolic, systemic, and endocrine disorders; infiltrative processes; obstructions; allergic inflammation; and neoplastic diseases. Key features in the history are the duration and course of the symptoms, complaints of pain, and unilateral or bilateral location.

Both viral and bacterial infections of the salivary gland can produce enlarged, swollen, painful masses. Suppurative sialoadenitis is most commonly caused by *S aureus* and occurs in patients who are elderly, diabetic, or have poor oral hygiene. It may also follow episodes of dehydration, such as those due to surgery or debilitation. Viral sialoadenitis, such as mumps, the most common cause of nonsuppurative parotitis, or HIV, is the most common cause of sialoadenitis. It occurs with a concomitant general viral illness and is usually bilateral, whereas bacterial infections are primarily unilateral.



FIGURE 5.63 ■ Suppurative Parotid Sialadenitis. Fever, swelling, and tenderness over the parotid gland along with purulent discharge expressed from Stensen duct suggest suppurative parotid sialadenitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.64 ■ Suppurative Parotid Sialoadenitis. Pus from Stensen duct confirms suppurative parotid sialoadenitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.65 ■ Suppurative Submandibular Sialoadenitis. Unilateral submandibular swelling. (Photo contributor: Jeffery D. Bondesson, MD.)



FIGURE 5.66 ■ Sialolithiasis. A stone is seen at the orifice of Wharton duct. Small ranula are also seen at the tongue base. (Photo contributor: David Effron, MD.)

Obstructive sialoadenitis occurs from a calculus in the salivary gland or duct, most commonly in the submandibular gland. The flow of saliva becomes obstructed, causing swelling, pain, and firmness. Patients with sialolithiasis note general xerostomia and recurrent worsening of swelling and pain during eating.

A thorough head and neck examination is essential, especially a bimanual examination of the major salivary glands. In suppurative sialoadenitis, purulent drainage may be expressed from the submandibular duct (Wharton) or parotid duct (Stensen), and the glands are very tender and painful to examination. Sialolithiasis can manifest as enlargement of the ducts with minimal saliva expressed on stripping and, rarely, a palpable or visible stone or duct thickening. Facial radiographs are of limited utility. Ultrasound or CT may be useful to detect abscesses.



FIGURE 5.67 ■ Suppurative Submandibular Sialadenitis. After applying firm pressure, purulent discharge is seen coming from Wharton duct. (Photo contributor: Jeffery D. Bondesson, MD.)



FIGURE 5.68 ■ Submandibular Sialolithiasis. Unilateral submandibular swelling. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Treatment of suppurative sialoadenitis requires antibiotics with coverage of *Staphylococcus* and oral flora, rehydration, proper oral hygiene, sialogogues, local heat, and occasionally surgical irrigation and drainage of abscesses. Obstructive sialoadenitis is rarely an emergency. Most salivary stones pass spontaneously without complication, and patients can be discharged home on lozenges to stimulate salivary secretions. Prompt follow-up of sialoadenitis is essential to prevent possible morbidity and mortality associated with infections or neoplasms.

Pearls

1. Examine secretions of both mouth and eyes, and elicit any history of dry eyes, keratoconjunctivitis, cutaneous lesions, or rheumatoid arthritis to establish the diagnosis of a systemic disorder.
2. Medications such as antihistamines, psychotropic drugs, and those possessing atropine-like side effects can cause xerostomia, exacerbating or predisposing to sialoadenitis.
3. Lack of improvement on antibiotics suggests an abscess or multiple loculated

abscesses that require drainage.



FIGURE 5.69 ■ Submandibular Sialolithiasis. A stone is seen at the orifice of Wharton duct. (Photo contributor: Kevin J. Knoop, MD, MS.)

MUCOCELE

Clinical Summary

Mucoceleles are mucous cysts occurring on the mouth or lip. Ranulas are mucoceleles (mucous retention cysts) that develop in the floor of the mouth, arising from obstructed sublingual or submandibular ducts or smaller minor salivary glands. Initially, the cysts are small and barely noticeable, but over time, they can expand outward or deeper into the neck (plunging ranula). Large cysts can displace the tongue forward and upward, making the patient uncomfortable. Unlike those with sialolithiasis, patients with ranulas may not always notice an

increase in swelling associated with eating. Mucoceles on the lip typically affect the lower lip. Physical examination reveals a soft, minimally tender, translucent cyst with dilated veins running over its surface. Unlike carcinomas, no ulceration is noted with ranulas, and they are generally softer.

Management and Disposition

Recognition by the physician is essential for proper referral. While most mucoceles resolve spontaneously, definitive treatment for recurrent or chronic mucoceles is excision or marsupialization, although needle aspiration of the cyst can provide temporary relief. Unless there is a secondary infection, no antibiotic coverage is required.



FIGURE 5.70 ■ Ranula. Sublingual ranula, or mucocele, lateral to Wharton duct. The patient was asymptomatic except for being aware of the lesion. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Most mucocèles are painless and are incidental findings on routine examinations.
2. Mucocèles often recur, requiring total excision of the offending salivary gland.



FIGURE 5.71 ■ Mucocèle. Focal swelling of the upper lip that developed months after local trauma consistent with a mucocèle. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.72 ■ Mucocele. Mucocele in an adult who suffered mild lip trauma 3 weeks prior. (Photo contributor: Lawrence B. Stack, MD.)

ACUTE SINUSITIS

Clinical Summary

Acute rhinosinusitis results from an impairment of mucociliary clearance and subsequent inflammation of the paranasal sinuses, due to infection, allergies, or mechanical obstruction. Sinusitis typically occurs in conjunction with inflammation of the nasal mucosa. The most common etiology is a viral infection; most bacterial cases are associated with antecedent viral upper respiratory tract infection.

Maxillary sinusitis (most common) is associated with para-nasal facial, retroocular, or maxillary dental pain, purulent rhinorrhea, and conjunctivitis. Ethmoid sinusitis, more common in children, produces a low-grade fever and periorbital pain. Frontal sinusitis can cause a severe supraorbital headache, which is exacerbated by leaning forward; a low-grade fever; upper lid edema; and rhinorrhea. Sphenoid sinusitis is rare, and patients typically complain of a vertex headache and retroocular pain. Sphenoid sinusitis can involve cranial

nerves, most commonly the abducens nerve, the pituitary gland, and the cavernous sinus. Involvement of all sinus cavities is referred to as pansinusitis. Important complications of sinusitis include periorbital and orbital cellulitis, cavernous sinus thrombosis, and intracranial abscess.

Pott's puffy tumor, a rare osteomyelitis of the cranium from direct extension of a frontal sinusitis, presents as a boggy, tender swelling overlying the frontal sinus.

H influenzae and *S pneumoniae* together represent 60% to 70% of bacterial causes. Immunocompromised patients are susceptible to fungal infections, including *Aspergillus* and *Mucor* species.

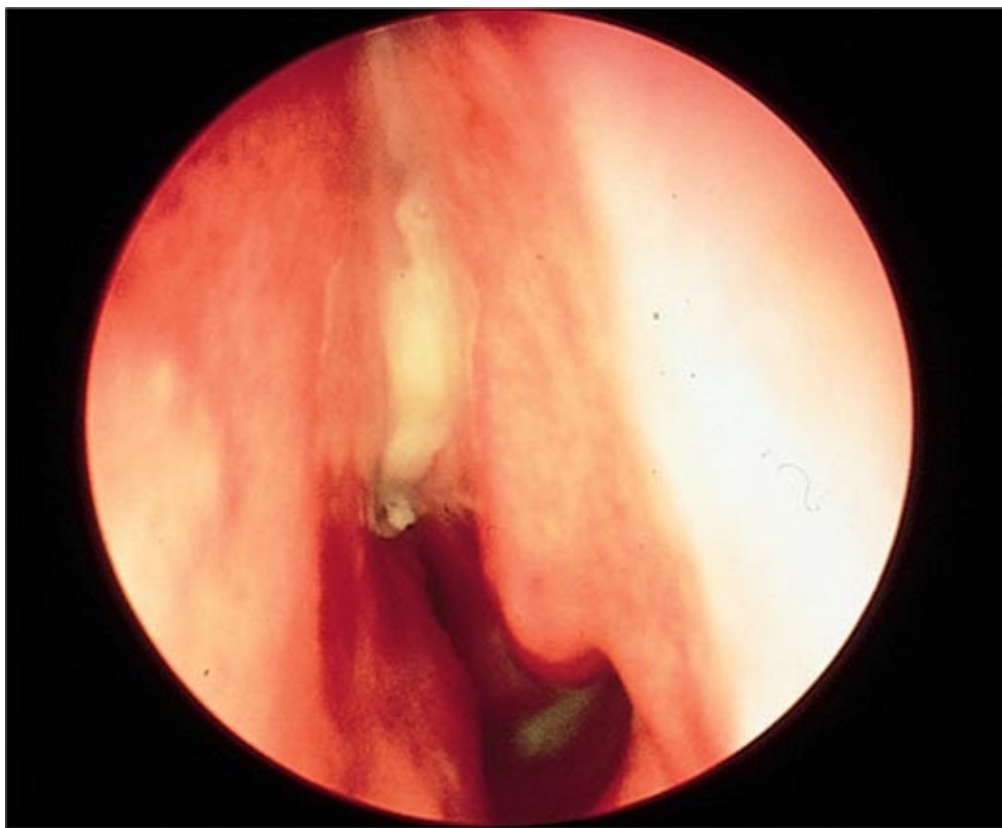


FIGURE 5.73 ■ Sinusitis. Purulent drainage from the maxillary sinus ostium in a patient with maxillary sinusitis. Drainage may not always be apparent, since the ostium may be occluded from swelling and inflammation. (Photo contributor: Robin T. Cotton, MD.)

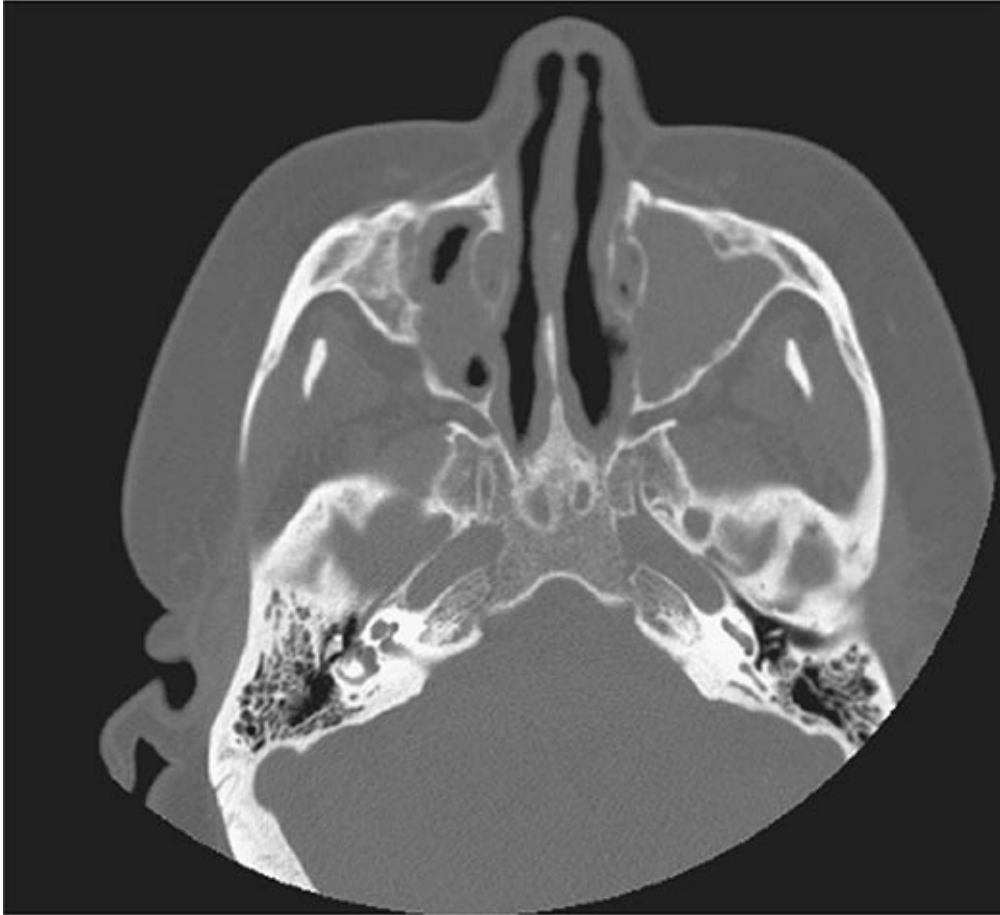


FIGURE 5.74 ■ Sinusitis. Bone windows of a sinus CT demonstrating bilateral maxillary sinus disease. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.75 ■ Pansinusitis—Pott's Puffy Tumor. Forehead and right periorbital edema in a patient with pansinusitis suggests osteomyelitis of the anterior table of the frontal sinus. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Most patients can be treated with oral or intranasal decongestants and analgesics alone. Humidified air, steam, or saline nasal sprays also facilitate drainage. If symptoms persist beyond 7 days, consider antibiotics.



FIGURE 5.76 ■ Sinusitis—Pott's Puffy Tumor. Marked swelling and erythema over the central forehead in a patient with sinusitis indicating osteomyelitis of the anterior table of the frontal sinus. (Photo contributor: Lawrence B. Stack, MD.)

Parenteral steroids are not used in acute or recurrent sinusitis. Intranasal steroids may have a role in allergic and chronic sinusitis. CT is the most sensitive and specific imaging modality.

Otolaryngologist or primary care follow-up should be made for routine cases within 3 weeks. Patients with comorbid illnesses or more complicated sinusitis should be admitted for parenteral antibiotic therapy and supportive care.

Pearls

1. Chronic sinusitis may be due to mucoid retention cysts, deviated septum, or nasal polyps. Refer for possible surgery.
2. Physicians must consider fungal etiologies in immunocompromised patients or those with comorbid illnesses.

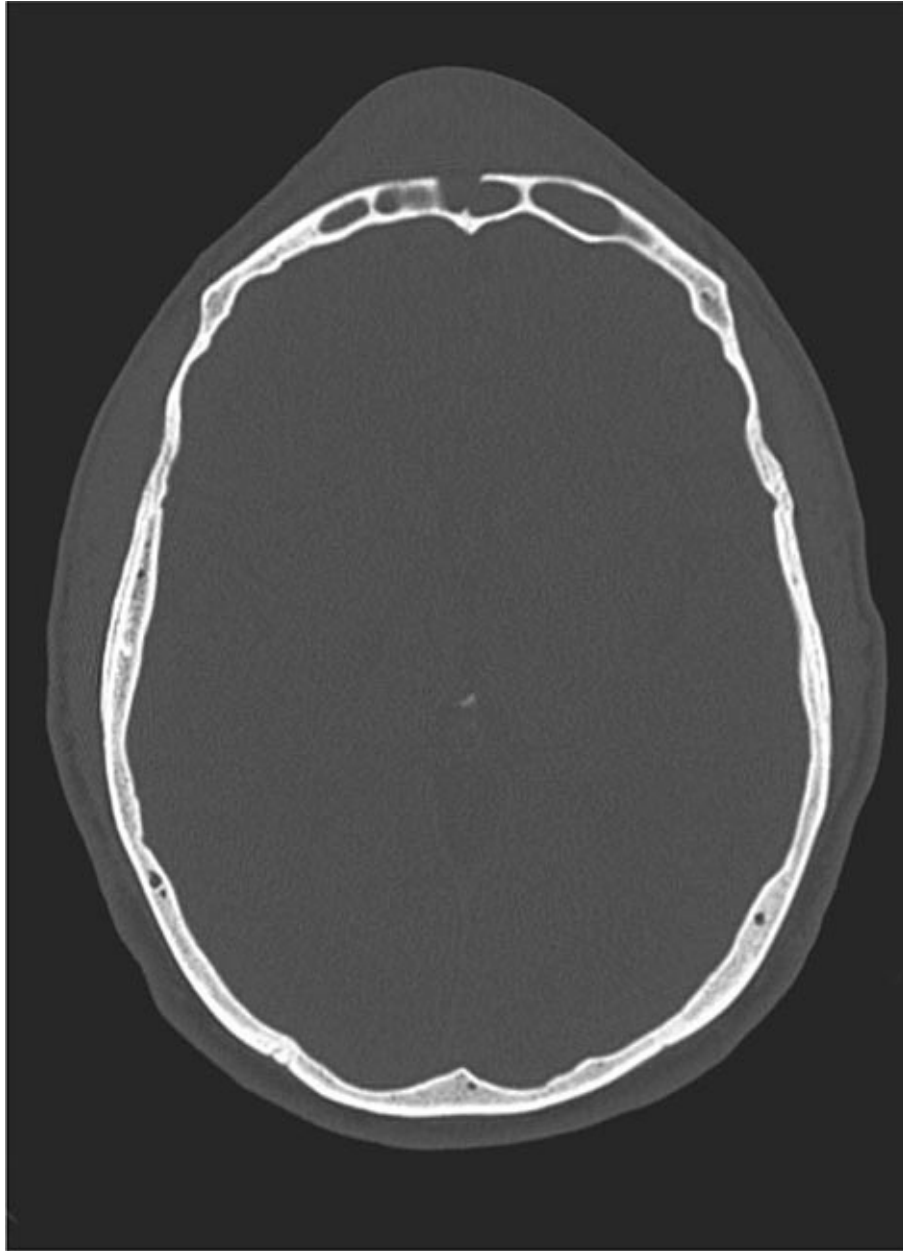


FIGURE 5.77 ■ Sinusitis—Pott's Puffy Tumor. CT (bone window) highlighting bony destruction of inner and outer table of frontal bone from sinusitis. (Photo contributor: Lawrence B. Stack, MD.)

The authors acknowledge David R. White, Sean Barbabella, and Francisco Fernandez for portions of this chapter written for the previous editions of this book.

Chapter 6

ORAL CONDITIONS

**Edward C. Jauch
J. Amadeo Valdez**



Herpes Labialis. Note the extensive painful ulcerations on the patient's upper lip and corners of the mouth. A prodromal period of fever, malaise, and cervical adenopathy may herald the onset of these painful ulcerations. (Photo contributor: R. Jason Thurman, MD.)

TOOTH SUBLUXATION

Clinical Summary

Tooth subluxation, the loosening of a tooth in its alveolar socket, is most commonly secondary to trauma; however, infection and periodontal disease may also produce subluxation. Gingival lacerations and alveolar fractures are associated with dental subluxations. Gentle pressure to the teeth with a tongue blade or fingertip may produce movement, mild displacement, or blood along the crevice of the gingiva, all signs of subluxation. Dental impaction and alveolar ridge fracture should be considered and ruled out clinically or radiographically.

Management and Disposition

1. *Primary teeth:* If the subluxated tooth is forced into close proximity to the underlying permanent tooth, follow-up for extraction is indicated. Otherwise, the patient should be instructed to follow a soft diet for 1 to 2 weeks, allowing the tooth to reimplant spontaneously.



FIGURE 6.1 ■ Tooth Subluxation. Note the presence of blood along the crevice of the gingival margin of both central incisors—an indication of subluxation following trauma. Mild displacement of the subluxated teeth is noted. (Photo contributor: James F. Steiner, DDS.)

2. *Permanent teeth:* Unstable teeth should be temporarily immobilized using gauze packing, a figure-eight suture around the tooth and an adjacent tooth, aluminum foil, or a special periodontal dressing, and the patient referred for dental follow-up.

Good oral hygiene should be maintained by using chlorhexidine 0.12% topically twice a day for a week.

Pearls

1. Any evidence of tooth mobility following trauma is a subluxation by definition.
2. Always consider an associated underlying alveolar or occult root fracture.



FIGURE 6.2 ■ Tooth Subluxation. Tooth subluxations can be quite subtle, as in this case of a slightly subluxed tooth 9 missed on initial examination. A careful dental examination is essential in patients with oral trauma. (Photo contributor: Kevin J. Knoop, MD, MS.)

TOOTH IMPACTION (INTRUSIVE LUXATION)

Clinical Summary

Impacted or intruded teeth result when a tooth is forced deeper into the alveolar socket or surrounding tissues as a result of trauma. The tooth appears shorter than its contralateral mirror. An impacted tooth may be partially visible or completely hidden by the gingiva and buried in the alveolar process. Completely impacted teeth may erroneously be considered avulsed until a radiograph demonstrates the intruded position. The apex of a completely impacted permanent central incisor may be driven through the alveolar bone into the floor of the nares, causing epistaxis. Associated injuries may include alveolar fractures, dental crown or root fractures, and oral mucosal or gingival lacerations. Pulp necrosis occurs in 15% to 50% of cases.

Management and Disposition

Impacted primary teeth usually re-erupt and reposition spontaneously within 1 to 3 months. Any intruded primary tooth whose apex is displaced toward or impacts on the follicle of its permanent successor requires dental follow-up for extraction and monitoring clinically and radiographically for 1 year. Permanent teeth do not re-erupt. Surgical reduction is indicated to prevent complications such as external root resorption and loss of supporting bone. Orthodontic repositioning and splinting are generally carried out over 3 to 4 weeks. Good oral hygiene should be maintained by using chlorhexidine 0.12% topically twice a day for a week.

Pearls

1. An undiagnosed impacted tooth is predisposed to infection and may have a poor cosmetic result.
2. The maxillary incisors are the most commonly impacted teeth.



FIGURE 6.3 ■ Tooth Intrusion. This impaction injury with multiple anterior maxillary tooth involvement shows various degrees of tooth impaction. Also note the complete absence of a central incisor. This may indicate a complete intrusion into the alveolar socket or an avulsion of the tooth. Radiographic studies are required when a tooth's location is in question. (Photo contributor: James F. Steiner, DDS.)

TOOTH AVULSION

Clinical Summary

Avulsion is the total displacement of a tooth from its socket. There is usually a history of trauma; however, infectious etiologies may result in complete disruption of the periodontal ligament from the affected tooth. Various degrees of bleeding from the socket and surrounding gingiva may be noted, and there may be underlying alveolar fracture depending on the mechanism of injury. Prompt inquiry into the location of any unaccountable tooth is indicated. Radiographic evaluation to rule out aspiration, soft tissue entrapment, impaction, or dentoalveolar fracture is required when teeth are missing.

Management and Disposition

Successful reimplantation decreases by approximately 1% for every minute the tooth is out of its socket. Permanent teeth should be replaced in their sockets as soon as possible. Successful reimplantation depends on the survival of periodontal ligament fibers; the tooth should be rinsed with saline, but not scrubbed, with care not to handle the root while replacing it in the socket. Emergent dental consultation, tetanus prophylaxis, and antibiotics targeting mouth flora are indicated. If not replaced, the avulsed tooth should be stored in the mouth of the patient or parent, or in a container of milk. Normal saline and commercial preservatives are reasonable alternatives, but tap water should not be used. Prior to reimplantation, rinse alveolar socket with saline to remove any clot. Primary teeth are not reimplanted; reimplanted teeth may interfere with eruptions of permanent teeth because of ankylosis and fusion to the bone. Patients should use antibiotics for oral microbiota and maintain good oral hygiene using chlorhexidine 0.12% topically twice a day for a week.

Follow-up should be obtained for possible orthodontia until the permanent tooth erupts.

Pearls

1. Primary teeth should not be reimplanted.
2. Successful reimplantation occurs best within the 1st 30 minutes.
3. Storage and transport media in decreasing order for preserving tooth viability include balanced salt solution or a tissue culture medium, chilled low-fat milk, saline, and saliva.

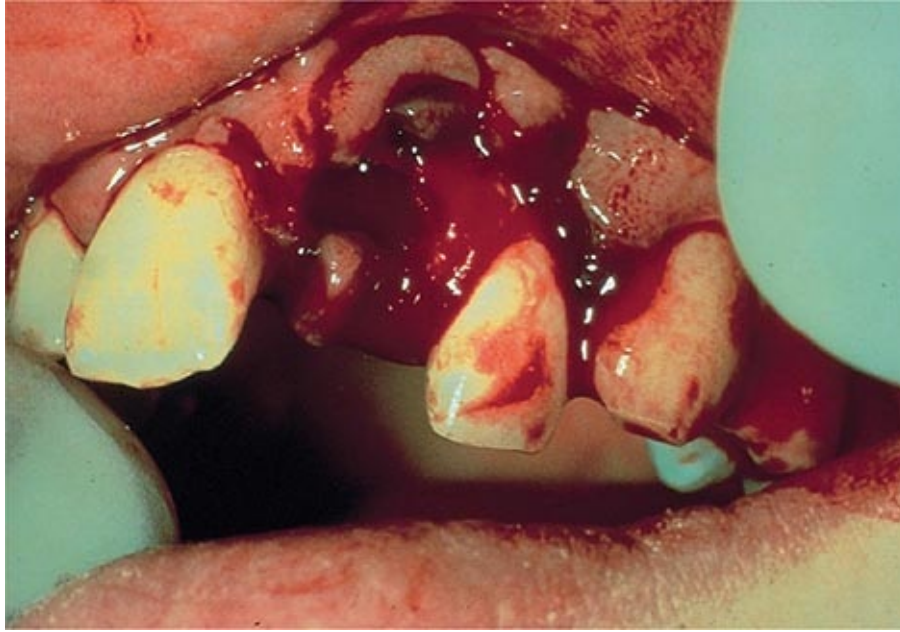


FIGURE 6.4 ■ Tooth Avulsion. Avulsion injury with angulation and displacement of teeth from the alveolar socket. (Photo contributor: James F. Steiner, DDS.)

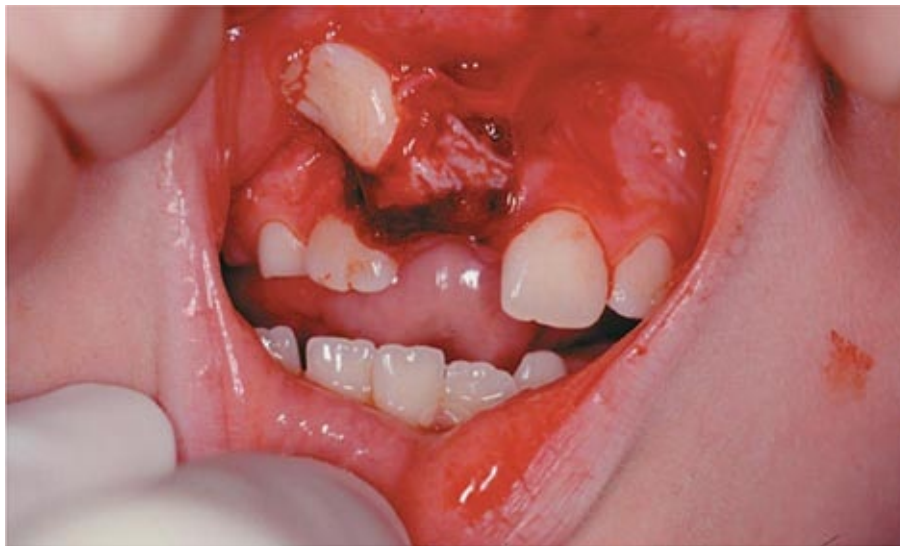


FIGURE 6.5 ■ Tooth Avulsion. Significant avulsion injury of tooth #8 in a patient with direct oral trauma. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 6.6 ■ Tooth Avulsion Reimplanted. Reimplantation of the avulsed tooth in [Fig. 6.5](#) was performed by an oral surgeon with the use of an arch bar to secure the tooth. (Photo contributor: Lawrence B. Stack, MD.)

TOOTH FRACTURES

Clinical Summary

Anatomically, each tooth has a crown and root portion. Externally, the crown is covered with white enamel and the root portion with cementum. The cemento-enamel junction (cervical line) is where the crown and root meet. The yellow-to-tan dentin is the 2nd innermost layer and comprises the bulk of the tooth. The red-to-pink pulp tissue is located in the center of the tooth and includes the tooth's neurovascular supply. The Ellis classification system is commonly used to describe tooth fractures above the cervical line in anterior teeth:

Ellis class I: Involves only the enamel.

Ellis class II: Involves the enamel plus exposure of the dentin.

The patient may complain of temperature sensitivity.

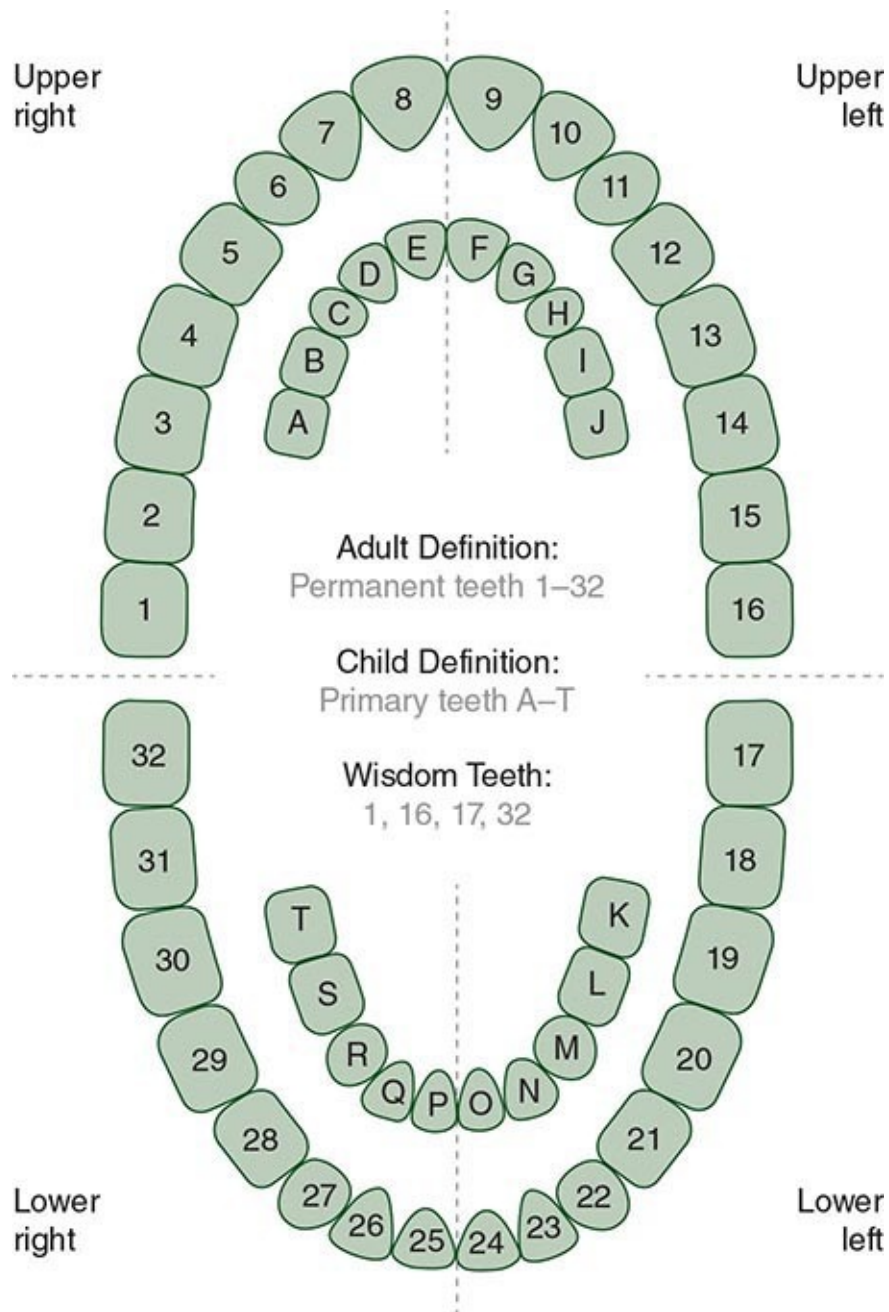


FIGURE 6.7 ■ Tooth Numbering System. The standard tooth numbering system should be used to descriptively identify which tooth or teeth are fractured or otherwise pathologically affected. (Source: Hay WW, Levin MJ, Detering RR, Abzug MJ. *Current Diagnosis & Treatment: Pediatrics*. 24th ed. New York, NY: McGraw Hill; 2018. Copyright McGraw Hill Education.)

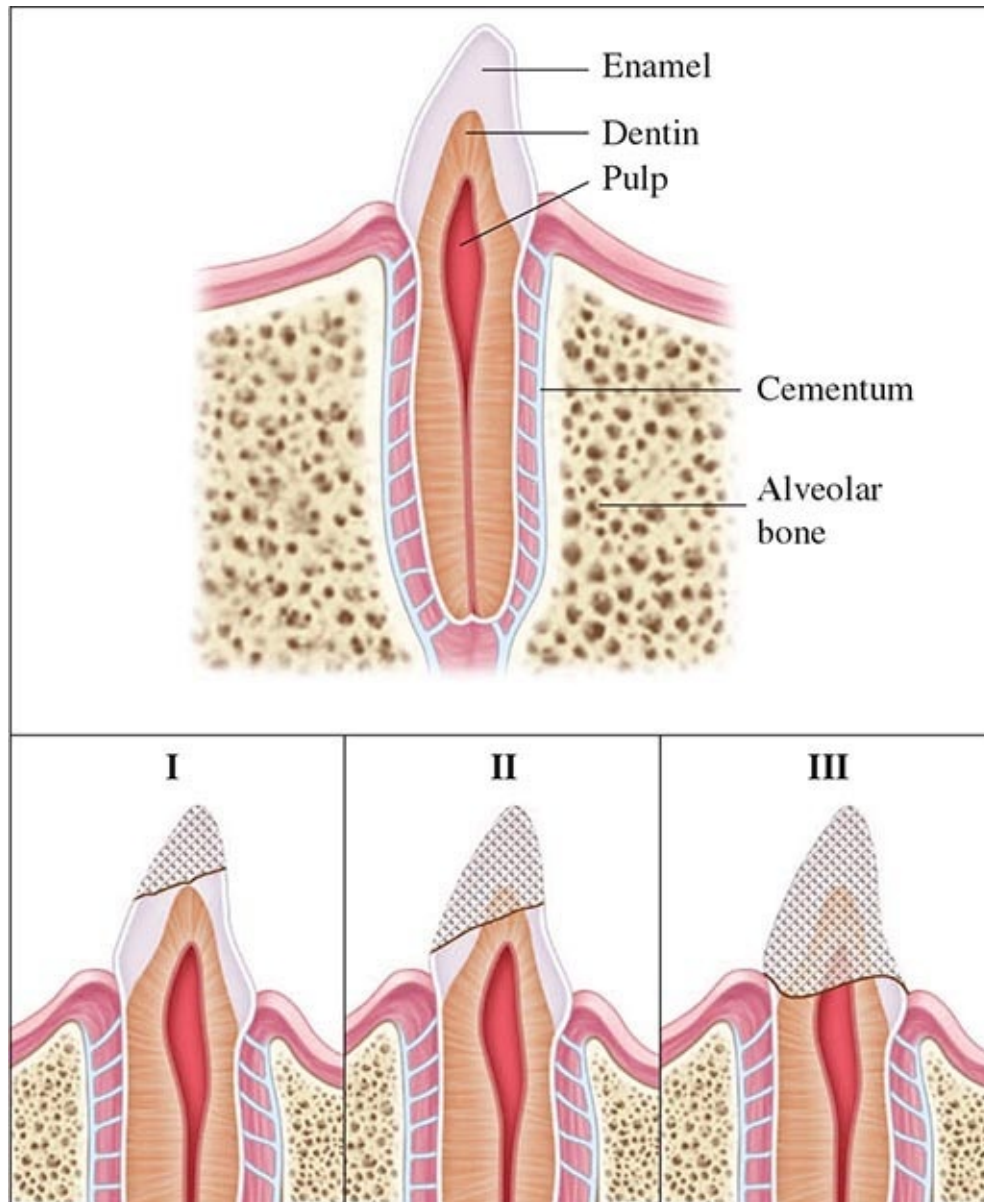


FIGURE 6.8 ■ Tooth Fractures. Enamel, dentin, and pulp are the anatomic landmarks used in the Ellis classification of tooth fractures.



FIGURE 6.9 ■ Ellis Class I Tooth Fracture. Note the fracture of the left upper central incisor. The sole involvement of the enamel is consistent with an Ellis class I fracture. (Photo contributor: James F. Steiner, DDS.)

Ellis class III: Fracture extends into the pulp. A pink or bloody discoloration on the fracture surface is diagnostic. The patient may have severe pain but may also have no pain due to loss of tooth nerve function.

Tooth fractures of the dental root may also occur below the cementoenamel junction and are commonly missed on initial evaluation. Bleeding may be observed at the gingival crevice with associated tooth tenderness on percussion. Radiographic evaluation may aid in differentiating these conditions.

Management and Disposition

Ellis class I: Pain control and referral for rough tooth edge and cosmetic management are indicated.

Ellis class II: Patients under 12 years of age have less dentin than older patients and are at risk for pulp infection. They should have a calcium hydroxide dressing placed, covered with gauze or aluminum foil, and be seen by a dentist within 24 hours. Older patients should see a dentist within 24 to 48 hours.

Ellis class III: This is considered a dental emergency, and dental consultation

within 24 to 48 hours is indicated. Delay in treatment may result in abscess formation.

Root fractures: Early reduction, immobilization/splinting, and dental referral within 24 to 48 hours is indicated. Most teeth sustaining root fractures maintain pulp viability.

Enteral antibiotics for oral microbiota are recommended for Ellis class II and III fractures.



FIGURE 6.10 ■ Ellis Class II Tooth Fractures. Bilateral maxillary central incisor injuries with exposed enamel and dentin consistent with an Ellis class II fracture. (Photo contributor: James F. Steiner, DDS.)

Pearl

1. Check for tooth mobility on initial examination to aid in differentiating mobility involving the entire tooth from involvement of only the fractured segment. Use the blunt end of instruments or tongue depressors on the lingual and buccal surfaces of the tooth to determine tooth mobility.

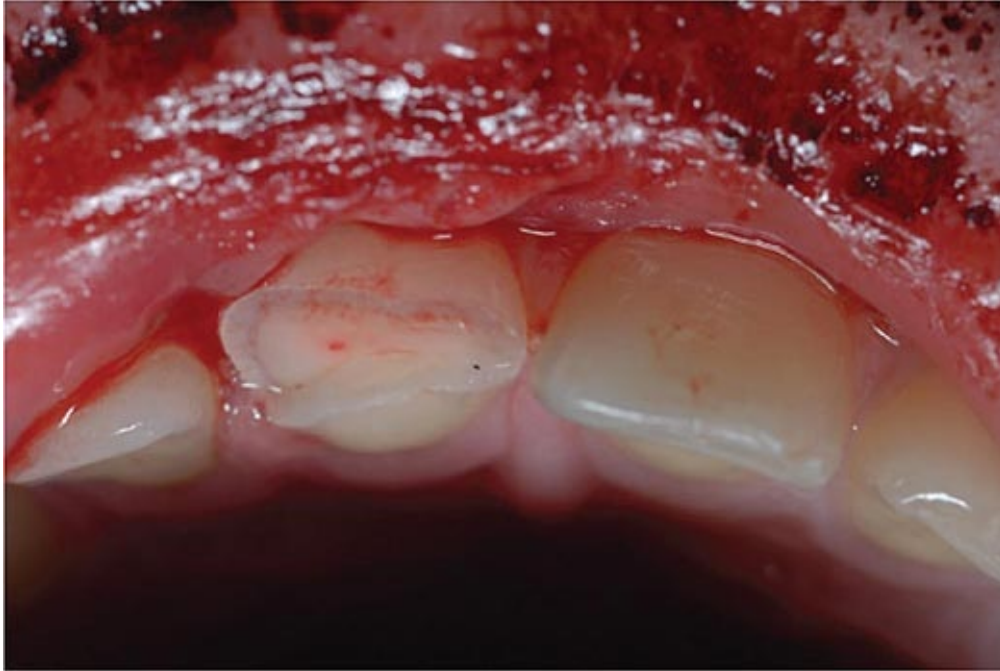


FIGURE 6.11 ■ Ellis Class III Tooth Fracture. A fracture demonstrating blood at the exposed dental pulp. This sign is pathognomonic for an Ellis class III fracture. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 6.12 ■ Multiple Ellis Fractures. Multiple Ellis fractures resulting from blunt trauma: Ellis I fractures are seen in teeth 7 and 11, Ellis II in teeth 8 and 10, and Ellis III in tooth 9. (Photo contributor: Rosie Korman, MD.)

ALVEOLAR RIDGE FRACTURE

Clinical Summary

The alveolus is the tooth-bearing segment of the mandible and maxilla. Fracture of the alveolar process tends to occur most often in the thinner maxilla. The anterior alveolar processes are at greatest risk for fracture due to more direct exposure to trauma. Both subluxation and avulsion of teeth may be associated with underlying alveolar fractures. Various degrees of tooth mobility and gingival bleeding may occur.

Management and Disposition

Significant cosmetic deformity may result from alveolar bone loss; preservation of viable tissue is important. Gentle direct pressure over the alveolar segment with saline-soaked gauze, avulsed teeth preservation, tetanus prophylaxis, and appropriate antibiotic therapy covering oral flora are indicated. Given the degree of trauma involved, computed tomography (CT) imaging may be required to rule out further injuries. Oral surgery consultation should be obtained for possible wire stabilization or arch bar fixation.

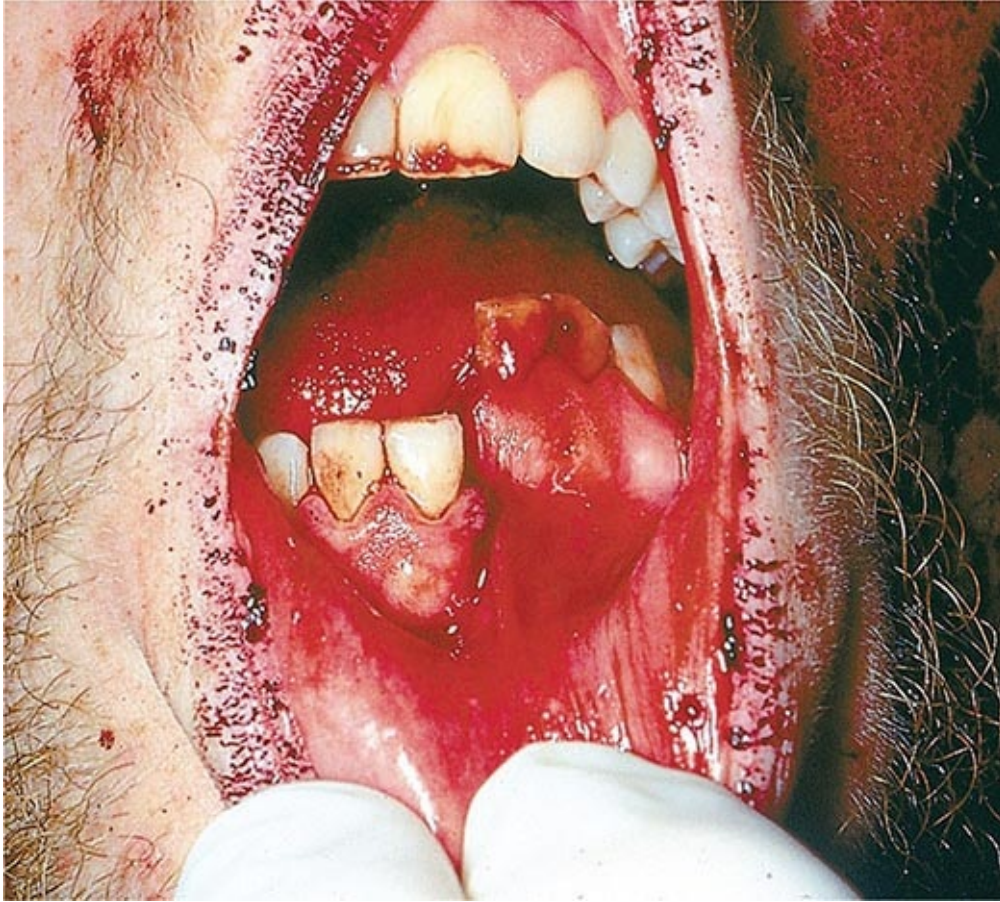


FIGURE 6.13 ■ Alveolar Ridge Fracture. Note the exposed alveolar bone segment and associated multiple teeth involvement. Attempts should be made to maximally preserve all viable tissue. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. Always consider the possibility of an associated cervical spine injury when evaluating patients with facial trauma.
2. Consider potential aspiration of avulsed teeth when teeth are missing.
3. Alveolar ridge fractures may occur in edentulous patients.



FIGURE 6.14 ■ Alveolar Ridge Fracture. This alveolar ridge fracture was caused by blunt trauma from a steering wheel in a frontal impact motor vehicle collision. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 6.15 ■ Alveolar Ridge Fracture. This open upper alveolar ridge fracture was caused by blunt force injury. (Photo contributor: Lawrence B. Stack, MD.)

TEMPORAL MANDIBULAR JOINT DISLOCATION

Clinical Summary

Temporomandibular joint (TMJ) dislocation generally occurs in predisposed individuals after a vigorous yawn or seizure, or less commonly from direct trauma to the chin while the mouth is open. Dislocation occurs when the mandibular condyles displace forward and become locked anterior to the articular eminence. Masseter muscle spasm contributes to prevention of spontaneous relocation. Weakness of the temporomandibular ligament, an overstretched joint capsule, and a shallow articular eminence are predisposing factors. Patients usually present with an inability to close an open mouth. Other associated symptoms include pain, discomfort, facial swelling near the TMJ, and difficulty speaking and swallowing. Anterior dislocations are most common; however, posterior dislocation may occur with significant trauma, often in association with basilar skull fractures. Unilateral dislocation results in deviation of the mandible to the unaffected side. TMJ hemarthrosis and dys-tonic reactions may mimic TMJ dislocations. Mandibular fractures should be considered if there is a history of facial trauma.



FIGURE 6.16 ■ TMJ Dislocation (Bilateral). This patient awoke from sleep with the inability to close her mouth. Note the dry lips and tongue secondary to prolonged exposure. (Photo contributor: Warren K. Russell, MD.)

Management and Disposition

Acute reduction of pain, muscle spasm, and anxiety is achieved using reassurance, analgesics, and benzodiazepine muscle relaxants. Panorex or TMJ x-ray films (prereduction and postreduction) should be considered to exclude a fracture. A reduction maneuver is performed while facing the sitting patient and grasping the angles of the mandible with both hands. The thumbs are wrapped in gauze for protection and rest on the occlusive surfaces of the molars while downward and backward pressure is steadily applied until the condyle slides back into the articular eminence. Reduction may require some time and force to overcome muscle spasm; sedation is often required to achieve reduction. Following reduction, instruct the patient to avoid excessively wide mouth opening while eating and yawning for 3 to 4 weeks. Warm compresses to the TMJ, a soft diet for 1 week, and the use of nonsteroidal anti-inflammatory drugs (NSAIDs) are advised. Dental/oral surgery follow-up should be arranged.

Pearls

1. TMJ dysfunction secondary to a neuroleptic or antipsychotic medication–related dystonic reaction is treated with diphenhydramine or benztropine.
2. When trauma is the cause of TMJ dislocation, maintain a high index of suspicion for mandible fractures and cervical spine injuries.
3. A reportedly successful technique for reduction of TMJ dislocation without sedation involves placing a 5- or 10-mL syringe between the posterior molars and having the patient roll the syringe back and forth while gently biting down until reduction is achieved.



FIGURE 6.17 ■ Bilateral TMJ Dislocation Panorex. This Panorex image demonstrates bilateral anterior displacement of the mandibular condyles. (Photo contributor: Jake Block, MD.)



FIGURE 6.18 ■ TMJ Dislocation, Edentulous Patient (Unilateral). The mandible is deviated toward the unaffected side. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 6.19 ■ TMJ Reduction Technique. Under sedation, the patient in [Fig. 6.18](#) undergoes reduction with inferior-posterior force applied by the physician. The thumbs are not wrapped with gauze in this case as the patient is edentulous. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 6.20 ■ TMJ Reduced. Improved anatomic alignment and a happy patient after reduction of her unilateral TMJ dislocation is accomplished. (Photo contributor: R. Jason Thurman, MD.)

TONGUE LACERATION

Clinical Summary

Injuries to the tongue or mouth floor can cause serious hemorrhage and potential airway compromise. Injury to or absence of teeth should be ascertained by

inspecting the wound for possibly entrapped dental elements. Dorsal surface tongue lacerations may be associated with a mandibular surface laceration.

Management and Disposition

Most tongue lacerations do not require repair. Lacerations involving the tip or lateral margins or lacerations greater than 1 cm in length that gape widely or actively bleed are best stabilized by a few rapidly absorbable interrupted sutures using large bites to include both mucosa and muscle. Anesthesia of the anterior two-thirds of the tongue may be obtained by an inferior alveolar/lingual nerve block. Local anesthesia, infiltrated at the site of the wound, may also be used. Tetanus status should be addressed. Good oral hygiene should be maintained by using chlorhexidine 0.12% topically twice a day for a week.

Pearls

1. Extensive complex tongue lacerations are at risk for infection and should be prophylactically treated with antibiotics covering oropharyngeal microbiota.
2. Regional anesthesia for tongue laceration repair avoids distortion of the anatomy prior to repair and is generally better tolerated than direct infiltration of local anesthesia into the tongue.



FIGURE 6.21 ■ Tongue Laceration. Due to its length and gaping, this tongue laceration was repaired with

absorbable sutures. (Photo contributor: Lawrence B. Stack, MD.)

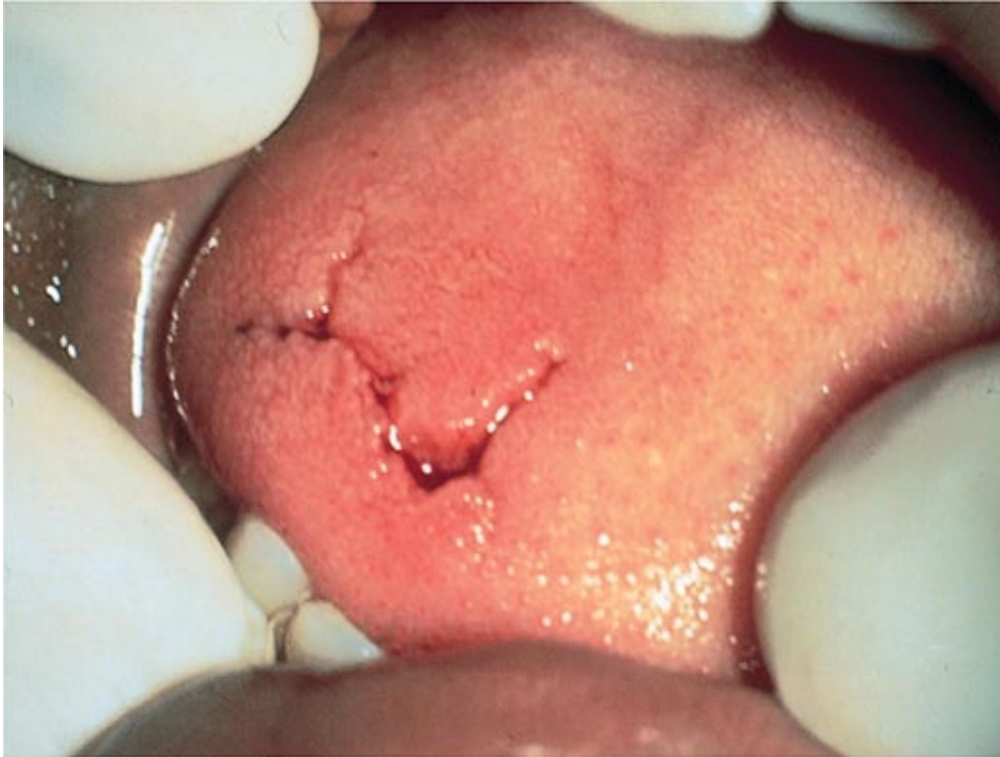


FIGURE 6.22 ■ Tongue Laceration. A stellate tongue laceration that does not require suturing is shown. The ventral aspect of the tongue should be examined for additional lacerations sustained from the mandibular teeth. (Photo contributor: James F. Steiner, DDS.)



FIGURE 6.23 ■ Healing Tongue Edge Laceration. Lacerations of the tip or edge of the tongue should be

reapproximated as chronic defects or “forked tongue” may result. (Photo contributor: Lawrence B. Stack, MD.)

VERMILION BORDER LIP LACERATION

Clinical Summary

Anatomically, the vermilion border of the lips represents the transition area from mucosal tissue to skin. Lip lacerations involving the vermilion border present a unique clinical situation, since relatively minor malalignment may produce an unacceptable cosmetic result. An associated underlying gingival or dental injury is a common finding.

Management and Disposition

Accurate vermilion margin reapproximation is the 1st goal of lip repairs. An unapproximated vermilion margin of 2 mm or greater results in a cosmetic deformity. A regional block of the mental or infraorbital nerve is recommended for anesthesia to avoid additional tissue edema and anatomic distortion produced by local infiltration. Deep or through-and-through lacerations involving the vermilion border should be closed in layers. After closure of the deeper tissue, the 1st skin suture is always placed at the vermilion border to reestablish the anatomic margin. Using 5-0 or 6-0 nylon, sutures should be placed along the vermilion surface until the moist mucous membrane is encountered. The deep muscular and dermal layer may be closed with 4-0 chromic or Vicryl sutures. Mucosal layers are loosely reapproximated with absorbable suture. Update the patient's tetanus status prior to discharge. The patient should be given wound care instructions and follow-up for wound evaluation and possible suture removal within 3 to 7 days.

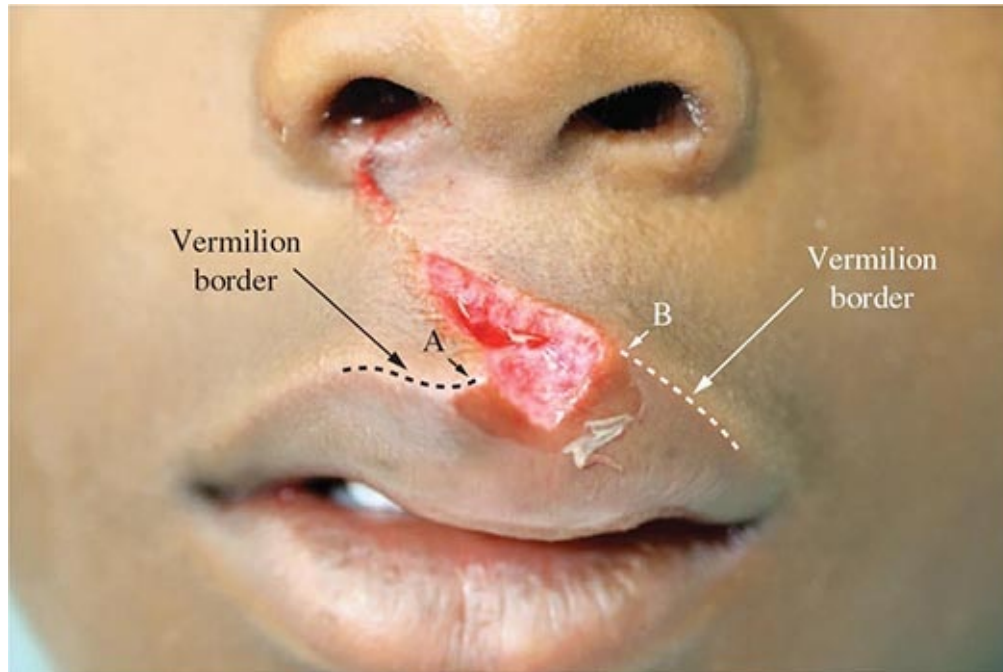


FIGURE 6.24 ■ Vermilion Border Lip Laceration. A lip laceration with disruption of the vermilion border. Wound repair begins at the vermilion-skin junction (precise approximation of A to B in this case) for a good cosmetic result. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. A vermilion border with as little as 2 mm of malalignment may produce a cosmetic defect.
2. Always carefully place the first skin suture at the vermilion border in any lip laceration.



FIGURE 6.25 ■ Complex Vermilion Border Lip Lacerations. This patient suffered vermilion border lacerations in two areas from an assault with a broken bottle. Following shaving of the moustache, each vermilion-skin junction was repaired before remaining superficial wound closure was achieved. (Photo contributor: R. Jason Thurman, MD.)

Odontogenic Infections

GINGIVAL ABSCESS (PERIODONTAL ABSCESS)

Clinical Summary

Gingival abscesses tend to involve the marginal gingiva and result from entrapment of food and plaque debris and subsequent staphylococcal, streptococcal, anaerobic, or mixed bacterial overgrowth. Localized swelling, erythema, tenderness, and fluctuance in the space between the tooth and gingiva ensue. There may be spontaneous purulent drainage from the gingival margin, or an area of abscess pointing. When the gingival abscess involves the deeper supporting periodontal structures, referred to as a periodontal abscess, the patient may present with a fluctuant vestibular abscess or with a draining sinus that opens onto the gingival surface.

In contrast, periapical abscesses are deep and not obvious on inspection. They usually present as tenderness to percussion or pain with chewing over the involved tooth. A parulis may also simulate a gingival abscess; however, a parulis represents the cutaneous manifestation of a deeper periapical abscess. Unlike a parulis or periapical abscess, gingival abscesses are not usually associated with dental caries or fillings. Pericoronal abscesses tend to involve the gingiva overlying a partially erupted 3rd molar.

Management and Disposition

The initial management is, after topical anesthesia, to create a small incision and irrigate with saline. Oral antibiotic therapy, analgesics, and dental follow-up are indicated. The patient's tetanus status should be addressed.

Pearls

1. Patients with gingival abscesses are usually afebrile. Consider more extensive abscess formation and oral disease processes in the febrile toxic-appearing patient.
2. Patients with chronic, deep periodontal abscesses complain of dull, gnawing pain as well as a desire to bite down on and grind the tooth.



FIGURE 6.26 ■ Periodontal Abscess. Localized gingival swelling, erythema, and fluctuance are seen in this periodontal abscess with spontaneous purulent drainage. (Photo contributor: Kevin J. Knoop, MD, MS.)

PERIAPICAL ABSCESS

Clinical Summary

Acute pain, swelling, and mild tooth elevation are characteristics of a periapical or dentoalveolar abscess. Exquisite sensitivity to percussion or chewing on the involved tooth is a common sign. The involved tooth may have had dental

caries, a filling, or a root canal. Periapical abscesses can enlarge over time and “point,” either internally on the lingual or buccal mucosal surfaces, or extraorally with swelling and redness of the overlying skin. Occasionally, these lesions may track up to the alveolar periosteum and gingival surface to form a parulis. Radiographically, these abscesses appear as well-circumscribed areas of radiolucency at the dental apex or along the lateral aspect of the root. Early acute periapical abscesses may not demonstrate any radiographic changes. Both deep periodontal and periapical abscesses may have sinuses draining purulent material onto the gingival surface. If the infection is allowed to progress, it can erode through cortical bone, manifesting itself in a variety of locations. Panorex films, dental radiographs, or a CT scan may aid in the diagnosis.

Management and Disposition

Analgesics, tetanus prophylaxis, and antibiotic therapy are indicated. A regional nerve block provides immediate temporary relief. Incision and drainage along with saline irrigation and prompt referral are indicated for parulis management. Dental follow-up in 1 to 2 days is recommended for endodontic evaluation or possible extraction of the involved tooth.

Pearls

1. More than one tooth may be involved.
2. Exquisite tenderness and pain on tooth percussion are key features on physical examination and identify the involved tooth.



FIGURE 6.27 ■ Periapical Abscess. This periapical abscess points externally to the overlying skin. (Photo contributor: Robin Cotton, MD.)



FIGURE 6.28 ■ “Gumboil” (Parulis). This lesion is a sinus tract extension of a periapical abscess. It is differentiated from a periodontal abscess by tenderness to tooth percussion. (Photo contributor: Alan B. Storrow, MD.)

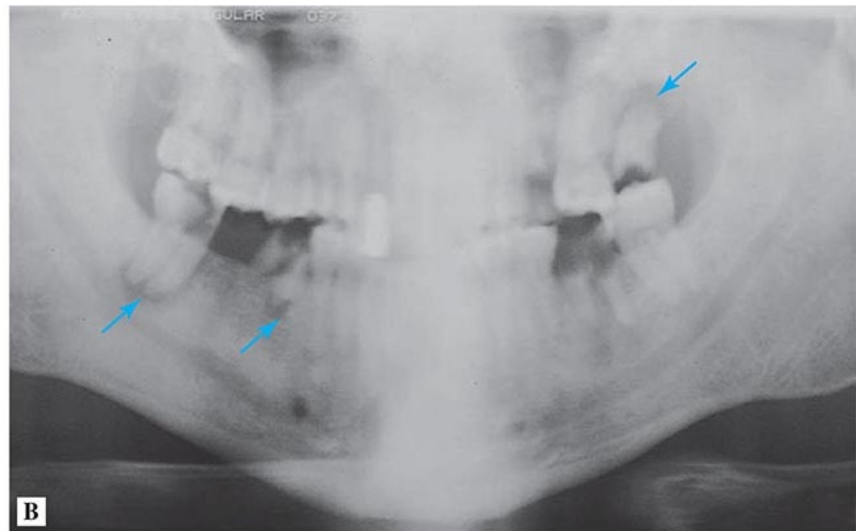


FIGURE 6.29 ■ Periapical Abscess. (A) Note the well-defined radiolucent area at the apex and lateral root of the tooth in this radiograph. (Photo contributor: James L. Kretzschmar, DDS, MBS.) (B) This Panorex film shows lucencies consistent with periapical abscesses, most notably of the right inferior posterior molar tooth (see arrows). (Photo contributor: David P. Kretzschmar, DDS, MS.)

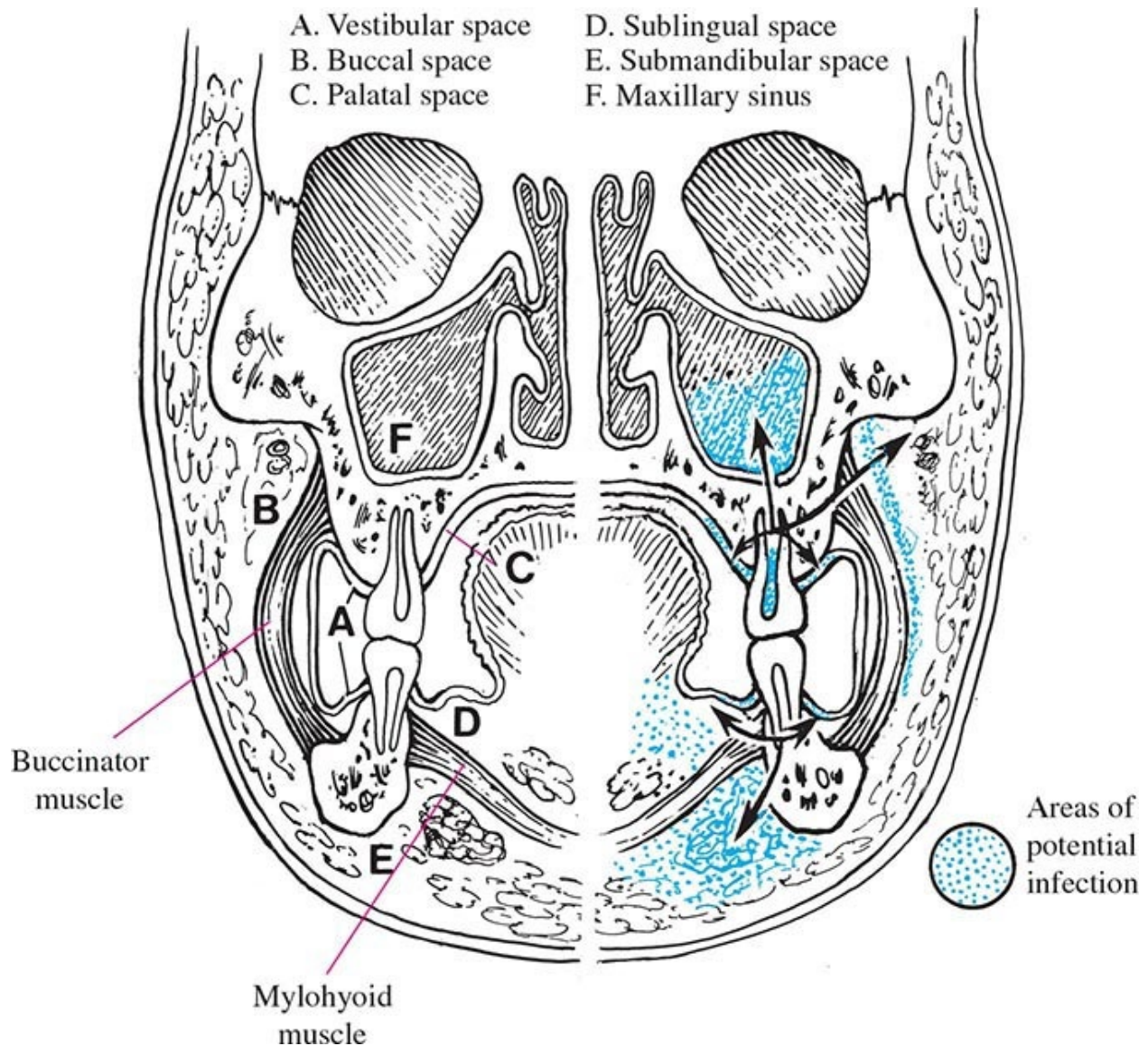


FIGURE 6.30 ■ Odontogenic Abscesses. As infection progresses from the pulp at the tooth apex, it erodes through the bone and can express itself in a variety of places. This illustration notes several possible locations or spaces. (Adapted with permission from Cummings C, Schuller D, eds. *Otolaryngology Head and Neck Surgery*. Chicago, IL: Mosby-Year Book; 1986.)

PERICORONAL ABSCESS

Clinical Summary

A partially erupted or impacted 3rd molar (wisdom tooth) is the most common site of pericoronitis and pericoronal abscesses. The accumulation of food and

debris between the overlying gingival flap and crown of the tooth creates a focus for pericoronitis and subsequent abscess formation. The gingival flap becomes irritated and inflamed, and the tissue is repeatedly traumatized by the opposing molar tooth. The inflamed gingival process may eventually become infected and form an abscess. Foul taste, inability to close the jaw, and fever may occur. Swelling of the cheek and angle of the jaw and localized lymphadenopathy are also characteristic. More advanced disease may spread posteriorly to the base of the tongue, oropharyngeal area, and deep cervical spaces with resulting Ludwig angina and peritonsillar abscesses.

Management and Disposition

Superficial incision and drainage of the abscess with warm saline irrigation, analgesia, and antibiotic coverage and referral for possible extraction of the involved teeth are indicated.

Pearls

1. Pericoronitis and abscess formation rarely occur in the pediatric population and tend to be late adolescent and adult processes; the mandibular 3rd molar is the most commonly involved tooth.
2. Airway compromise is a rare but potential complication from posterior extension of a pericoronal abscess.

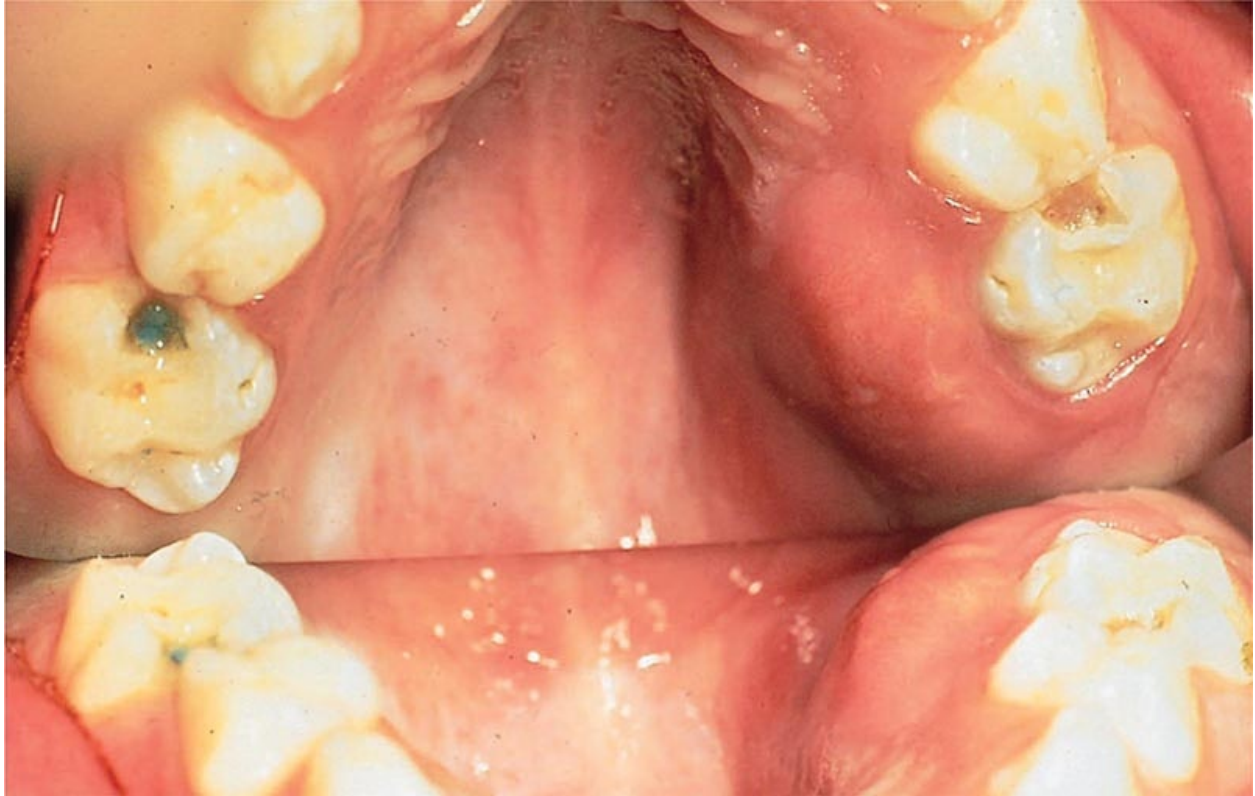


FIGURE 6.31 ■ Pericoronal Abscess. Note the inflamed fluctuant gingival tissue approximating the incompletely erupted third molar. (Photo contributor: James F. Steiner, DDS.)

HARD PALATE ABSCESS

Clinical Summary

Hard palate abscesses are most commonly related to maxillary lateral incisors or palatal roots of the posterior teeth, especially maxillary 1st molar and premolars. The infection starts at the tooth apex and erodes through the palatal bone, accumulating into the palatal mucoperiosteum. The lesion is fluctuant, is usually very painful with a paramedian location, and typically does not cross the palate's midline. Palatal abscesses need to be differentiated from other pathologies such as salivary gland tumors, neural tumors, cysts, fibromas, and torus palatinus.



FIGURE 6.32 ■ Hard Palate Abscess. This palatal abscess originated from the palatal roots of teeth 6 and 7. The area was markedly fluctuant to palpation. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 6.33 ■ Hard Palate Abscess. A hard palate abscess originating from the palatal root of the patient's right second molar. A large amount of purulence was expressed with incision. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Treatment is the same as for any other orofacial abscess: incision and drainage, antibiotics, and dental follow-up are the mainstays of therapy. Definitive treatment, usually performed by a dentist, includes extraction or root canal therapy of the offending tooth. The patient's tetanus status should be addressed.

Pearls

1. Incision and drainage of this area should be done parallel to dentition, near the border of the gingivae or toward the midline, avoiding injury to the greater palatine neurovascular bundle.
2. The absence of fever does not preclude the diagnosis of palatal abscess.

BUCCAL SPACE ABSCESS

Clinical Summary

The buccal space lies between the buccinator muscle and the overlying superficial fascia and skin. The maxillary 2nd and 3rd molars are the usual nidus of buccal space infections, eroding either superiorly through the maxillary alveolar bone or, rarely, inferiorly from the 3rd mandibular molar through the mandibular alveolar bone into the buccal space. Patients usually present with unilateral facial swelling, redness, and tenderness of the cheek. Trismus is generally not present. Parotid gland enlargement due to mumps and suppurative bacterial parotitis should also be considered. The former lacks erythema and warmth of the overlying skin, while the latter is accompanied by trismus and purulent drainage from Stensen's duct. Inspection of all the maxillary and 3rd mandibular molar teeth is essential to make the diagnosis. A CT scan may help localize the infection.

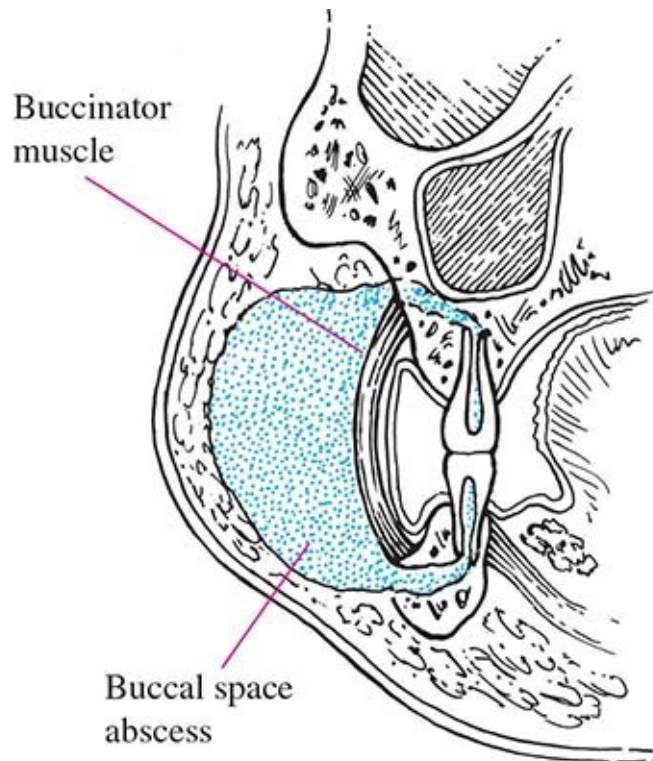


FIGURE 6.34 ■ Buccal Space Anatomy. The buccal space lies between the buccinator muscle and the overlying skin and superficial fascia. This potential space may become involved secondary to maxillary or mandibular molar infections. (Adapted with permission from Cummings C, Schuller D, eds. *Otolaryngology Head and Neck Surgery*. 2nd ed. Chicago, IL: Mosby-Year Book; 1993.)

Management and Disposition

Broad-spectrum parenteral antibiotic therapy, oral analgesics, and dental or oral surgical consultation for endodontic therapy, abscess drainage, and possibly dental extraction are indicated.

Pearls

1. Odontogenic infections of the 2nd or 3rd maxillary molars are the most common source for buccal space abscesses.
2. Infection can spread from the buccal space to the cavernous sinus via the transverse facial vein. Care should be taken to evaluate for any signs of cavernous sinus thrombosis in patients with buccal space infections.



FIGURE 6.35 ■ Buccal Space Abscess. Note the ovoid cheek swelling with sparing of the nasolabial fold. This finding, along with accompanying redness and tenderness, helps to identify buccal space abscess formation. (Photo contributor: Michael J. Nowicki, MD.)



FIGURE 6.36 ■ Buccal Space Abscess. Note the marked facial asymmetry, slight overlying erythema, and flattening of the left nasolabial fold in this adult patient presenting with a left-sided buccal space abscess. (Photo contributor: R. Jason Thurman, MD.)

CANINE SPACE ABSCESS

Clinical Summary

The canine space lies between the anterior surface of the maxilla and levator labii superioris muscle of the face. Erosion of a maxillary tooth infection through the alveolar bone into the canine space leads to abscess formation, although cutaneous infections from the upper lip and nose are rare sources. Uni-lateral facial redness, pain, and swelling lateral to the nose with obliteration of the nasolabial fold are characteristic. Severe upper lip and lower eyelid swelling may cause drooling at the corner of the mouth or eye. Maxillofacial CT scan may aid in differentiating these lesions.

Management and Disposition

Parenteral broad-spectrum antibiotic therapy including anaerobic coverage is indicated. Dental or oral surgical consultation for incision and drainage is the most definitive treatment for canine space abscesses. Extraction or endodontic treatment of the involved anterior maxillary teeth is usually necessary.

Pearls

1. Loss of the nasolabial fold is usually present with canine space abscesses, but may not be seen with buccal space abscesses.
2. Canine space infections may lead to orbital cellulitis and cavernous sinus thrombosis.
3. Although these patients may drool when significant upper lip swelling is present, they typically do not have trismus, dysphagia, or odynophagia.



FIGURE 6.37 ■ Canine Space Abscess. Unilateral facial swelling lateral to the nose with associated redness and the typical loss of the nasolabial fold is shown. The maxillary canine tooth is usually the source of this process. (Photo contributor: Frank Birinyi, MD.)

LUDWIG ANGINA

Clinical Summary

Ludwig angina is defined as rapidly spreading bilateral cellulitis of the submandibular and sublingual spaces with associated tongue elevation. A

characteristic painful, brawny induration is present in the involved tissue. The posterior mandibular molars are the usual odontogenic origin for the infection. *Streptococcus*, *Staphylococcus*, and *Bacteroides* species are the most common pathogens. Affected individuals are typically 20 to 60 years old, with a male predominance. Patients are usually febrile and may demonstrate impressive trismus, dysphonia, and odynophagia. Dysphagia and drooling are secondary to tongue displacement and oropharyngeal swelling. Potential airway compromise or spread of infection to the deep cervical layers and the mediastinum is possible.

Management and Disposition

Acute laryngospasm with airway compromise is a potentially life-threatening complication; thorough plans for definitive airway management should be prepared since up to one-third of patients require definitive airway placement. Broad-spectrum aerobic and anaerobic parenteral antibiotic therapy should be initiated. Analgesia should be given as needed. CT or magnetic resonance imaging (MRI) can identify abscess location, but great care should be taken to ensure the patient can protect the airway while tolerating the imaging process. Emergent otolaryngologic or oral surgical consultation is warranted for definitive intraoperative incision and drainage of the abscess. Admission to the intensive care unit is indicated for airway surveillance and management.



FIGURE 6.38 ■ Ludwig Angina. Note the diffuse submandibular swelling and fullness. Direct palpation of this area would reveal a characteristic brawny induration. Potential airway compromise is a key concern in all patients with Ludwig angina. (Photo contributor: Jeffrey Finkelstein, MD.)

Pearls

1. Brawny submandibular induration and tongue elevation are common and characteristic clinical findings.
2. The 2nd mandibular molar is the most common site of origin for Ludwig angina.
3. Acute laryngospasm with sudden total airway obstruction may be precipitated by attempts at oral or nasotracheal intubation.



FIGURE 6.39 ■ Ludwig Angina. Sagittal view on CT scan demonstrates fluid collection and abscess formation within the floor of the mouth along with superior displacement of the tongue in this patient with Ludwig angina. (Photo contributor: Jake Block, MD.)

PARAPHARYNGEAL SPACE ABSCESS

Clinical Summary

The parapharyngeal space, also known as the lateral pharyngeal or pharyngomaxillary space, is a pyramidal-shaped space with its apex at the hyoid bone and base at the base of the skull. Laterally, it is bound by the internal pterygoid muscle and the parotid gland with the superior pharyngeal constrictor muscle acting as the medial border. The posterior aspect of this space is in close proximity with the carotid sheath and cranial nerves IX through XII. Presenting symptoms of parapharyngeal space abscesses may include fever, dysphagia, odynophagia, drooling, and ipsilateral otalgia. Unilateral neck and jaw angle

facial swelling in association with rigidity and limited neck motion is common. Potentially disastrous complications include cranial neuropathies, jugular vein septic thrombophlebitis, and erosion into the carotid artery. The origin of parapharyngeal abscesses may be from bacterial pharyngitis, sinuses, dentition, or lymphatic spread.

Management and Disposition

Preparations for definitive airway management via endotracheal intubation or surgery are vital. Early recognition and anticipation of other potentially disastrous complications should be considered and managed appropriately. Broad-spectrum parenteral antibiotic coverage for mixed aerobic and anaerobic infections (penicillin or clindamycin with metronidazole; consider adding vancomycin in high-risk groups) should be initiated. Radiologic modalities used to assess parapharyngeal and other deep-space neck infections include contrast-enhanced CT, ultrasound, plain radiography, and MRI. Emergent otolaryngologic or oral surgical consultation is warranted for definitive intraoperative incision and drainage of the abscess.

Pearls

1. Suspected oropharyngeal abscesses in association with cranial nerves IX through XII involvement are pathognomonic of parapharyngeal abscesses.
2. Early preparation and well-planned advanced airway management are critical in the management of parapharyngeal abscesses.



FIGURE 6.40 ■ Parapharyngeal Space Abscess. Unilateral facial, jaw angle, and neck swelling is seen in this patient. Nuchal rigidity may also be present. (Photo contributor: Sara-Jo Gahm, MD.)

Oral Conditions

TRENCH MOUTH (ACUTE NECROTIZING

ULCERATIVE GINGIVITIS)

Clinical Summary

Painful, severely edematous interdental papillae are characteristic of acute necrotizing ulcerative gingivitis (ANUG), or trench mouth. Other associated features include the presence of ulcers with an overlying grayish pseudomembrane and a “punched out” appearance. The inflamed gingival tissue is friable and necrotic and represents an acute destructive disease process of the periodontium. Fever, malaise, and regional lymphadenopathy are commonly associated signs. Patients may also complain of foul breath and a strong metallic taste. Poor oral hygiene, emotional stress, smoking, and immunocompromised states (eg, HIV, steroid use, diabetes) all predispose for ANUG. Anaerobic *Fusobacterium* and spirochetes are the predominant bacterial organisms involved. The anterior incisor and posterior molar gingival regions are the most commonly affected oral tissues. Acute herpetic, gonococcal, or streptococcal gingivostomatitis, aphthous stomatitis, desquamative gingivitis, and chronic periodontal disease may mimic ANUG.

Management and Disposition

Initial management includes warm saline irrigation, analgesics, topical anesthetics, 0.12% chlorhexidine oral rinses, and antibiotic treatment with oropharyngeal coverage. Follow-up within 1 to 2 days is recommended. Patients with more advanced disease may require admission and oral surgical consultation.

Pearls

1. Dramatic relief of symptoms within 24 hours of initiating antibiotics and supportive treatment is characteristic.



FIGURE 6.41 ■ Acute Necrotizing Ulcerative Gingivitis. Note the inflamed, friable, and necrotic gingival tissue. An overlying grayish pseudomembrane or punched out ulcerations of the interdental papillae are pathognomonic. (Photo contributor: David P. Kretschmar, DDS, MS.)

2. Periodontal abscesses and underlying alveolar bone destruction are common complications of ANUG and require dental follow-up.
3. Gingivitis is a nontender inflammatory disorder and is not synonymous with ANUG.
4. Consider underlying medical condition (immunodeficiency, nutritional deficiency).



FIGURE 6.42 ■ Acute Necrotizing Ulcerative Gingivitis. Excessive purulent drainage was associated with this patient's ANUG. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 6.43 ■ Severe Acute Necrotizing Ulcerative Gingivitis. Note the extensive gingival necrosis in this advanced case of trench mouth. (Photo contributor: David Effron, MD.)

ACID TOOTH EROSION (BULIMIA)

Clinical Summary

Bulimia nervosa is an eating disorder with significant associated physical complications. It is characterized by binge eating with self-induced vomiting, laxative use, dieting, and exercise to prevent weight gain. Patients with bulimia are at risk for damage to the dental enamel and dentin as a result of repeated episodes of vomiting with chronic exposure to regurgitated acidic gastric contents. The lingual dental surfaces are most commonly affected. In severe cases, all surfaces of the teeth may be affected. Trauma to the oral and esophageal mucosa may also result from induced vomiting. The quantity, buffering capacity, and pH of both the resting and stimulated saliva are found to be reduced, and salivary gland enlargement, most commonly the parotid, may occur as well.

Included in the differential diagnosis of acid tooth erosion are conditions that involve recurrent vomiting. Xerostomia is a condition of excessive mouth dryness (associated with Sjögren syndrome) that can also accelerate the process of enamel loss. Tooth abrasions and erosions may be brought about by the use of chewing tobacco, eating betel nuts (Indian *paan*), dentifrice use, bruxism, abnormal swallowing, and jaw clenching.



FIGURE 6.44 ■ Acid Tooth Erosion (Bulimia). Erosive dentin exposure of the maxillary teeth secondary to chronic vomiting. The involvement of the lingual dental surfaces is characteristic of bulimia. (Photo contributor: David P. Kretzschmar, DDS, MS.)

Management and Disposition

The initial emergency department management of patients with bulimia should address any medical complication of the disorder (eg, hypokalemia, metabolic acidosis). Hospitalization to stabilize medical complications and provide nutritional support may be indicated. Dental treatment should begin with vigorous oral hygiene to prevent further destruction of tooth structures. Regular professional fluoride treatments to cover exposed dentin and cosmetic procedures should be instituted once the patient is adequately stabilized psychologically. A multidisciplinary team approach is necessary and should involve psychiatry, internal medicine, and dental consultation as needed.

Pearls

1. The lingual surfaces of the teeth are the most commonly involved tooth surfaces.
2. Bruxism tends to cause enamel loss from occlusal and incisal dental surfaces.
3. The labial and buccal surfaces of the teeth tend to show enamel loss from repeat or prolonged chemical contact (eg, lemon sucking or tobacco).

products).



FIGURE 6.45 ■ Acid Tooth Erosion (Snuff User). Note the typical dentin exposure on the buccal dental surfaces resulting from prolonged snuff use and its accompanying acid erosion. (Photo contributor: David P. Kretzschmar, DDS, MS.)

THRUSH (ORAL CANDIDIASIS)

Clinical Summary

White, flaky, curd-like plaques covering the tongue and buccal mucosa with an erythematous base are typical of thrush. These lesions tend to be painless, although some patients experience a burning sensation. Painful inflammatory erosions or ulcers may be noted, particularly in adults. Predisposing factors include antibiotic use, inhaled and oral corticosteroids, radiation to the head and neck, extremes of ages, patients with immunologic deficiencies, and chronic irritation (eg, denture use and xerostomia). Colonization of surface epithelium by *Candida* occurs due to altered oral microflora. Hairy leukoplakia, lingual lichen planus, flecks of milk or food debris, and liquid antacid adhering to the tongue may be confused with candidiasis. Hairy leukoplakia cannot be removed with a tongue depressor (note [Fig. 20.4](#)). This helps differentiate this process from thrush or residue from ingested materials. Microscopic examination of the

removed specimen for the presence of hyphae in potassium hydroxide mount will aid in the identification of *Candida*.



FIGURE 6.46 ■ Oral Candidiasis (Thrush). Whitish plaques are seen here on the buccal mucosa. These plaques are easily removed with a tongue blade, differentiating them from lichen planus or leukoplakia. (Photo contributor: James F. Steiner, DDS.)

Management and Disposition

Nystatin oral tablets or swish and swallow suspension, fluconazole, or clotrimazole oral troches are therapeutic. Topical analgesic cocktails may provide comfort for patients (eg, liquid antacid with diphenhydramine, viscous lidocaine).

Pearls

1. Thrush is most common in premature infants and immuno-suppressed patients.
2. In young adults, thrush may be the first sign of AIDS; a history of HIV risk

factors should be elicited.

3. Failure of oral candidiasis to respond to topical antifungal agents may suggest an underlying immune deficiency.



FIGURE 6.47 ■ Oral Candidiasis (Thrush). Extensive thrush is seen on the hard and soft palate and uvula of this immunocompromised patient. (Photo contributor: Lawrence B. Stack, MD.)

ORAL HERPES SIMPLEX VIRUS (COLD SORES)

Clinical Summary

Oral herpes simplex may present acutely as a primary gingivostomatitis or as a recurrence. Painful vesicular eruptions on the oral mucosa, tongue, palate, vermillion borders, and gingiva are highly characteristic. A 2- to 3-day prodromal period of malaise, fever, and cervical adenopathy is common. The vesicular lesions rupture to form a tender ulcer with yellow crusting and an erythematous margin. Pain may be severe enough to cause drooling and odynophagia, which can discourage eating and drinking, particularly in children. The disease tends to run its course in a 7- to 10-day period with nonscarring resolution of the lesions. Recurrent herpes labialis may present with an aura of burning, itching, or tingling prior to vesicle formation. Oral trauma, sunburn, stress, and any variety of febrile illnesses can precipitate this condition. Oral erythema multiforme or

Stevens-Johnson syndrome, aphthous lesions, oral pemphigus, and hand-foot-mouth (HFM) syndrome are in the differential diagnosis. It should be noted that aphthous ulcers tend to occur on movable oral mucosa and rarely on immovable mucosa (ie, hard palate and gingiva). The vermilion border is a characteristic location for herpes labialis as opposed to aphthous lesions. Posterior oropharyngeal ulcerations with associated hand and foot lesions help to define HFM syndrome.



FIGURE 6.48 ■ Herpes Simplex Virus (HSV) Stomatitis. Note the extensive painful ulcerations on the patient's lower lip. A prodromal period of fever, malaise, and cervical adenopathy may herald the onset of these painful ulcerations. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Supportive care with rehydration and pain control is the mainstay of therapy. Temporary pain relief may be achieved with topical analgesics. Viscous lidocaine may be used as an oral rinse in an age-appropriate dose and form. Oral antiviral agents may be useful with primary infections. Secondary infection of herpetic lesions should be treated with antibiotics.

Pearls

1. Fatal viremia and systemic involvement may occur in infants and children

with herpetic gingivostomatitis.

2. Primary acute oral herpetic infection occurs most commonly in children and young adults.
3. Initiation of suppressive therapy with oral antivirals should be considered within the 1st 72 hours from symptom onset.



FIGURE 6.49 ■ HSV Stomatitis. Extensive vesicular lesions along the vermilion border and surrounding tissues are consistent with HSV infection. (Photo contributor: Frank Birinyi, MD.)

APHTHOUS ULCERS (CANKER SORES)

Clinical Summary

Aphthous ulcers are shallow, 1- to 15-mm, painful mucosal ulcers. A prodromal burning sensation may be noted 2 to 48 hours before an ulcer is noted. The initial lesion is a small white papule that ulcerates and enlarges over 48 to 72 hours. Lesions are typically round or ovoid with a raised yellow border and surrounding erythema. Multiple aphthous ulcers may occur on the lips, tongue, buccal mucosa, floor of the mouth, or soft palate. Spontaneous healing occurs in 7 to 10 days without scarring. The exact etiology is unknown but is believed to involve an immune response to various triggers. Deficiencies of vitamin B₁₂, folic acid, and iron as well as viruses have been implicated. Stress, local trauma, and immunocompromised states have all been cited as possible precipitators.

Primary or recurrent herpetic oral lesions may present with an almost identical prodrome and similar appearance. Herpetic lesions, unlike aphthous ones, tend to occur on the gingiva, hard palate, and vermilion border. Oral erythema multiforme may also present similarly to aphthous stomatitis; however, like oral herpes, it may tend to present with multiple vesicles in the early stages. Stevens-Johnson syndrome represents a severe form of erythema multiforme characterized by hemorrhagic anogenital and conjunctival lesions as well as oral lesions. Herpangina results from coxsackieviruses and Echoviruses, with oral ulcerations typically involving the posterior pharynx. Oral pemphigus should also be considered. Behçet disease can present with recurrent oral lesions, genital ulcers, and uveitis.



FIGURE 6.50 ■ Aphthous Ulcerations. Note the multiple ulcers of various sizes located on the lip and gingival mucosa. These lesions rarely occur on the immobile oral mucosa of the gingiva or hard palate.

(Photo contributor: James F. Steiner, DDS.)

Management and Disposition

Supportive care, rehydration, and pain control constitute the focus of therapy. A topical anesthetic agent such as 2% viscous lidocaine or liquid antihistamine/antacid mix as an oral rinse every 3 to 4 hours is palliative. Use of oral antimicrobial rinses promotes healing. Protective dental paste may be applied every 6 hours to prevent irritation of lesions. Triamcinolone acetonide in an emollient dental paste three to four times daily may reduce pain and promote healing.

Pearls

1. Aphthous ulcers may be associated with Crohn disease.
2. Topical anesthetics may be used as a temporary adjunct in pain relief. Care must be taken to avoid overdose or complications such as methemoglobinemia from overuse.
3. Aphthous ulcers almost never occur on gums or hard palate.



FIGURE 6.51 ■ Aphthous Ulcers on the Tongue. This patient presented with severe pain on the tongue along with difficulty eating and drinking secondary to the pain. Multiple aphthous ulcers are present. (Photo contributor: Lawrence B. Stack, MD.)

STRAWBERRY TONGUE

Clinical Summary

Reddened hypertrophied lingual papillae, called strawberry tongue, are primarily associated with scarlet fever caused by group A streptococcal enterotoxin. The tongue initially appears white with the erythematous papillae sticking through the white exudate. After several days, the white coating is lost and the tongue appears bright red. Other signs of group A streptococcal infection include fever, an exudative pharyngitis, a scarlatiniform rash, and the presence of Pastia lines (pete-chial linear rash in the skin folds). Kawasaki syndrome may also present with an injected pharynx and an erythematous strawberry-like tongue. It is essential to make the distinction between streptococcal infection and Kawasaki syndrome, since the latter is associated with a high incidence of coronary artery aneurysm if left untreated. Also important to consider is toxic shock syndrome, in which one-half to three-fourths of patients have pharyngitis with a strawberry-red tongue.



FIGURE 6.52 ■ Strawberry Tongue. Note the white exudate with bulging red papillae. The white coating is eventually lost after several days, and the tongue then appears bright red. (Photo contributor: Michael J.

Nowicki, MD.)

Management and Disposition

Rapid streptococcal immunoassay testing may help expedite the diagnosis. Pharyngeal cultures are useful for confirming the diagnosis. Antistreptolysin O (ASO) titers can be used for confirmation in the convalescent stage if the diagnosis is in question. Penicillin is the drug of choice for group A *Streptococcus*.

Pearls

1. Strawberry tongue initially appears white in color, with prominent red papillae bulging through the white exudate. After several days, the tongue becomes completely beefy red.
2. Kawasaki patients may also present with conjunctivitis and discoloration of the palms and soles.



FIGURE 6.53 ■ Strawberry Tongue. This patient with scarlet fever demonstrates the bright red appearance of strawberry tongue after most of the white exudate is lost. (Photo contributor: Kevin J. Knoop, MD, MS.)

BLACK HAIRY TONGUE

Clinical Summary

Black hairy tongue (BHT) represents a benign reactive process characterized by hyperplasia and dark pigmentation of the tongue's filiform papillae. The elongated filiform papillae may reach up to 2 cm in length and vary in actual degree of pigmentation from light tan to black. Predisposing factors include excessive smoking, gastroesophageal reflux, poor oral hygiene, chemotherapy, and the use of broad-spectrum oral antibiotics. Pigment from consumed food, beverages, and tobacco products stains the entrapped food debris and desquamated papillary keratin. Some antibiotics may alter normal oral

microflora and promote the growth of chromogenic organisms, also contributing to the tongue's discoloration. The darkly pigmented filament-like papillae give the tongue a black, hairy appearance. Males are more often affected than females; this condition very rarely occurs in children. Patients with BHT frequently note alterations of taste perception and fetid breath.

Geographic tongue and orolingual candidiasis may resemble more lightly pigmented forms of BHT. Similarly, dark discoloration of normal tongue papillae may also mimic BHT. This exogenous pigmentation of normal papillae may come from ingested food dyes and certain medications, such as bismuth-containing compounds, ketoconazole, and azidothymidine. The lack of hyperplastic filiform papillae with additional pigmentation of other oral mucosal surfaces may aid in distinguishing these conditions.



FIGURE 6.54 ■ Black Hairy Tongue. Hyperplasia of the filiform papillae on the dorsum of the tongue accompanied by deposition of dark pigment is characteristic of black hairy tongue. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

Management and Disposition

Improved oral hygiene with gentle tongue brushing and a reduction in the

ingestion of exogenous pigment-containing substance represent the cornerstones of treatment. Removal of other predisposing factors (eg, antibiotic withdrawal and smoking cessation) will also promote resolution of this condition. The use of topically applied retinoid preparations and antifungal agents has been advocated for more refractory instances.

Pearls

1. BHT involves the superior aspect of the tongue.
2. The tongue is not always black and can be a tan or yellow color.



FIGURE 6.55 ■ Black Hairy Tongue. Deposition of black pigment secondary to bismuth ingestion. This patient ingested Pepto-Bismol. (Photo contributor: Kevin J. Knoop, MD, MS.)

ORAL EXOSTOSES

Clinical Summary

Oral exostoses are benign boney growths on the mandible or hard palate, including tori and buccal exostosis. Tori are benign nodular overgrowths of cortical bone, occurring in the midline of the palate where the maxilla fuses (palatinus) or on the mandible, typically on the lingual aspect of the molar teeth (mandibularis). Although their physical appearance may be alarming, there is generally no need for concern. Tori are covered by a thin epithelium, which is easily traumatized and ulcerated. These ulcerations tend to heal very slowly because of the poor vascularization of the tori. Torus palatinus, in particular, is slow growing and may occur at any age; however, it is most commonly noted prior to age 30 in adults and affects women twice as frequently as men. Buccal exostoses are typically found on the buccal surface of the maxillary or mandibular alveolar ridge adjacent to premolar teeth. There are a variety of oral conditions that may be confused with exostoses. Gingival fibromatosis, fibroma formation secondary to irritation, granulomas, abscesses, and oral neurofibromatosis located on the palate may resemble torus palatinus. Nodular bony enlargement in the oral cavity may also result from fibrous dysplasia, osteomas, and Paget disease. Oral malignancies may manifest themselves on the palate as primary lesions, although these are rare. Oral radiographs, CT scans, and biopsy may aid in differentiating these conditions.



FIGURE 6.56 ■ Torus Palatinus. Note the nodular appearance and characteristic central palatal location. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 6.57 ■ Torus Palatinus. Due to location, abrasions and ulcerations can occur on the thin overlying epithelium secondary to trauma. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 6.58 ■ Torus Mandibularis. The typical location of torus mandibularis, with exostosis on the lingual aspect of the molar teeth. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 6.59 ■ Torus Mandibularis. Oral exostoses are benign overgrowths of cortical bone but can have an alarming appearance that mimics oral malignancies. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Exostoses are normal structural variants and do not represent an inflammatory or neoplastic process. They require no treatment unless associated with a complication. Exostoses may enlarge enough to interfere with eating or speaking and impair proper fitting of dental prostheses. For some patients, the mere presence of exostoses may be bothersome and undesirable. Referral is indicated for further evaluation and definitive diagnosis.

Pearls

1. Exostoses are nontender and otherwise asymptomatic unless traumatized.
2. Buccal exostoses are commonly bilateral.

GINGIVAL HYPERPLASIA

Clinical Summary

Gingival hyperplasia, or overgrowth of the gingiva, is a relatively rare condition. Most commonly, gingival hyperplasia is drug-induced, with phenytoin, cyclosporine, and calcium channel blockers most commonly implicated. Gingival hyperplasia may also occur from genetic disorders, myelodysplastic syndromes, granulomatous disease, and neoplastic processes, and may occur in pregnancy or puberty. Generally, the condition is asymptomatic, but patients may present to the emergency department (ED) due to bleeding gums, traumatized hyperplastic tissue, dental pain, gingival soreness, or associated periodontal disease.

Management and Disposition

ED treatment is geared toward symptomatic relief and identification of the underlying etiology. History may reveal a causative medication, while laboratory testing should rule out underlying blood dyscrasias. Good dental hygiene and dental follow-up are essential. Removal or reduced dosing of the offending agent may be considered. Gingivoplasty or gingivectomy may be necessary in extreme cases.

Pearls

1. Gingival hyperplasia may occur in up to 50% of patients taking phenytoin.
2. Underlying malignancies, especially leukemias, should be considered in the differential diagnosis of patients presenting to the ED with gingival hyperplasia.
3. Minor gingival bleeding may be treated by placing a moistened black tea bag on the bleeding mucosa.



FIGURE 6.60 ■ Gingival Hyperplasia. This patient presented to the ED complaining of recurrent gingival trauma and bleeding. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 6.61 ■ Gingival Hyperplasia. Examination of the oral cavity of the patient in [Fig. 6.60](#) reveals marked hyperplasia of the gingival tissues. (Photo contributor: R. Jason Thurman, MD.)

ORAL MALIGNANCIES

Clinical Summary

Oral cancers are malignant neoplasias that arise on the lip or within the oral cavity. The malignant lesion may originate as a primary lesion, as a metastasis from a distant organ, or by extension from a proximate site. Early symptoms include non-healing ulcers, indurated areas, erythematous or white patches, swelling or enlargement, discolorations, dental mobility, hoarseness, and oral bleeding. If the lesion goes undetected, the symptoms can progress to airway obstruction, paresthesias of the tongue, otalgia, trismus, lymphadenopathies, blurry vision, and persistent pain.

Management and Disposition

Detailed head and neck examination is fundamental for the diagnosis of the lesion. CT scan may be helpful to establish a tentative diagnosis, but biopsy of the area is necessary to confirm the diagnosis. An incisional biopsy can be

surgically performed with a #15 blade or biopsy punch and stored on 10% neutral buffered formalin medium for submission to pathology. A prompt referral to ENT or oral surgery is strongly recommended for further treatment. A combination of surgery, chemotherapy, and radiation is frequently used for definitive treatment.



FIGURE 6.62 ■ Squamous Cell Carcinoma of the Tongue. Squamous cell carcinoma on lateral border of the tongue; this is the most common site for intraoral cancers. (Photo contributor: Michael T. Brennan, DDS, MHS.)



FIGURE 6.63 ■ Squamous Cell Carcinoma of the Tongue. Patients may present with advanced lesions. Note the ulcerations on the superior aspect of this malignant tongue mass. (Photo Contributor: Lawrence B. Stack, MD.)



FIGURE 6.64 ■ Squamous Cell Carcinoma of the Palate. Ulcerated lesions involving the hard and soft palate consistent with squamous cell carcinoma. (Photo contributor: Michael T. Brennan, DDS, MHS.)



FIGURE 6.65 ■ Lingual Lymphoma. This patient had metastatic lymphoma, with unfortunate and extensive involvement of the tongue. (Photo contributor: R. Jason Thurman, MD.)

Pearls

1. Early diagnosis and treatment remain the key to improving survival of patients. Questionable lesions should be urgently referred for appropriate follow-up and definitive diagnosis.
2. CT scan and biopsy of the area are necessary for diagnosis but may generally be performed on an outpatient basis.

EXTRAVASCULAR (HEMORRHAGIC) ORAL LESIONS

Clinical Summary

Extravascular (hemorrhagic) oral lesions are purplish/red discoloration of the oral tissue due to the extravasation of blood. The lesions can be divided based on size into petechiae (1- to 2-mm pinpoint red areas), purpura (< 1 cm), and ecchymosis (> 1 cm). The lesions are usually the result of trauma but may also be a manifestation of platelet disorders (thrombocytopenia), leukemia, vascular

disorders (vasculitis), and coagulation disorders (scurvy, disseminated intravascular coagulation). The most common sites are the ones most susceptible to trauma, such as the tongue, buccal mucosa, and palate.

Management and Disposition

When the lesion is caused by trauma, the blood will slowly reabsorb over a period of a few days and no treatment will be necessary. Larger lesions might take weeks to resolve.

If there is no history or signs of trauma, further workup is needed. A complete blood count and peripheral blood smear along with coagulation studies may help rule out underlying systemic disease.

Pearls

1. Hemorrhagic oral lesions are negative to diascopy. Diascopy involves the application of pressure with a glass slide onto the surface of the lesion and examining the lesions through the glass for blanching; hemorrhagic oral lesions do not blanch.
2. In many cases, buccal ecchymosis is the first sign of thrombocytopenia.



FIGURE 6.66 ■ Buccal Ecchymosis. This patient had extensive buccal ecchymosis secondary to benign trauma from chewing. The patient had a platelet count of less than 5000 secondary to idiopathic

thrombocytopenic purpura. Note also the small petechiae on the soft palate. (Photo contributor: R. Jason Thurman, MD.)

METHAMPHETAMINE-INDUCED CARIES

Clinical Summary

Methamphetamine-induced caries (MIC) is hypothesized to be caused by the combination of mental and physiologic side effects of methamphetamine (MA) use such as xerostomia (dry mouth), gingival inflammation, poor oral hygiene, and frequent ingestion of high-sugar carbonated beverages (for relief of dry mouth). The appearance of MIC is characteristically smooth and dark in color; caries tend to start near the gingiva and can progress to the complete destruction of the coronal portion of the tooth (see also [Figs. 17.4](#) and [17.5](#)). Patients with MIC may not experience dental pain because MA can block the expected odontalgia. In addition, MA users tend to clench or grind teeth (bruxism), resulting in severe dental wear patterns, trismus, cracked teeth, disorders of the TMJ, and myofascial pain.

Management and Disposition

Dental or oral surgical consultation is recommended. The teeth are usually in such disrepair that extraction is required. If dental abscesses are present, incision and drainage in conjunction with antibiotic therapy are indicated.

Pearls

1. MIC resembles early childhood tooth decay (“baby bottle caries”).
2. Users of MA may experience xerostomia, gingival inflammation, sugar cravings, and poor oral hygiene that exacerbate the patient’s oral condition.
3. Patients with MIC may have an unexpectedly low amount of pain associated with the condition. As such, patients frequently present with severely advanced disease.



FIGURE 6.67 ■ Methamphetamine-Induced Caries. Rampant caries and heavy accumulation of dental plaque associated with methamphetamine use. (Photo contributor: J. Amadeo Valdez, DDS, MAS.)



FIGURE 6.68 ■ Methamphetamine-Induced Caries. Severe decay on the facial surface of the mandibular teeth and dental plaque accumulation in relation with methamphetamine use. (Photo contributor: J. Amadeo

ORAL CAVITY PIERCING COMPLICATIONS

Clinical Summary

Facial piercings are increasingly common. While external piercings, such as ear and eyebrow rings, are visibly apparent, intra-oral and extraoral piercing may not be readily visible on initial inspection. An awareness and suspicion for recent lip, labret, tongue, or other oral mucosal piercings should be performed in any trauma patient as they may cause intraoral damage, produce artifact and diminished CT scan quality if not removed, and interfere during intubation. Common nontraumatic complications of intraoral include peri-piercing cellulitis, abscess formation, hemorrhage, penetrating wounds, dental injury, and granuloma or keloid formation around a stud. Although infrequent, systemic infection may result. If the ball comes off the post, it may be swallowed or, rarely, aspirated. Playing with tongue rings may result in lingual surface tooth abrasion, erosion, chipping, fracture, and gingival resorption.

Management and Disposition

Piercing should be removed prior to any CT scan if possible. For localized hemorrhage after a recent piercing, direct pressure should be applied. If bleeding persists, consider removal of the piercing. Any infection surrounding a piercing warrants removal of the stud as it will serve as a nidus for continued infection. Chest radiographs are required to evaluate for stud aspiration. Advise patients with chipped teeth to remove the bar; these patients should be treated for the commensurate form of dental fracture.



FIGURE 6.69 ■ Tongue Piercing Cellulitis. This patient presented with fever and tongue pain surrounding his piercing site. Note the erythema and swelling of the tongue and sublingual areas extending from the piercing site (central bubble). (Photo contributor: David Effron, MD.)

Pearls

1. Remove piercings prior to cross-sectional imaging.
2. Piercings may act as a nidus for ongoing infection if not removed.
3. Ensure the prompt identification and removal of intraoral and extraoral piercings in any trauma patient potentially requiring emergent airway management, as the presence of jewelry can be a hindrance to successful intubation.



FIGURE 6.70 ■ Oral Piercing Complication. This patient suffered a painful injury when the stud of her lip ring came off, resulting in the penetration of her gingiva and inability to open her mouth. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 6.71 ■ Oral Piercing Complication. A close-up photograph of the oral piercing complication seen in [Fig. 6.70](#). (Photo contributor: Lawrence B. Stack, MD.)

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Chapter 7

CHEST AND ABDOMEN

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Seat Belt Injury. Abrasions from a 3-point restraint causing rib fractures and a pneumothorax. (Photo contributor: Brad Russell, MD.)

Chest and Abdominal Trauma

TRAUMATIC ASPHYXIA

Clinical Summary

The clinical findings of traumatic asphyxia are due to a sudden increase in intrathoracic pressure against a closed glottis. The elevated pressure is transmitted to the veins, venules, and capillaries of the head, neck, extremities, and upper torso, resulting in capillary rupture. Strangulation and hanging are common mechanisms. Survivors demonstrate plethora, ecchymoses, petechiae, and subconjunctival and retinal hemorrhages. Severe injuries may produce central nervous system injury with blindness, seizures, posturing, and paraplegia.



FIGURE 7.1 ■ Traumatic Asphyxia. This 45-year-old man was pinned when the truck he was working under fell on his chest. He was unable to breathe for 3 to 4 minutes until his coworkers rescued him. The violaceous coloration of the shoulders, face, and upper chest is apparent. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.2 ■ Traumatic Asphyxia. A closer view showing the petechial nature of this rash. The patient was observed in the hospital overnight and recovered completely. (Photo contributor: Stephen W. Corbett, MD.)

Management and Disposition

Treatment is supportive, with attention to other concurrent injuries. Long-term morbidity is related to the associated injuries.

Pearls

1. Facial petechiae are known as Tardieu spots.
2. One should be alert for associated rib and vertebral fractures.
3. Perthes syndrome is traumatic asphyxia following thoracic crush injury.



FIGURE 7.3 ■ Tardieu Spots. Facial petechiae, also known as Tardieu spots, are seen in this child after an accidental strangulation injury. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.4 ■ Subconjunctival Hemorrhages. Bilateral subconjunctival hemorrhages are seen after an intentional strangulation injury. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.5 ■ Traumatic Asphyxia. Marked facial petechiae and plethora after being pinned between two vehicles.

FLAIL CHEST

Clinical Summary

A flail chest occurs when segmental rib fractures allow a section of the thoracic cage to move independently. The negative inspiratory pressure created by the diaphragm is less effective since the flail segment paradoxically moves inward and interferes with ventilation. Pulmonary contusion, hemothorax,

pneumothorax, and great vessel injuries frequently accompany a flail chest.

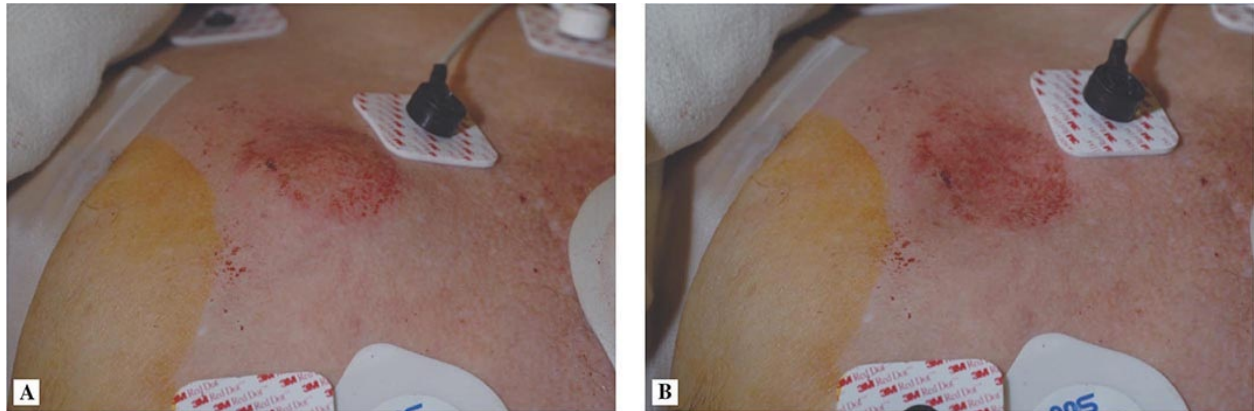


FIGURE 7.6 ■ Flail Chest. Localized blunt trauma to the left anterior chest 4 cm inferior to the midclavicle, with resultant flail segment. Positive intrathoracic pressure (**A**) and negative intrathoracic pressure (**B**) demonstrate the paradoxical movement of the flail segment. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Pain control and pulmonary hygiene are initial standard therapy. Mechanical ventilation is reserved for those with respiratory failure, not as a mechanism to stabilize the flail segment. Treatment of underlying pulmonary injuries and intensive care unit admission are required for these critically ill patients.

Pearls

1. Intercostal nerve blocks may help provide adequate analgesia so that pain does not prevent the patient from ventilating adequately.
2. Continuous positive airway pressure (CPAP) with patient-controlled analgesia may be as effective as mechanical ventilation in patients with a flail chest.
3. Paradoxical movement of the flail segment is seen during the respiratory cycle in flail chest.



FIGURE 7.7 ■ Flail Chest: Trauma Chest X-Ray. Fractures in two places of left ribs 3 and 4 result in a flail segment. Ribs 2 and 5 have single fractures. This is the chest x-ray of the patient in [Fig. 7.6](#). (Photo contributor: Lawrence B. Stack, MD.)

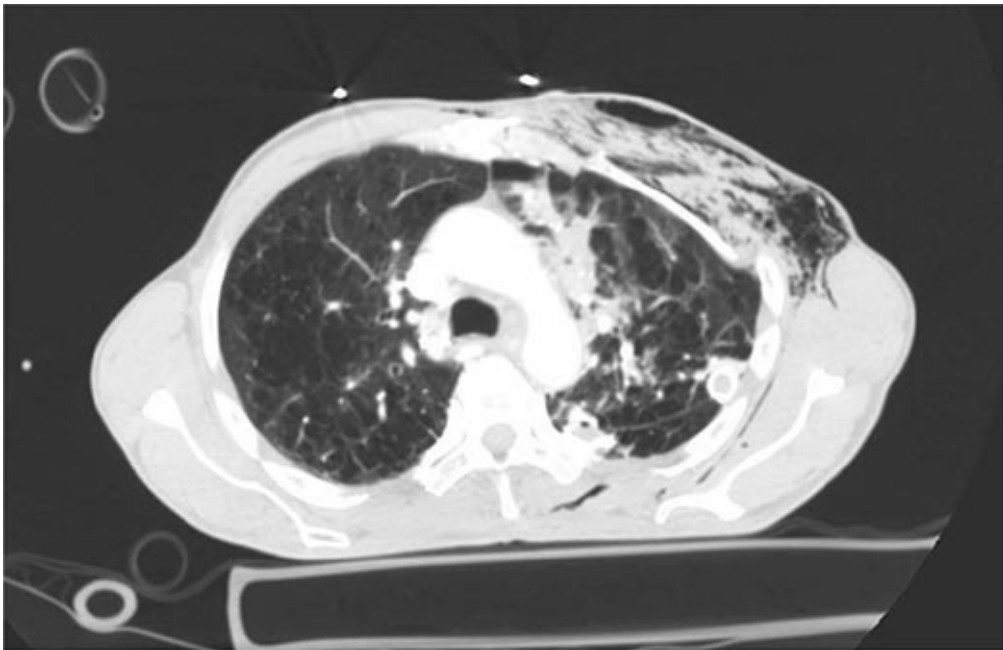


FIGURE 7.8 ■ Flail Chest: Chest Computed Tomography (CT) Scan. A flail segment is seen in the left anterior chest on an axial cut of this chest CT scan. A chest tube is seen in the left pleural space. Subcutaneous emphysema is seen in the area of the fractures and tracking posteriorly. This is the chest CT of the patient in [Fig. 7.6](#). (Photo contributor: Lawrence B. Stack, MD.)

LUNG HERNIATION

Clinical Summary

Lung herniation is a rare cause of chest pain and dyspnea in which the lung parenchyma protrudes through a defect in the thoracic wall. It typically occurs due to a preexisting weakness in the thoracic wall combined with an acute rise in intrathoracic pressure such as during a cough, sneeze, or Valsalva. Obesity, steroid use, prior injury, and surgery are risk factors for herniation. Symptoms include chest pain with cough, local pain, and swelling in the area of the herniation, which may vary in size during respirations. The deformity is typically reducible.

Management and Disposition

Diagnosis is confirmed with chest computed tomography (CT). Thoracic surgery consultation timing depends on the severity of symptoms or presence of strangulation, but most cases are nonemergent, and routine referral is adequate. Conservative treatment includes compressive pads, corsets, weight loss, and treatment of underlying lung disease.

Pearls

1. Pneumothorax is often associated with coughing with a closed glottis during deep inhalation (including marijuana).
2. Rupture of the intercostal musculature and lung herniation are lesser known complications of increased intrathoracic pressure.



FIGURE 7.9 ■ Lung Herniation. Chest wall deformity during inspiration in a patient with lung herniation after remote robotic coronary artery bypass grafting. (Photo contributor: Harry Stark, MD.)



FIGURE 7.10 ■ Lung Herniation. Chest wall deformity during expiration in a patient with lung herniation after remote robotic coronary artery bypass grafting. (Photo contributor: Harry Stark, MD.)



FIGURE 7.11 ■ Lung Herniation and Pneumothorax on CT of Chest. Chest wall defect and pneumothorax of the same patient. (Photo contributor: Harry Stark, MD.)



FIGURE 7.12 ■ Lung Herniation on CT of Chest. Spontaneous lung herniation after a vigorous cough while using marijuana in a different patient than above. Chest CT reveals lung herniation through a chest wall defect. (Photo contributor: Kevin Barlotta, MD.)

STERNOCLAVICULAR DISLOCATION

Clinical Summary

Dislocations of the sternoclavicular joint (SCJ) are uncommon due to the strength of the supporting ligaments. Anterior dislocations are nine times more frequent than posterior dislocations. Posterior dislocations are clinically more important due to the potential for injury to underlying structures. SCJ dislocations typically occur from motor vehicle crashes and sports injuries. Examination findings of an anterior SCJ dislocation include tenderness and prominence of the proximal clavicle from the sternum. The arm is often held in adduction. Posterior SCJ dislocations may be more difficult to identify. Pneumothorax, great vessel, tracheal, and esophageal injuries often accompany posterior SCJ dislocations. Pain and depression of the medial clavicle relative to the sternum may be seen. Superior and inferior dislocations may also be seen. CT scan through the SCJ is the best diagnostic study to evaluate this injury.

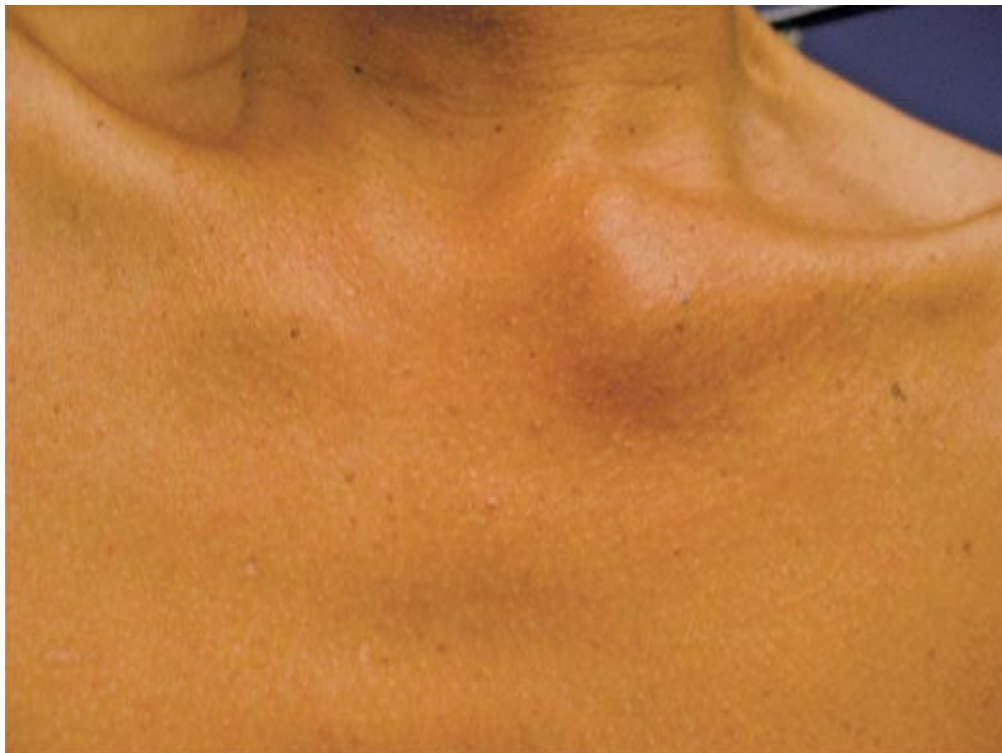


FIGURE 7.13 ■ Sternoclavicular Dislocation. A 43-year-old man complains of left sternoclavicular pain after direct trauma to the shoulder. Prominent proximal clavicle is seen prompting CT scan confirmation. (Photo contributor: R. Jason Thurman, MD.)

Management and Disposition

Orthopedic follow-up is required. Closed reduction of anterior SCJ dislocations is performed by placing the patient supine with a sandbag between their shoulders and placing downward pressure directly over the clavicle. A figure-of-eight clavicle harness is applied. Recurrence is common. Closed reduction of posterior SCJ dislocations may be attempted by an orthopedic surgeon by placing a towel clip around the medial clavicle and pulling the clavicle forward. Open reduction is often necessary.

Pearls

1. While it typically requires a tremendous force to cause an SCJ dislocation, spontaneous dislocations have been reported.
2. In the apical lordotic radiograph (serendipity view), the clavicular head will be higher than the unaffected side in anterior and lower than the unaffected side in posterior dislocations.
3. One-fourth of posterior SCJ dislocations result in mediastinal injury; they always require reduction.

CLAVICLE FRACTURE

Clinical Summary

Clavicle fractures are common and are classified by location into proximal, middle, and distal thirds. Most clavicle fractures occur in the middle third. Point tenderness and contusion typically overlie the fracture site. Deformity is often seen. Proximal fractures are least common and require significant force, and therefore, they may have underlying life-threatening mediastinal injuries. Displaced distal clavicle fractures suggest injury to the coracoclavicular and acromioclavicular (AC) ligaments.

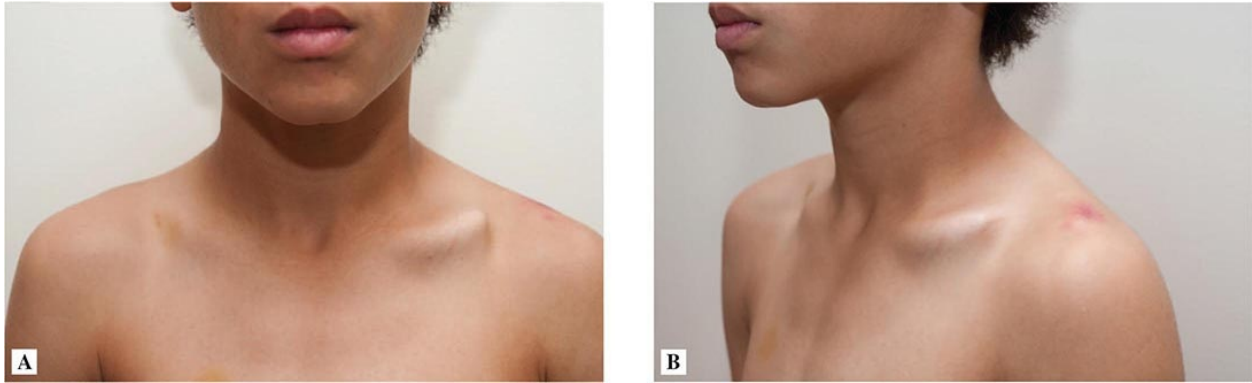


FIGURE 7.14 ■ Clavicle Fracture. This 12-year-old boy complains of pain, tenderness, and deformity over the mid left clavicle after an injury incurred during football drills. A “kick-stand” injury is seen on plain films (see [Fig. 7.15](#)). Fracture, though overriding, healed without reduction. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Most clavicle fractures are identified on an anteroposterior (AP) view of the shoulder. A 45-degree AP cephalad view may be helpful if AP films are normal and suspicion is high for fracture. A contrast-enhanced chest CT should be performed if a proximal fracture is identified, looking for mediastinal injuries. Nonoperative management using a simple sling, analgesics, and ice is adequate for most injuries. Orthopedic consultation is indicated for any medial fracture, tenting of the skin, fractures with 100% displacement, severely comminuted fractures, and displaced distal third fractures.

Pearls

1. Tenting requires closed reduction or surgical repair.
2. Stress radiographs (weighted views) help differentiate minimally or nondisplaced distal third fractures from an AC joint separation.
3. Glenoid neck fracture plus a clavicle fracture represents a “floating” shoulder and requires urgent orthopedic consultation.
4. Sixty percent of childhood clavicular fractures are nondisplaced.



FIGURE 7.15 ■ Clavicle Fracture Radiograph. Left midclavicle fracture with 100% displacement and overriding fragments. A “kick-stand” injury is seen. Fracture, though overriding, healed without complication. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.16 ■ Clavicle Fracture with Skin Tenting. A 30-year-old unrestrained woman in a motor vehicle crash (MVC) complains of left clavicle pain. Skin tenting is seen. Closed reduction was required to avoid skin ischemia. (Photo contributor: R. Jason Thurman, MD.)

TENSION PNEUMOTHORAX WITH NEEDLE THORACENTESIS

Clinical Summary

A tension pneumothorax results when air enters but does not exit the pleural space. Air in the pleural space accumulates and compresses the ipsilateral lung and vena cava, with a rapid decrease in cardiac output. The contralateral lung may suffer ventilation/perfusion mismatch. Subcutaneous air, tracheal deviation, jugulovenous distention (JVD), and diminished or hyperresonant ipsilateral breath sounds are clues. Subcutaneous emphysema may be visible on the neck and chest radiographs and is easily diagnosed by palpation. The released air from a tension pneumothorax can be heard escaping during a needle thoracostomy.

Management and Disposition

Treatment requires rapid recognition of the tension pneumothorax, frequently without benefit of chest radiographs. A 14-gauge or larger needle should be placed over the superior rib surface of the 2nd interspace in the midclavicular line. A rush of air with improvement of vital signs confirms the diagnosis. If there is no immediate improvement, do not hesitate to place a 2nd needle in the next interspace. A chest tube should be placed immediately.

Pearls

1. “Stacking” breaths in patients with obstructive lung disease trap air in the lungs and predispose to bleb rupture and pneumothorax. The pathophysiology of this disease requires a prolonged expiratory phase.
2. The diagnosis of a tension pneumothorax should be made clinically and treated immediately with needle or finger thoracostomy and tube thoracostomy.
3. Tension pneumothorax may be a consequence of positive-pressure ventilation in patients with undetected pneumothorax and a persistent air leak such as bronchopleural fistula.
4. Tension pneumothorax is a treatable cause of pulseless electrical activity

(PEA).



FIGURE 7.17 ■ Tension Pneumothorax. A 35-year-old man with severe asthma suffered respiratory arrest during transport by ambulance. He was intubated on arrival but soon became hard to ventilate and developed subcutaneous emphysema followed by hypotension. Needle thoracostomy produced a rush of air and bubbling from the needle with stabilization of vital signs. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.18 ■ Deep Sulcus Sign. Deepening of the left costophrenic angle is seen. Left to right mediastinal shift is also present suggesting a tension pneumothorax. (Photo contributor: Lawrence B. Stack, MD.)

CARDIAC TAMPONADE WITH PERICARDIOCENTESIS

Clinical Summary

Cardiac tamponade occurs when fluid or blood accumulates in the pericardium at a rate that does not allow adequate filling of the ventricles resulting in diminished cardiac output and shock. Symptoms include shortness of breath, orthopnea, dyspnea on exertion, syncope, and symptoms of inadequate perfusion. Hypotension, tachycardia, pulsus paradoxus, and JVD are

examination findings seen in tamponade. Causes include blunt or penetrating trauma, malignancy, hypothyroidism, uremia, myocardial or aortic rupture, and pericarditis. Rapid filling of the pericardial sac (50-100 mL) is more likely to cause cardiovascular compromise than gradual accumulation.

Management and Disposition

Cardiac tamponade should be suspected in patients at risk with shock. Point-of-care cardiac ultrasound (POCUS) provides rapid identification of tamponade physiology and should prompt pericardiocentesis if the patient is in shock. Electrocardiogram (ECG), chest x-ray, complete blood count (CBC), and cardiac injury markers are less specific clues for tamponade but are helpful for the identification of other causes of shock. CT of chest will identify cardiac tamponade but will take longer than POCUS to obtain.

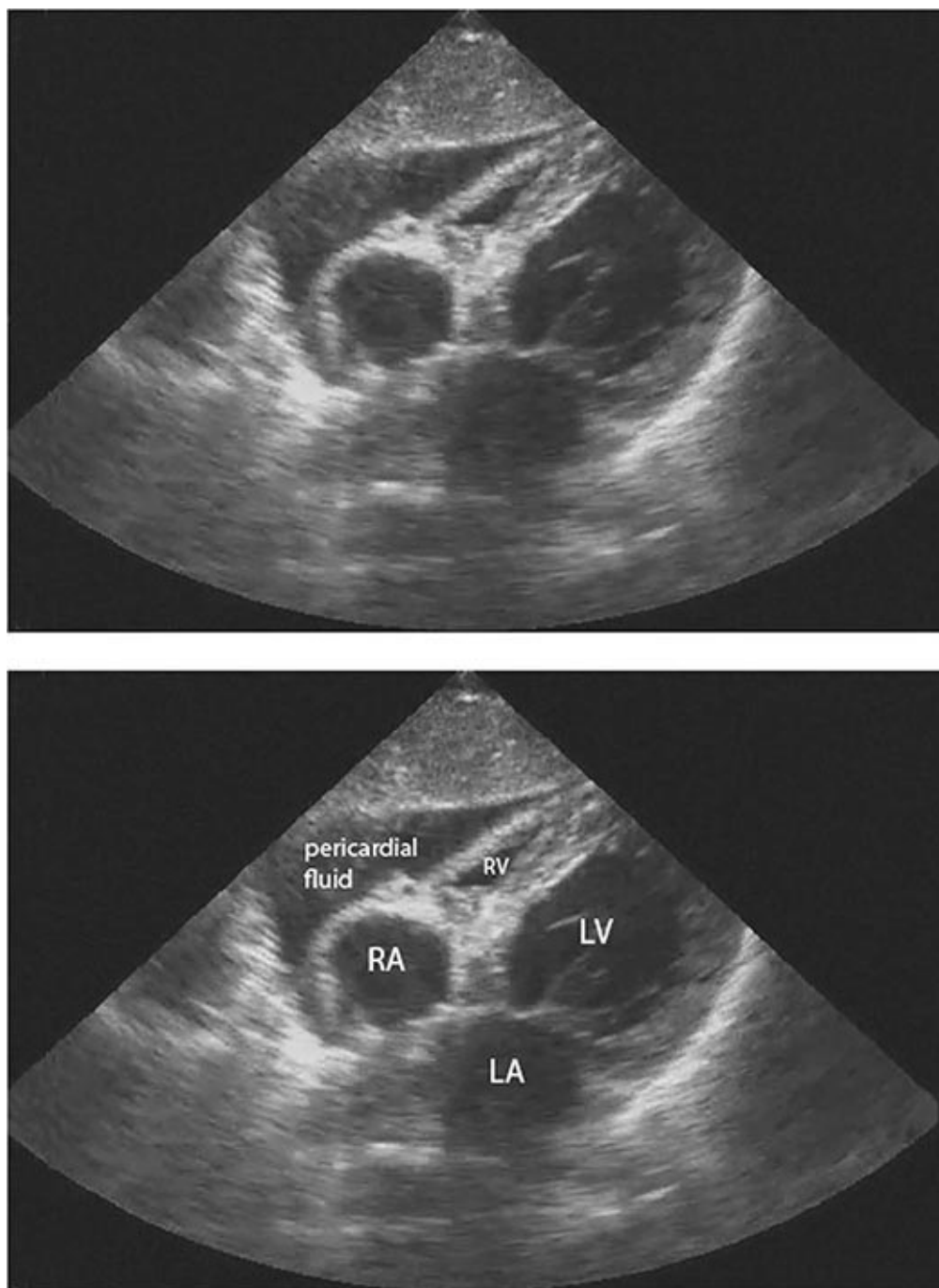


FIGURE 7.19 ■ Ultrasound of Cardiac Tamponade. This subxiphoid view of the heart reveals a large pericardial fluid collection and compression of the right ventricle (RV) suggesting tamponade physiology. (Photo contributor: Department of Emergency Medicine, Ultrasound Section, Vanderbilt University.)

Pearls

1. Electrical alternans seen on a 12-lead ECG suggests pericardial effusion.
2. Cardiac tamponade may cause a narrow pulse pressure, pulsus paradoxus (fall

of > 10 mm Hg in systolic blood pressure during inspiration), and low-voltage ECG.



FIGURE 7.20 ■ Emergency Department Pericardiocentesis. A positive pericardiocentesis in a patient with a sudden onset of shortness of breath and electrical alternans. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.21 ■ Electrical Alternans. Lead V4 demonstrates a beat-to-beat change in axis, diagnostic of electrical alternans, suggesting pericardial effusion. Low voltage may also be a clue for pericardial effusion. (Photo contributor: R. Jason Thurman, MD.)

3. Rapid ultrasonographic identification of pericardial fluid in patients with penetrating chest trauma may lead to lifesaving pericardiocentesis or thoracotomy.
4. Cardiac tamponade is a treatable cause of PEA.
5. Beck's triad of acute cardiac tamponade includes JVD from an elevated

central venous pressure, hypotension, and muffled heart sounds but is present only 10% to 40% of the time.

6. JVD in tamponade may not be seen in hypovolemic states such as trauma.
7. The simultaneous appearance of all three physical signs of Beck's triad is a late manifestation of tamponade and usually seen just prior to cardiac arrest.

EMERGENCY DEPARTMENT THORACOTOMY

Clinical Summary

Emergency department (ED) thoracotomy is a resuscitative procedure performed in patients with penetrating chest trauma who have lost signs of life in the presence of prehospital or ED personnel. Thoracotomy in the ED has specific goals once the chest is opened: relief of cardiac tamponade, support of cardiac function (internal cardiac compressions, cross-clamping the aorta to improve coronary perfusion, and internal defibrillation), and control of hemorrhage from the heart, pulmonary vessels, thoracic wall, and great vessels.



FIGURE 7.22 ■ Emergency Department Thoracotomy. An unsuccessful resuscitative emergency department thoracotomy with pericardiotomy in a patient with penetrating chest trauma who lost signs of life in the field after the paramedics arrived at the scene. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Patients with penetrating thoracic trauma who lose their vital signs on arrival or have unstable vital signs despite resuscitation should receive an immediate ED thoracotomy. Survival rates following ED thoracotomy for penetrating trauma are 9%; stab wounds fare much better than gunshot wounds. Patients with blunt trauma who lose their vital signs en route to the ED should not undergo an ED

thoracotomy, since they rarely survive. Surgical support should be obtained as soon as possible.

Pearls

1. Injuries most likely to be responsive to ED thoracotomy include cardiac tamponade, pulmonary parenchymal and tracheobronchial injuries, large-vessel injuries, air embolism, and penetrating heart injuries.
2. ED thoracotomy should be performed immediately once the indications have been met, since the likelihood of survival is greater when this is performed earlier in the resuscitation.
3. Stab wounds and measurable ED vital signs are the best predictors of survival.

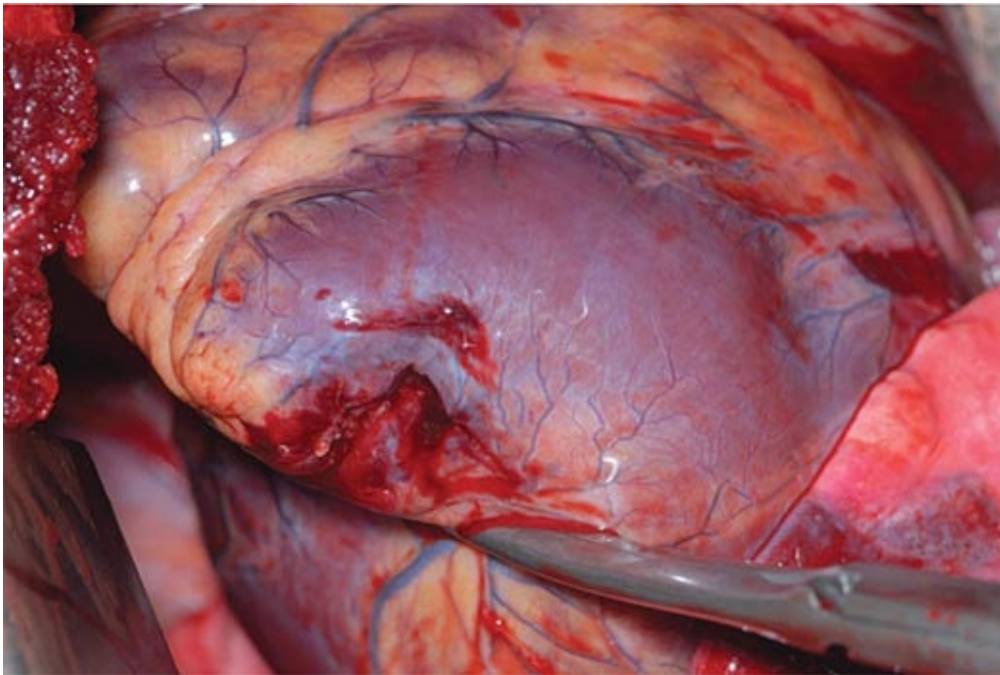


FIGURE 7.23 ■ Emergency Department Thoracotomy— Ventricular Ballistic Injury. Left ventricular ballistic injury seen after unsuccessful resuscitation and ED thoracotomy. (Photo contributor: Lawrence B. Stack, MD.)

SEAT BELT INJURY

Clinical Summary

Two- and three-point seat belt restraints have reduced mortality and the severity of trauma due to motor vehicle crashes; however, they occasionally produce injury. Lap/shoulder belts are known to produce abdominal (most common), thoracic, and spinal injuries. The “seat belt sign,” abrasions or ecchymoses to the neck, chest, and abdomen in the pattern of the belt, occurs in less than 20% of patients but is associated with a fourfold risk of intrathoracic and an eightfold risk of intra-abdominal injury.



FIGURE 7.24 ■ Seat Belt Injury. Ecchymosis from the lap belt is evident. A subtle Destot’s sign, which is inguinal or perineal ecchymosis, in a patient with a pelvis fracture. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 7.25 ■ Seat Belt Injury. Abrasions from a three-point restraint causing rib fractures and a pneumothorax. (Photo contributor: Brad Russell, MD.)

Management and Disposition

Patients with seat belt ecchymosis should undergo contrast-enhanced CT of the chest, abdomen, pelvis, and thoracic and lumbar spine due the increased probability of injury. Alternatively, hospital admission with serial examinations and laboratory studies (CBC, hepatic panel, lipase, urinalysis) is a reasonable management option.

Pearls

1. Up to 36% of patients with a seat belt sign have an abdominal injury requiring laparotomy.
2. Lap belt-only passengers are more likely to have a Chance fracture and small bowel injury if they have an abdominal seat belt abrasion after a motor vehicle crash.



FIGURE 7.26 ■ Seat Belt Injury. Neck abrasions from a three-point restraint in a patient involved in a head-on motor vehicle crash. (Photo contributor: David Effron, MD.)

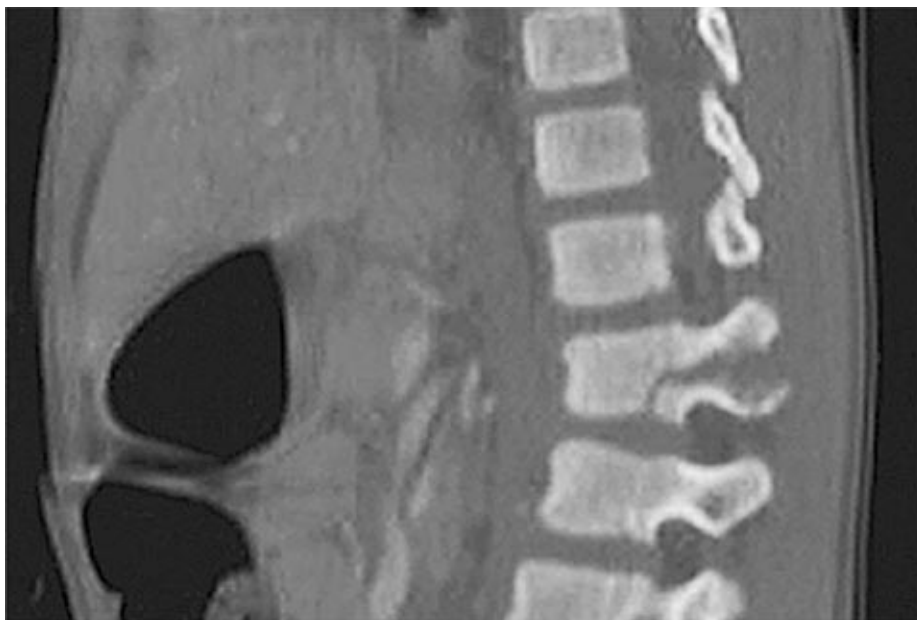


FIGURE 7.27 ■ Chance Fracture. T-12 Chance fracture in a restrained passenger involved in a head-on motor vehicle crash. (Photo contributor: Lawrence B. Stack, MD.)

GREY TURNER SIGN AND CULLEN SIGN

Clinical Summary

Bluish to purplish periumbilical discoloration (Cullen sign) and flank discoloration (Grey Turner sign) represent retroperitoneal hemorrhage that has dissected through fascial planes to the skin. Retroperitoneal blood may also extravasate into the perineum, causing a scrotal hematoma or inguinal mass. This hemorrhage may represent a hemodynamically significant bleed.



FIGURE 7.28 ■ Grey Turner and Cullen Signs. This patient displays both flank and periumbilical ecchymoses characteristic of Grey Turner and Cullen signs. (Photo contributor: Michael Ritter, MD.)

Cullen sign and Grey Turner sign are most frequently associated with hemorrhagic pancreatitis, are seen in 1% to 2% of cases, and typically are seen 2 to 3 days after onset. These signs may also be seen in ruptured ectopic pregnancy, severe trauma, leaking or ruptured abdominal aortic aneurysm, coagulopathy, or any other condition associated with bleeding into the retroperitoneum.

Management and Disposition

Initial treatment of hemodynamically unstable patients with Grey Turner sign or Cullen sign includes resuscitation with crystalloid and blood products.

Laboratory studies (CBC, amylase, lipase, human chorionic gonadotropin, prothrombin time [PT]/international normalized ratio [INR], blood type and crossmatch) and diagnostic imaging (POCUS and contrast-enhanced CT of the abdomen/pelvis) should occur simultaneously with resuscitation to identify the cause of the bleeding. Because of the severity of diseases associated with Grey Turner and Cullen signs, these patients are usually admitted to the hospital.

Pearls

1. Mortality rate in patients with Cullen or Grey Turner sign may be as high as 37%.
2. These signs are typically seen 2 to 3 days after the acute event.
3. These signs are seen in only 1% to 2% of patients with hemorrhagic pancreatitis.



FIGURE 7.29 ■ Grey Turner Sign. A 68-year-old man with flank ecchymosis due to hemorrhagic pancreatitis. (Photo contributor: Stephen W. Corbett, MD.)

Clinical Summary

Stab wounds cause injury to tissue in their path. Stab wounds to the chest, in addition to causing pneumothorax or hemothorax, may also cause life-threatening injuries to the heart and major blood vessels. One-quarter of anterior abdominal stab wounds do not penetrate the peritoneum. Half of those that do penetrate require no surgical intervention. For these reasons, local exploration, focused assessment with sonography for trauma (FAST), contrast-enhanced CT, and serial examinations are typical management strategies. Penetrating flank injuries are evaluated with contrast-enhanced CT. The size of the external wound frequently underestimates the internal injury. Impaled foreign bodies to the chest or abdomen pose a complex problem. The object inflicting the injury may also be preventing significant blood loss and therefore should be removed by the trauma surgeon in the operating room.

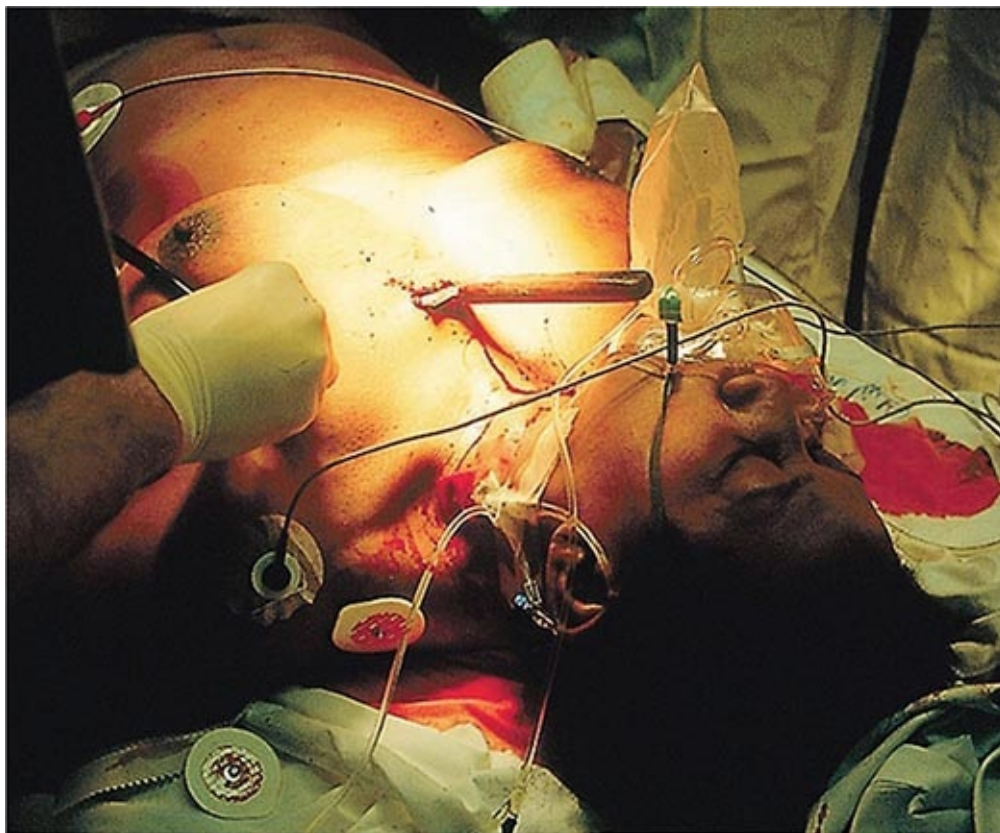


FIGURE 7.30 ■ Impaled Chest Wound. This patient was stabbed in the chest with a butcher knife in a family dispute. The knife was stabilized by EMS providers at the scene and removed in the operating room. Injury was isolated to the right atrium. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 7.31 ■ Impaled Abdominal Foreign Body. Impaled knife to the left abdomen after a domestic argument. (Photo contributor: Ian D. Jones, MD.)

Management and Disposition

Initial management of the unstable patient with an impaled object includes crystalloid, blood, and oxygen administration; airway stabilization; and cardiovascular monitoring. Laboratory evaluation includes blood type and crossmatching, CBC, lactate, comprehensive metabolic panel, PT/INR, and urinalysis. Trauma team mobilization is an important step in the initial management of penetrating chest or abdominal trauma. Stabilization of the impaled foreign object should be performed to prevent further injury.



FIGURE 7.32 ■ Impaled Nail in the Abdomen. Impaled 16-penny nail to the right abdomen after an accidental discharge from a pneumatic nail driver. The nail penetrated the peritoneal cavity but did not injure any organ. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.33 ■ Impaled Chest Foreign Body. Impaled car window frame to the left chest causing rib fractures and a pulmonary laceration. (Photo contributor: R. Jason Thurman, MD.)

Pearls

1. Impaled chest or abdominal foreign bodies should be removed only by a trauma surgeon in a controlled setting.
2. Seeing the size, shape, and trajectory of the impaled foreign body gives the trauma team clues to injured structures, which is another reason to leave the impaled object in place when possible.

ABDOMINAL EVISCERATION

Clinical Summary

Evisceration of abdominal contents usually occurs after a stab or slash wound to the abdomen or after recent laparotomy. It is an indication for laparotomy. Other indications for laparotomy in penetrating abdominal trauma include unexplained shock and evidence of blood in the stomach, bladder, or rectum.

Management and Disposition

Initial resuscitation includes crystalloid and blood administration, oxygen, airway security, trauma laboratory studies, and cardiovascular monitoring. Mobilization of the trauma team is important management of penetrating abdominal trauma. Eviscerated bowel should be covered with saline-soaked gauze. Antibiotics with enteric coverage should be administered.



FIGURE 7.34 ■ Abdominal Evisceration. Self-induced evisceration with bowel perforation and spillage of food particles is clearly seen in this photograph. This patient went directly to the operating room. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.35 ■ Abdominal Evisceration. Evisceration of small bowel after assault and stab wound to the

right lower abdomen. (Photo contributor: Frank Birinyi, MD.)

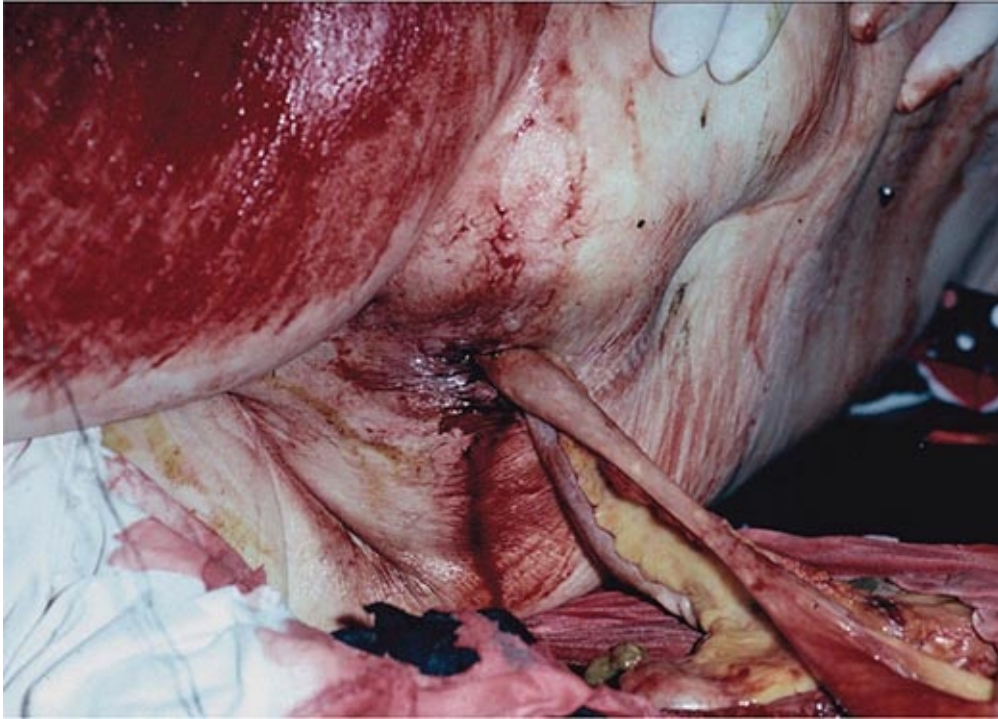


FIGURE 7.36 ■ Bowel Evisceration through the Anus. High-energy blunt abdominal trauma to this elderly man resulted in evisceration of small bowel and omentum through his anus. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. Evisceration, unexplained shock, or blood in the stomach, bladder, or rectum is an indication for laparotomy.
2. Selected patients with stab wounds to the abdomen and peritoneal penetration may be conservatively observed for delayed complications. Some centers are using a nonoperative approach for patients with gunshot wounds to the abdomen as well.
3. As many as 25% of patients with stab wounds to the abdomen can be discharged from the ED based on a negative wound exploration.
4. Evisceration should not distract the provider from systematic resuscitation priorities.

TRAUMATIC ABDOMINAL HERNIA

Clinical Summary

Blunt traumatic abdominal hernia is defined as herniation through disrupted musculature and fascia associated with adequate trauma, without skin penetration, and no evidence of a prior hernial defect at the site of injury. This occurs when a considerable blunt force is distributed over a surface area large enough to prevent skin penetration but small enough to cause a focal defect in the underlying fascia or muscle wall. Most of these injuries are due to seat belt injuries in motor vehicle crashes; handlebar injuries are the 2nd most common cause. Up to 44% of these patients require bowel resection.

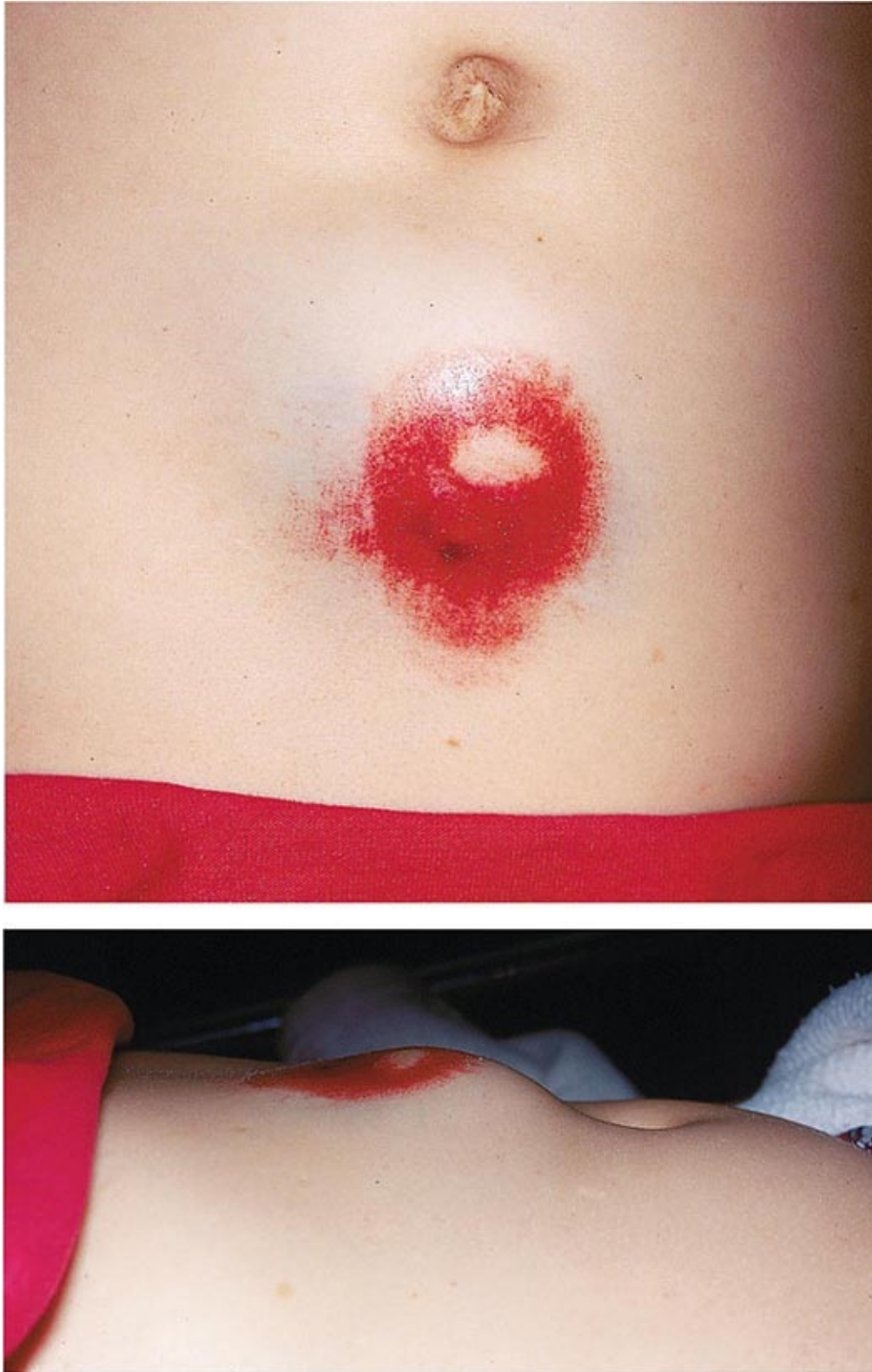


FIGURE 7.37 ■ Traumatic Abdominal Wall Hernia. This 5-year-old boy suffered a traumatic hernia from a handlebar injury. (Photo contributor: Lawrence B. Stack, MD.)

Contrast-enhanced CT of the abdomen and pelvis is the preferred diagnostic study for the evaluation of a traumatic abdominal hernia. Ultrasound may play a limited role in the diagnosis of abdominal wall hernia.

Management and Disposition

Identification and treatment of life-threatening associated injuries take priority over the hernia. The hernial defect should be repaired after the patient has been stabilized.

Pearls

1. Abdominal hernia due to blunt trauma is a rare injury, most frequently due to seat belt injuries in motor vehicle crashes.
2. Contrast-enhanced CT scan is the preferred diagnostic study for abdominal wall hernias.

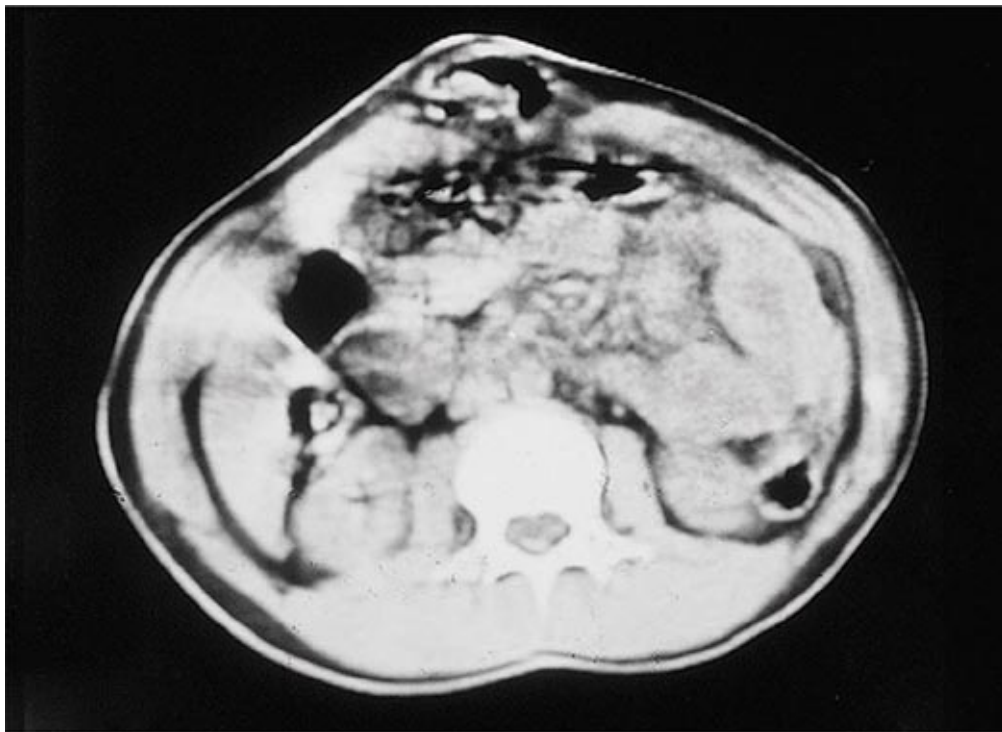


FIGURE 7.38 ■ CT Scan, Abdominal Wall Hernia. Abdominal contents are seen extruding through a fascial defect. (Photo contributor: Lawrence B. Stack, MD.)

PELVIC FRACTURE

Clinical Summary

Pelvic fractures are most often the result of motor vehicle crashes or falls and are fraught with complications. The pelvis should be regarded as a ring; identification of one fracture or dislocation should prompt surveillance for another. Trauma to the genitourinary (GU) tract is suggested by blood at the urethral meatus, a high-riding prostate, gross hematuria, or scrotal hematoma. Spinal nerves, the lumbosacral plexus, the sacral plexus, and the major lower extremity peripheral nerves, such as the sciatic, femoral, obturator, and pudendal nerves, are found in close proximity to the pelvis. A neurologic examination of the lower extremities should include a rectal examination to assess tone. The iliac arteries, veins, and their branches are also enveloped by the bony architecture of the pelvis, and severe hemorrhage is a common complication. While ecchymosis of the anterior abdominal wall, flank, sacral, or gluteal region suggests hemorrhage, there may be no outward signs of a severe hemorrhage. Blood found during rectal or vaginal examination may indicate a puncture wound from the fracture.

The overall mortality in patients with a pelvic fracture is 8%. Mortality rates are higher in men, elderly, and in patients who present in shock.

Management and Disposition

An AP x-ray of the pelvis may serve as a screening tool or a rapid confirmation of suspected major injury. Contrast-enhanced CT of the abdomen and pelvis will be required for known fractures to identify active bleeding and associated injuries. A retrograde urethrogram may also be necessary if a urethral injury is suspected. Angiography by interventional radiology with selective embolization may be performed to control arterial bleeding. In the face of a widened pubic symphysis or “open book” pelvic fracture and continued hemodynamic instability, orthopedic consultation for emergent external fixation can help to reduce blood vessel tension and reduce hemorrhage.

Pearls

1. A commercial pelvic binder or a sheet secured around the pelvis and extend

below the greater trochanters, may be used to temporarily stabilize pelvic fractures.

2. Posterior pelvic fractures are more likely to result in neurovascular injuries, while anterior pelvic fractures are more likely to cause urogenital injuries.
3. FAST sensitivity is 26% in detecting hemoperitoneum in patients with pelvic fractures. A negative FAST is therefore inadequate to eliminate the need for laparotomy in an unstable patient.



FIGURE 7.39 ■ Pelvic Fracture. Scrotal and perianal ecchymosis is seen in this patient with a vertical shear pelvic fracture due to a fall. (Photo contributor: Lawrence B. Stack, MD.)

Nontraumatic Chest and Abdominal Conditions

RESPIRATORY RETRACTIONS

Clinical Summary

Increased respiratory effort may be manifested by increased respiratory rate, increased chest wall excursion, and retractions of the less rigid structures of the thorax. Retractions of the sternum or suprasternal notch, intercostal retractions, and paradoxical abdominal movement reflect increased respiratory effort. This may be due to obstructive disease such as asthma or upper airway obstruction, pneumonia, or restrictive disease. The presence of stridor, wheezing, or rhonchi will help distinguish the cause.

Management and Disposition

An aggressive search for the cause of the retractions is required to direct therapy. Rapid evaluation of the airway for patency and breathing for oxygenation should be done immediately. High-flow oxygen by face mask is administered to patients in respiratory distress. Preparation for intubation is initiated for patients in severe distress or respiratory failure. Bilevel positive airway pressure and high-flow nasal canula therapy may buy time prior to intubation or prevent intubation. Arterial blood gas analysis may help identify impending respiratory failure. Routine measures for the mildly symptomatic patient depend on the cause of the retractions. For asthma or exacerbations of chronic obstructive pulmonary disease (COPD), nebulized β_2 agonists and steroid therapy may be appropriate. Patients with croup may require nebulized epinephrine or dexamethasone as initial therapy. Foreign-body aspiration requires imaging and consultation for confirmation of the suspected diagnosis and removal.

Pearls

1. Retractions are best observed with the patient at rest and the chest exposed.
2. Retractions from obstructive airway disease can be intercostal and supraclavicular and are usually accompanied by nasal flaring, increased expiratory phase, and increased respiratory rate.
3. Other causes of respiratory retractions include vocal cord paralysis, severe

metabolic acidosis as seen in diabetic ketoacidosis, and salicylate toxicity.

4. Most patients with airway or respiratory problems should be positioned for their comfort, not ours.



FIGURE 7.40 ■ Sternal Retractions. Sternal retractions in a patient with croup. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.41 ■ Suprasternal Retractions. Suprasternal retractions in an adolescent with severe asthma. (Photo contributor: Kevin J. Knoop, MD, MS.)

SUPERIOR VENA CAVA SYNDROME

Clinical Summary

Superior vena cava syndrome (SVCS) develops from obstruction of venous

drainage from the upper body, resulting in increased venous pressure, which leads to dilation of the collateral circulation. SVCS is most commonly caused by vascular compression from malignant mediastinal tumors. Dyspnea; swelling of the face, upper extremities, and trunk; chest pain; cough; or headache may be present. Physical findings include dilation of collateral veins of the trunk and upper extremities, facial edema and erythema (plethora), cyanosis, and tachypnea.



FIGURE 7.42 ■ Superior Vena Cava Syndrome. A 27-year-old man with SVCS. Note the prominent collateral veins of the chest and neck. (Photo contributor: William K. Mallon, MD.)



FIGURE 7.43 ■ Superior Vena Cava Syndrome. A middle-aged woman with plethora, distended superficial upper thorax veins, and headache with SVCS due to lymphoma.

Management and Disposition

Radiation therapy is the initial treatment for most malignant mediastinal tumors causing SVCS. An exception is small cell carcinoma, which responds better to chemotherapy. Elevating the head of the bed, oxygen, and administration of corticosteroids and diuretics initiated in the ED may provide temporary relief pending definitive therapy.

Pearls

1. SVCS is most commonly caused by malignant mediastinal tumors.
2. Treatment of most mediastinal tumors causing SVCS is radiation therapy.
3. Contrast-enhanced CT scan of the chest is the diagnostic study of choice for patients with SVCS.
4. Signs of decreased cardiac output, cerebral edema, and laryngeal edema are life-threatening findings in patients with SVCS.



FIGURE 7.44 ■ Facial Plethora. A 53-year-old woman presents with shortness of breath and facial plethora. She has SVCS due to lung cancer. (Photo contributor: R. Jason Thurman, MD.)

SUPRACLAVICULAR ADENOPATHY

Clinical Summary

Adenopathy or a mass in the supraclavicular fossa should heighten suspicion for metastatic or locally invasive disease. A Virchow node, also called a sentinel node (Troisier sign), is a left supraclavicular node in the area where the thoracic duct enters the superior vena cava (SVC). This node, located behind the sternocleidomastoid muscles, suggests metastatic abdominal cancer, particularly gastric cancer spread via lymphatics. Carcinoma affecting right supraclavicular nodes often arises from cancer of the breast or lung and is typically lateral to a Virchow node.



FIGURE 7.45 ■ Apical Lung Mass. This 68-year-old male cigarette smoker complained of cough and weight loss. A chest radiograph shows a left apical tumor. There is erosion of the tumor into the chest wall, with an indurated supraclavicular and infraclavicular mass. Moderate JVD is apparent, suggesting venous outflow obstruction. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.46 ■ Virchow Node. This middle-aged woman presents with multiple complaints including lateral neck swelling. She was diagnosed with abdominal lymphoma. (Photo contributor: David Effron, MD.)

A Pancoast tumor involves the apical lung and may affect contiguous structures such as the brachial plexus, sympathetic ganglion, vertebrae, ribs, SVC, and recurrent laryngeal nerve (more common for left-sided tumors). Horner syndrome, extremity edema, nerve deficits, hoarseness, and SVCS may result. Erosion of tumor through the chest wall can cause compression of venous outflow, with resultant JVD.

Management and Disposition

ED management focuses on identification of the cause of the mass or adenopathy. Contrast-enhanced CT of the neck, chest, abdomen, and pelvis should provide a clue to the cause. Unstable patients and those who lack support and resources should be admitted for continued workup.



FIGURE 7.47 ■ Left Supraclavicular Node. This 16-year-old patient developed left supraclavicular swelling and intermittent fever. She was diagnosed with lymphoma. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.48 ■ Right Supraclavicular Node. This 25-year-old patient developed right supraclavicular swelling and intermittent fever. She was diagnosed with lymphoma. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. Malignancies originating in the abdomen and pelvis are significantly more likely to metastasize to the left supraclavicular lymph nodes.
2. The primary site and types of malignancies that involve the left supraclavicular lymph nodes are different from those involved the right supraclavicular lymph nodes.

3. A Virchow node is made more evident if the patient performs a Valsalva.

JUGULOVENOUS DISTENSION

Clinical Summary

Central venous (right atrial) pressure is reflected by distention of the internal or external jugular veins. Distention greater than 4 cm above the sternal angle of Louis with the head of the bed elevated 30 to 60 degrees is abnormal. The presence of crackles, murmurs, rubs, percussed hyperresonance, or crepitus may help disclose the etiology.

The abdominojugular test, previously known as hepatojugular reflux, is performed by looking for JVD when placing pressure on the right upper quadrant. It is a marker for right ventricle dysfunction, constrictive pericarditis, cardiac tamponade, and tricuspid regurgitation.

Causes of JVD include right ventricular failure, left ventricular failure, biventricular failure, parenchymal lung disease, pulmonary hypertension, pulmonic stenosis, restrictive pericarditis, pericardial tamponade, SVC syndrome, pulmonary embolus, valvular disease, tension pneumothorax, increased circulating blood volume, and atrial myxoma. Temporary venous engorgement may result from Valsalva maneuver, positive-pressure ventilation, and Trendelenburg position.

Management and Disposition

Initial ED management focuses on determining the cause of the JVD as treatment varies depending on the cause. Preload reduction may help in cases of congestive heart failure. Tension pneumothorax requires a finger or needle thoracostomy and a chest tube. Pericardial tamponade requires a pericardiocentesis. Pulmonary embolism in unstable patients require thrombolytics or embolectomy. SVCS requires radiation therapy.

Pearls

1. Right-sided myocardial infarction may produce JVD with clear lung fields.
2. JVD may be absent in the presence of the above-listed causes if hypovolemia

is present.



FIGURE 7.49 ■ Jugulovenous Distention. An engorged external jugular vein is noted as it crosses the sternocleidomastoid muscle into the posterior triangle of the neck and disappears beneath the clavicle to join the brachiocephalic vein and the superior vena cava. This patient has severe congestive heart failure. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Veins of the abdomen normally are scarcely visible within the abdominal wall. Engorged veins, however, are often visible through the normal abdominal wall. Engorged veins forming a knot in the area of the umbilicus are described as caput medusae. The extent of associated findings depends on the underlying etiology. It is usually secondary to liver cirrhosis, with subsequent portal hypertension and development of circulation circumventing the liver.

Management and Disposition

Treatment is directed at the underlying cause. This finding by itself does not require acute treatment.

Pearls

1. Caput medusae have the same clinical significance as the more common patterns of venous engorgement.
2. If pressure on a prominent abdominal wall vein results in flow of blood to the head, the likely cause is inferior vena cava obstruction. If the flow is to the feet, it is caput medusa.



FIGURE 7.50 ■ Caput Medusae. This elderly female with alcoholic cirrhosis has engorged abdominal veins in the knotted appearance consistent with caput medusae. (Photo contributor: Gary Schwartz, MD.)

ABDOMINAL HERNIAS

Clinical Summary

A hernia is a defect in the abdominal wall allowing intra-abdominal contents to protrude outside the abdominal cavity. Most abdominal wall hernias occur at the groin and umbilicus. Incarceration is defined as the inability to reduce the protruding tissue to its normal position. Strangulation occurs when the blood supply of the hernia's contents is obstructed and tissue necrosis ensues. An *incisional* hernia may manifest clinically as a mass or palpable defect adjacent to

a surgical incision and can be reproduced by having the patient perform the Valsalva maneuver. Peristomal hernias are a type of incisional hernia where the abdominal wall opening is too large, allowing intra-abdominal contents to accumulate outside the abdominal cavity. Obesity and wound infection, which interfere with wound healing, predispose to the formation of incisional hernias. The defect of an *indirect* inguinal hernia is the internal (abdominal) inguinal ring and may be manifest in either sex by a bulge over the midpoint of the inguinal ligament that increases in size with Valsalva maneuver. A fingertip placed into the external ring through the inguinal canal may palpate the defect. A *direct* hernia may be manifested by a bulge midway adjacent to the pubic tubercle and may be felt by the pad of the finger placed in the inguinal canal. The defect is in the posterior wall of the inguinal canal. Direct inguinal hernias are usually painless and occur in males. Femoral canal hernias are more common in women and are prone to both strangulation and incarceration.

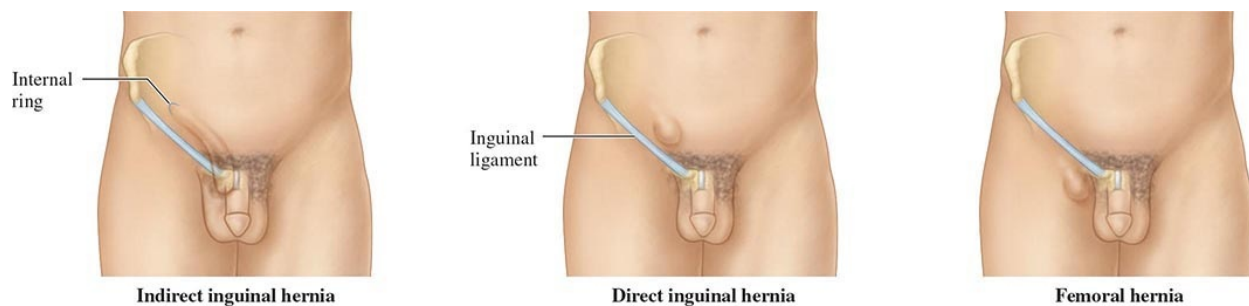


FIGURE 7.51 ■ Hernia Types. Drawings of the different hernia types using the landmarks of the inguinal ligament and pubic tubercle.



FIGURE 7.52A ■ Incisional Hernia. An asymptomatic incisional hernia in an obese male that developed after coronary artery bypass graft. The CT in [Fig. 7.52B](#) demonstrates a loop of bowel protruding through the abdominal wall defect. (Photo contributor: Lawrence B. Stack, MD.)

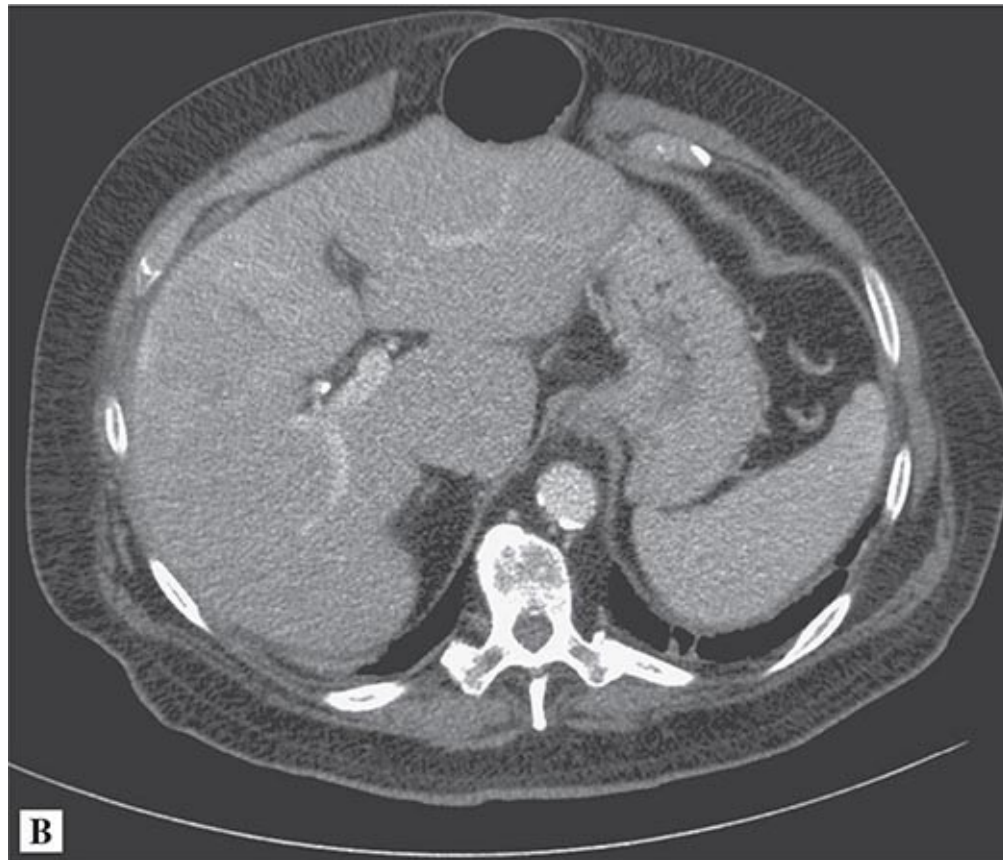


FIGURE 7.52B ■ Incisional Hernia CT Scan.



FIGURE 7.53 ■ Indirect Inguinal Hernia. A left indirect inguinal hernia in a female patient. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.54 ■ Direct Inguinal Hernia. A direct inguinal hernia. Note the bulge adjacent to the left pubic tubercle. (Photo contributor: Lawrence B. Stack, MD.)

Nausea and vomiting may be present if incarceration with bowel obstruction occurs. Strangulation can lead to fever, peritonitis, and sepsis.

Management and Disposition

When patients present with strangulation (fever, leukocytosis, elevated lactate, systemic signs of toxicity), surgery should be consulted for operative reduction. In the absence of these signs, ED reduction should be attempted and is facilitated with systemic analgesia, muscle relaxation and placing the patient in the Trendelenburg position, and applying a cold pack to the hernia. Routine consultation for operative repair is indicated in asymptomatic patients with reducible hernias.

Pearls

1. Acutely strangulated or incarcerated hernias require immediate surgical evaluation.
2. Direct inguinal hernias are usually painless.
3. Treatment of concomitant exacerbating conditions (cough, constipation, vomiting) prevent recurrences.



FIGURE 7.55 ■ Peristomal Hernia with Abscess. Peristomal pain, induration, and fever prompted a CT, which reveals a peristomal hernia and cutaneous abscess. (Photo contributor: Lawrence B. Stack, MD.)

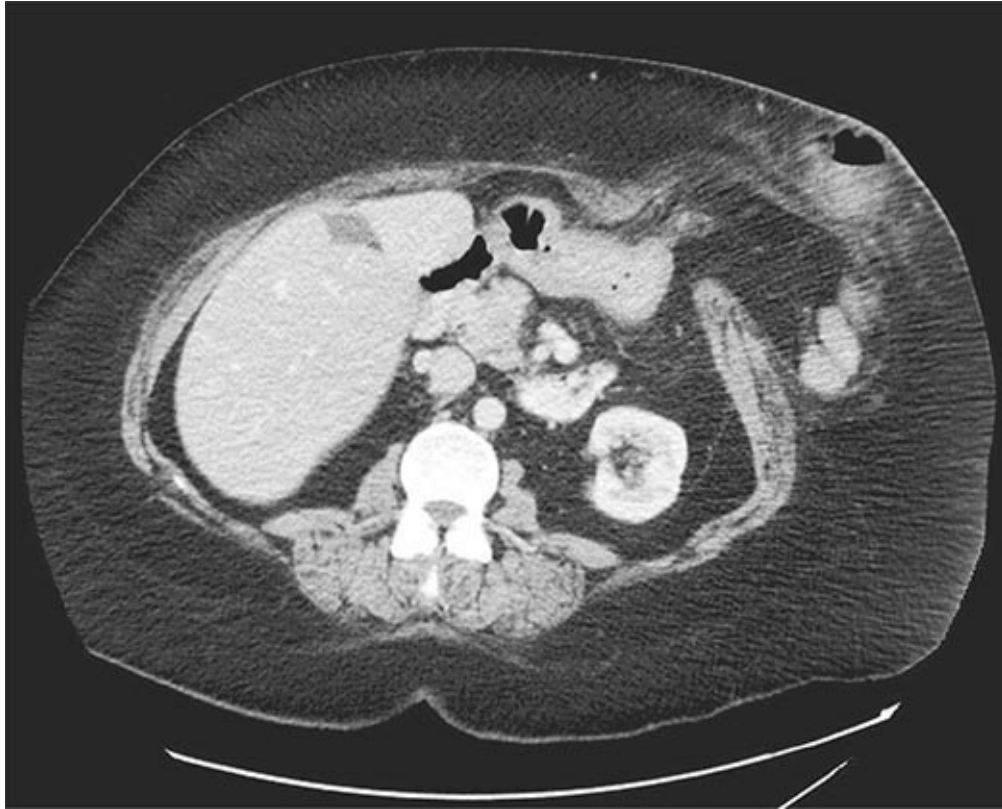


FIGURE 7.56 ■ Peristomal Hernia with Abscess—CT. Peristomal pain, induration, and fever prompted a CT, which reveals a peristomal hernia and cutaneous abscess. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.57 ■ Indirect Inguinal Hernia—Incarcerated. Left inguinal hernia with colon incarcerated in the inguinal canal. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.58 ■ Indirect Inguinal Hernia—Incarcerated CT. Contrast-enhanced CT demonstrates bowel within the inguinal canal. Enhancement of the bowel wall and fat stranding suggest early strangulation. (Photo contributor: Lawrence B. Stack, MD.)

UMBILICAL HERNIA

Clinical Summary

The umbilicus is a common site of abdominal hernias. Predisposing conditions in adults include ascites and prior abdominal surgery. The size of the defect determines the symptomatology and incidence of incarceration, with smaller defects resulting in more pronounced symptoms and an increased incidence of incarceration. Pain is located in the area of the fascial defect. Contents of the hernia may be palpable and tender. Symptoms of obstruction (nausea, vomiting, and abdominal distention) may be present. If the hernia becomes strangulated, erythema of the overlying skin with fever and hypotension may occur.

Flood syndrome is a rare complication of chronic and recurrent ascites. It is named after the sudden rush of fluid that accompanies spontaneous rupture of an umbilical hernia. Complications include cellulitis, peritonitis, evisceration, and sepsis. Ulceration or necrosis of an umbilical hernia signals impending rupture.

Large-volume paracentesis can precipitate incarceration or strangulation in patients with ascites and an umbilical hernia.

Management and Disposition

Reduction is attempted in the stable patient without clinical evidence of strangulation. Treatment of any predisposing conditions (eg, abdominal paracentesis in the patient with tense ascites) may cause spontaneous reduction and avoid progression of the hernia to strangulation. Routine consultation for elective repair is indicated in asymptomatic patients with reducible hernias.



FIGURE 7.59 ■ Strangulated Umbilical Hernia. The skin overlying a strangulated umbilical hernia is erythematous and tender. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Umbilical hernias in children usually resolve without treatment.
2. Umbilical hernias in adults usually become worse and require elective repair.
3. Eighty percent of flood syndrome is preceded by umbilical hernia necrosis.



FIGURE 7.60A ■ Umbilical Hernia. A 53-year-old man with umbilical pain and swelling. (Photo contributor: Lawrence B. Stack, MD.)

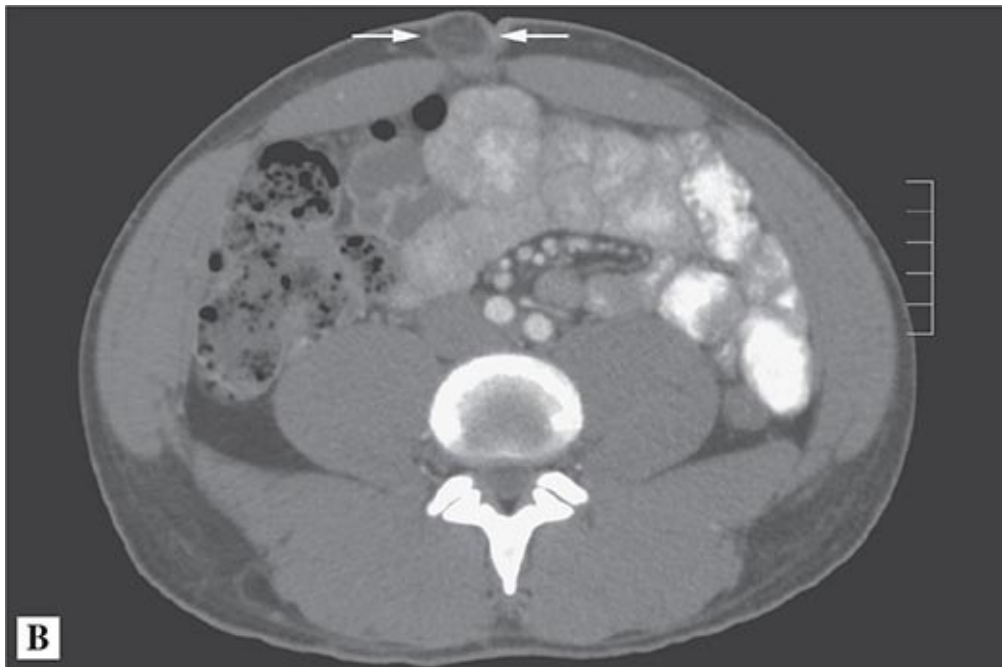


FIGURE 7.60B ■ Umbilical Hernia CT Scan. CT reveals omentum in the umbilical hernia defect (arrows). (Photo Contributor: Lawrence B. Stack, MD.)



FIGURE 7.61 ■ Flood Syndrome. Patient with umbilical hernia and ascites with fluid leaking from the stump. This photograph was taken shortly after a large gush of fluid came from the opening in the stump. (Photo contributor: Thomas E. Davis, MD.)



FIGURE 7.62 ■ Umbilical Hernia with Incarceration. Large-volume paracentesis has induced this incarcerated umbilical hernia. Hernia reduction during early phases of paracentesis may prevent incarceration. (Photo contributor: Emily A. Long, MD.)



FIGURE 7.63 ■ Umbilical Hernia with Incarceration—CT. Axial and sagittal CT cuts demonstrating an umbilical hernia in a patient with ascites. (Photo contributor: Emily A. Long, MD.)

PATENT URACHAL DUCT

Clinical Summary

When the vestigial urachal duct is not obliterated during development, drainage can occur from the bladder to the umbilicus. Urachal cysts may also be present, detectable as a painful mass between the umbilicus and pubis when they become infected or by ultrasound. Rarely, adenocarcinoma may form in these remnants.

Management and Disposition

Acute treatment is usually not required unless an infection is evident. Routine urologic consultation for surgical revision is indicated. A retrograde study with radiopaque dye will outline the patent duct.

Pearl

1. This finding should prompt a careful search for other urogenital anomalies.



FIGURE 7.64 ■ Patent Urachal Duct. This 19-year-old man presented to the emergency department with clear fluid (urine) draining from the umbilicus, suggestive of a patent urachal duct. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 7.65 ■ Urachal Cyst. Umbilical pain and discoloration suggested an umbilical mass or

inflammation, prompting a CT scan that revealed a urachal cyst. (Photo contributor: Martin D. Klinkhammer, MD, MPH.)

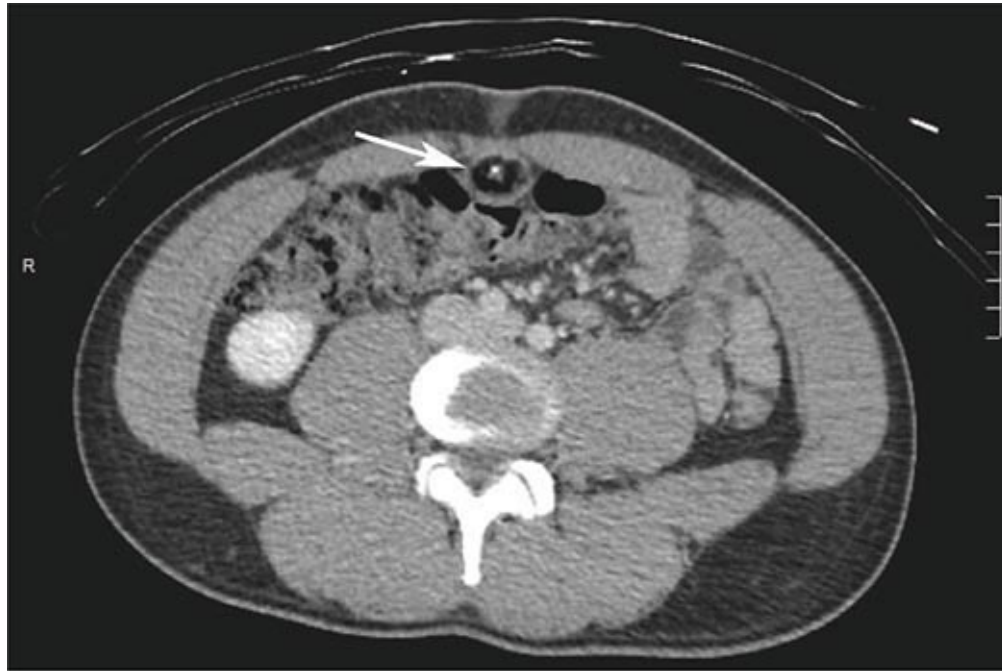


FIGURE 7.66 ■ Urachal Cyst—CT. Urachal cyst confirmed by contrast-enhanced CT scan of the abdomen/pelvis. (Photo contributor: Martin D. Klinkhammer, MD, MPH.)

SISTER MARY JOSEPH NODE (NODULAR UMBILICUS)

Clinical Summary

A Sister Mary Joseph node is a metastasis manifesting as a periumbilical nodule secondary to abdominal or pelvic cancers (one-half are gastrointestinal, one-quarter are gynecologic). Cancers of the colon may cause pain, change in bowel habits, anemia, and obstruction. In general, left-sided cancers cause obstruction, whereas right-sided tumors may have significant metastases before they create signs and symptoms. These metastases typically involve peritoneal and omental spread with distant metastases to the liver. Spread to the umbilicus is colloquially known as the Sister Mary Joseph node.

Prognosis is poor if cancer is the cause (survival time of 10 months).

Management and Disposition

Prompt referral for staging and treatment of the tumor is indicated. Other signs and symptoms (from obstruction, blood loss, malnutrition, and pain) should be addressed and treated.

Pearls

1. Virchow node, presenting as a supraclavicular mass, also heralds bowel carcinoma.
2. A Sister Mary Joseph node is commonly due to gastric carcinoma.
3. All umbilical masses require evaluation with contrast-enhanced CT scan of the abdomen/pelvis.



FIGURE 7.67 ■ Sister Mary Joseph Node. Sister Mary Joseph nodule of a patient with ovarian cancer. (Photo Contributor: Stephen W. Corbett, MD.)



FIGURE 7.68 ■ Sister Mary Joseph Node. This 63-year-old woman presents with abdominal swelling and ascites. She was diagnosed with ovarian cancer. Axial CT scan of the abdomen at the level of the umbilicus demonstrates ascitic fluid and the umbilical nodularity (arrows). (Photo contributor: R. Jason Thurman, MD.)



FIGURE 7.69 ■ Nodular Umbilicus. Firm umbilical nodule and abdominal distension due to a germ cell tumor. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.70 ■ Nodular Umbilicus—CT. Contrast-enhanced CT of the abdomen/pelvis reveals an umbilical nodule and large mass, which is a germ cell tumor. (Photo contributor: Lawrence B. Stack, MD.)

ABDOMINAL DISTENSION

Clinical Summary

Abdominal distention may be a symptom—described by the patient as the feeling of being bloated—or a sign, a protuberance of the patient’s abdomen. Obesity, ascites, pregnancy, neoplasms, aneurysm, tympanites (excess gas), organomegaly, urinary retention, bowel obstruction, hemoperitoneum, and constipation are important etiologies to consider in the differential.

In obesity, the abdomen is uniformly rounded, while an increase in girth and fat concurrently accumulates in other parts of the body.

In patients with ascites, there may be shifting dullness, a fluid wave, bulging flanks, or hepatomegaly. The profile of the fluid-filled abdomen of ascites is a single curve from the xiphoid process to the pubic symphysis. The umbilicus may be everted, and there may be prominent superficial abdominal veins.

In patients with neoplasms, there may be a palpable mass.

In gravid patients, fetal heart tones may be present and fetal motion may be

felt. The pregnant abdomen profile shows the outward curve to be more prominent in the lower half of the abdomen. The umbilicus may be everted in the last trimester of pregnancy. Prominent abdominal wall veins may also be seen.



FIGURE 7.71 ■ Ascites. Ascites in a male with alcoholic cirrhosis. Note the everted umbilicus and prominent superficial abdominal veins. (Photo contributor: Lawrence B. Stack, MD.)

In patients with excess gas from bowel obstruction, there may be absent or high-pitched bowel sounds and absence of bowel movements or flatus. Excess abdominal air can be located in the lumen of the stomach or intestines or free in

the peritoneum. This abdominal profile is a single curve from the xiphoid process to the pubic symphysis. Nausea, vomiting, decreased bowel sounds, and colicky pain are present in a small bowel obstruction. Large bowel obstruction may be accompanied by feculent vomiting and absent production of flatus.

The abdominal profile of a patient with a leaking abdominal aortic aneurysm shows a mottled abdominal wall reflective of hypoperfusion of this structure. There may be a curve of the midabdomen to either side of the aorta, more often on the left. Palpation of a pulsatile mass supports the diagnosis. Ultrasound or CT of the abdomen will confirm the diagnosis.

Management and Disposition

ED management is directed at determining the etiology. Point of care ultrasound (POCUS) may rapidly identify an abdominal aortic aneurysm, pregnancy, bowel obstruction, bladder distension, free fluid, ascites, and masses. Contrast-enhanced CT scan of the abdomen and pelvis will give more detail on these conditions than POCUS. Life-threatening causes (aneurysm, obstruction, neoplasms, hemoperitoneum) require resuscitation and consultation for definitive treatment.



FIGURE 7.72A ■ Ascites with Paracentesis. Midline approach to a paracentesis in the patient with ascites. (Photo contributor: Lawrence B. Stack, MD.)

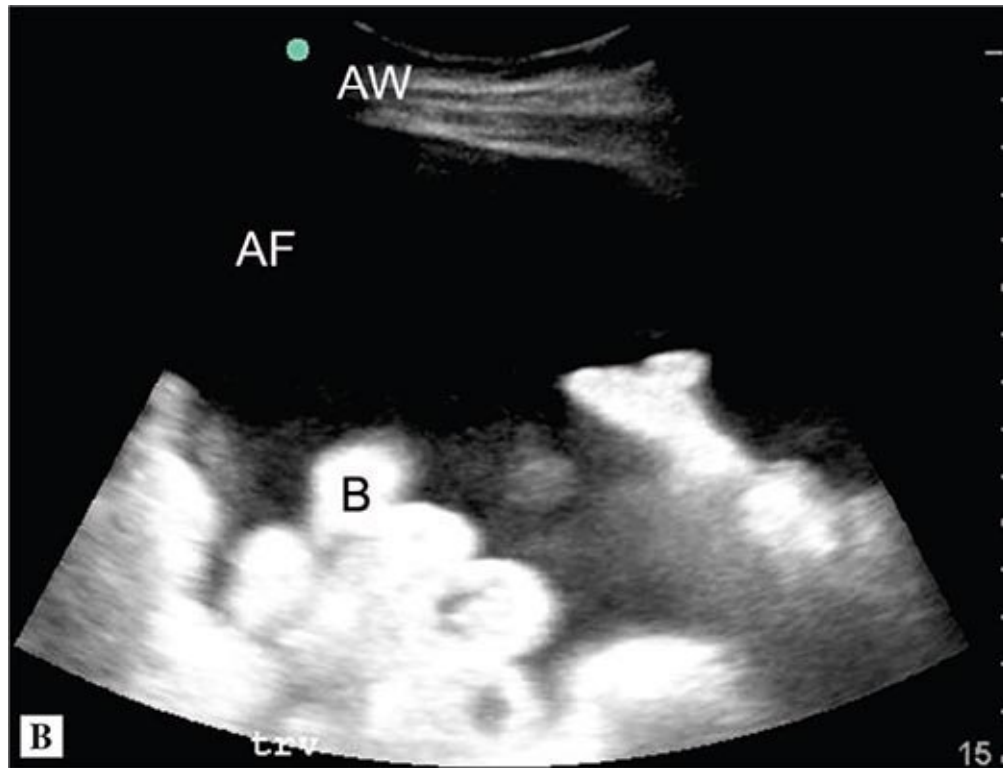


FIGURE 7.72B ■ Ascites on Ultrasound. Ascitic fluid (AF) seen between the abdominal wall (AW) and bowel (B) on this bedside ultrasound. (Photo contributor: Jeremy Simpson Boyd, MD.)

Pearls

1. The “six f ’s” can categorize conditions causing abdominal distention: fat, flatus, fetus, fluid, feces, and fatal growth.
2. POCUS measuring transrectal diameter (> 3.8 cm as cutoff) performed better than KUB (kidney, ureter, bladder x-ray) for diagnosing constipation.



FIGURE 7.73 ■ Abdominal Aortic Aneurysm. (A) The abdomen of a patient with a leaking abdominal aortic aneurysm. Note the mottled abdominal wall and the prominent curvature of the right side of the abdomen. (Photo contributor: Stephen W. Corbett, MD.) (B) Eight-centimeter abdominal aortic aneurysm seen in cross-section on an ultrasound in different patient. (Photo contributor: Jeremy Simpson Boyd, MD.)



FIGURE 7.74 ■ Gravid Abdomen. The abdomen of a woman at 39 weeks' gestation. Note the abdominal wall striae, everted umbilicus, and prominent superficial abdominal wall veins. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.75 ■ Pseudoobstruction. An 85-year-old woman was brought from a nursing home with a complaint of abdominal distention and pain for 1 to 2 days. An eventual diagnosis of Ogilvie syndrome, or pseudoobstruction of the large bowel, was made. This is usually seen in debilitated patients and can be treated with decompression. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.76 ■ Constipation. An 11-year-old boy complains of abdominal pain and abdominal distention. Stool is palpated throughout the abdomen. Flat and upright plain films of the abdomen reveal a large stool burden. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.77 ■ Urinary retention. Localized abdominal distension due to urinary retention from recreational drug use. One liter of urine was drained from this patient's bladder. (Photo contributor: Lawrence B. Stack, MD.)

ABDOMINAL WALL HEMATOMA

Clinical Summary

Mild trauma may produce hematomas of the rectus sheath. This injury results in intense abdominal pain, which can mimic an acute abdomen. The diagnosis is difficult by physical exam since most hematomas are in the posterior rectus sheath and are not palpable or visible. Palpation of the abdominal wall may reveal a tender mass that is accentuated by contraction of the rectus. Ultrasound and CT can confirm the diagnosis.



FIGURE 7.78 ■ Abdominal Wall Hematoma. This 50-year-old man with chronic obstructive pulmonary disease developed right lower quadrant pain after an episode of coughing. A repeat examination on the second visit showed clearly visible ecchymosis. There was no coagulopathy, and amylase was normal. A CT scan revealed a 10- by 8-cm hematoma in the right rectus abdominis sheath. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.79 ■ Rectus Abdominis Hematoma. Periumbilical ecchymoses after a coughing episode while on apixaban resulting in rectus abdominis hematoma. (Photo contributor: Lawrence B. Stack, MD.)

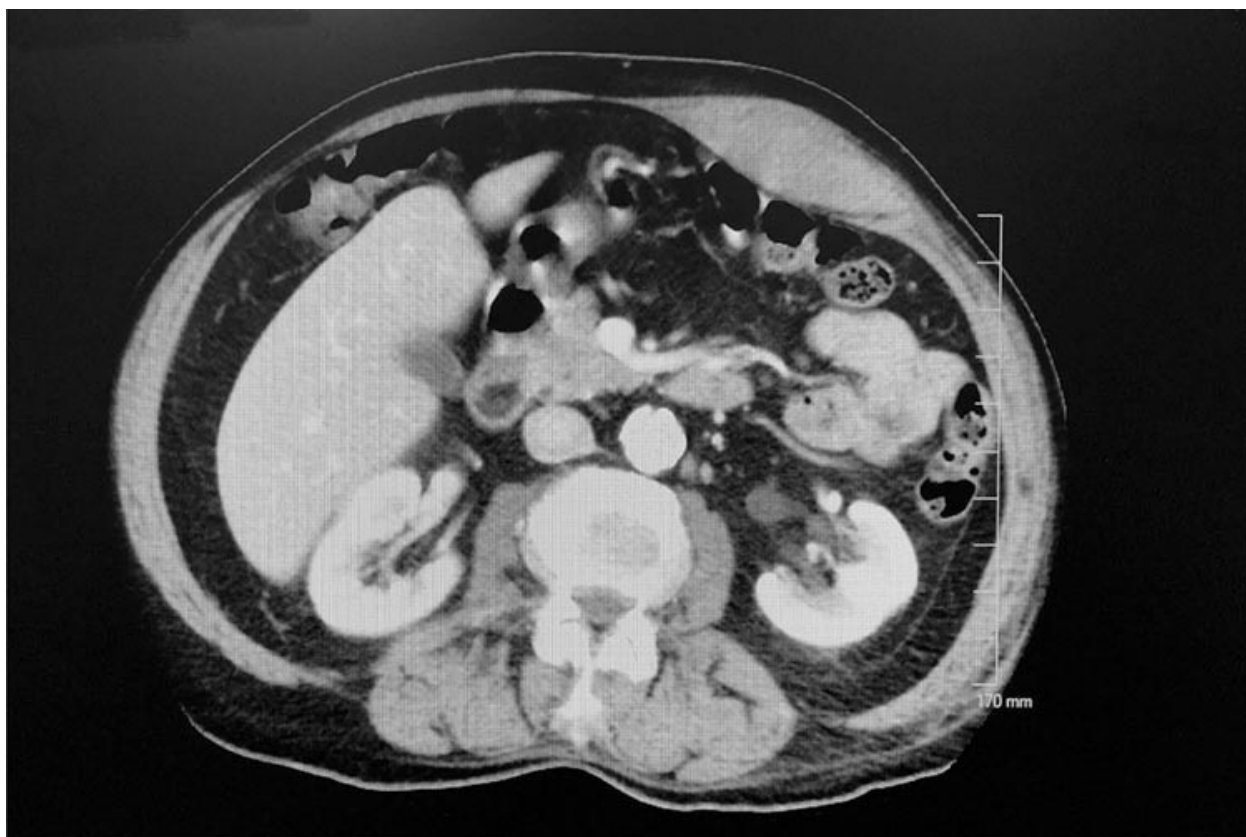


FIGURE 7.80 ■ Rectus Abdominis Hematoma—CT. Axial slice of contrast-enhanced CT in a patient with a left rectus abdominis hematoma. (Photo contributor: Lawrence B. Stack, MD.)

Major traumatic force, especially when belted in a vehicle crash, may result in a massive abdominal wall hematoma that requires intervention. Active extravasation, demonstrated by a contrast blush within the hematoma, suggests active bleeding within the hematoma.

Management and Disposition

Assuming that there is no underlying blood dyscrasia or coagulopathy, hematomas of the rectus sheath usually resolve in 1 to 2 weeks. Expanding abdominal wall hematomas with active extravasation of blood may require embolization by interventional radiology.

Pearls

1. Fothergill sign is enhancement of a rectus sheath hematoma when the abdominal wall is tensed. The mass should not cross the midline and should be easier to palpate with abdominal muscle contractions. Intra-abdominal

masses are more difficult to palpate with such contractions.

2. Carnett sign is also assessed after the abdominal wall muscles are tensed; decreased pain suggests an intra-abdominal source, whereas increased pain suggests an abdominal wall source.



FIGURE 7.81 ■ Abdominal Wall Hematoma. A 60-year-old woman, who was a restrained passenger in a motor vehicle crash, has an expanding abdominal wall hematoma. Contrasted CT scan of the abdomen (Figure 7.82) reveals contrast extravasation into the hematoma. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.82 ■ CT of Abdominal Wall Hematoma. Contrast-enhanced CT of the patient in [Fig. 7.81](#) demonstrates active extravasation of contrast (arrow) into the hematoma. Embolization of the hematoma ceased the hemorrhage. (Photo contributor: Lawrence B. Stack, MD.)

PECTUS DEFORMITIES

Clinical Summary

Pectus excavatum is a common developmental deformity of the anterior chest wall present in 0.25% of births. The cause is not well understood and may be related to intrauterine growth defects or muscle and connective tissue abnormalities within the thorax. In many cases, there is a family history of the condition.

The defect is a concavity of the anterior chest wall. Although the condition is occasionally associated with Marfan, scoliosis, and congenital heart disease, most patients are asymptomatic. In some, there is slightly decreased exercise tolerance, felt to be the effect of the consequence of the increased work of breathing caused by the chest wall mechanics.

Pectus carinatum is a chest wall deformity where the sternum protrudes forward and may involve the mid and lower sternum (chondrogladiolar prominence: 95%) or upper sternum (chondromanubrial prominence: 5%). It is an uncommon deformity and becomes evident in late childhood and worsens during adolescence. Most patients are asymptomatic, and repair is most often for cosmesis.

Management and Disposition

No definite treatment is needed, although cosmetic repairs of the anterior chest wall are sometimes recommended.

Pearls

1. Congenital heart disease and Marfan syndrome are present in 1.5% of patients with pectus excavatum.
2. Pectus excavatum may create a radiologic artifact that appears like a right middle lobe opacity.

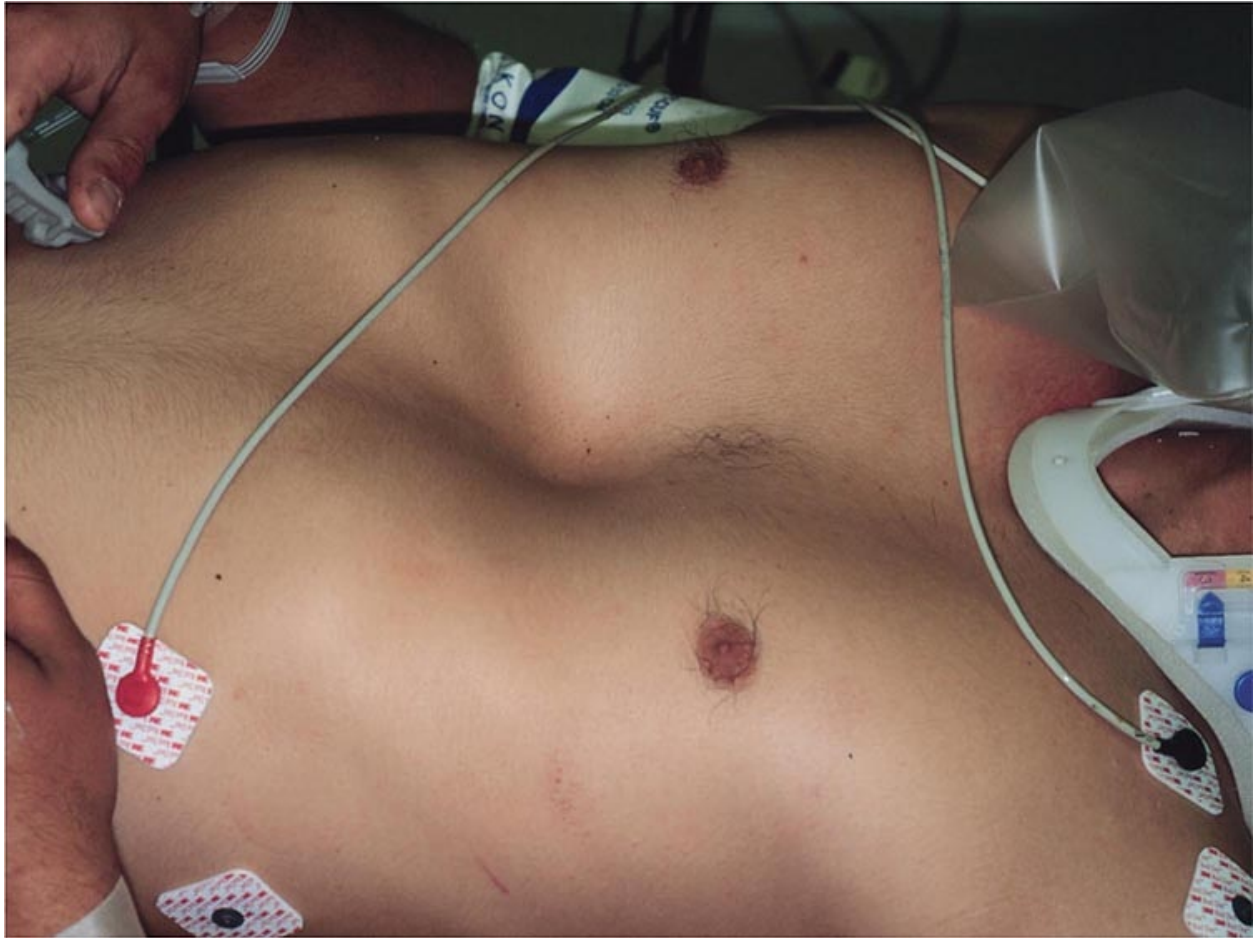


FIGURE 7.83 ■ Pectus Excavatum. A 26-year-old man presents to the emergency department with multisystem trauma after a motor vehicle crash. A longstanding pectus deformity is seen here. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.84 ■ Pectus Carinatum. A 14-year-old boy with sore throat was found to have pectus carinatum deformity of the chondrogladiolar prominence type. He had no chest complaint. (Photo contributor: Lawrence B. Stack, MD.)

STOMA PROLAPSE

Clinical Summary

When a stoma prolapses, bowel telescopes out on itself, lengthening the stoma. A stoma of the ileum may prolapse in 3% to 11% of patients over a lifetime. Causes of stomal prolapse include stomal construction difficulties, abdominal wall abnormalities such as obesity, increased intra-abdominal pressure, and weak abdominal musculature. Clinical findings of stomal prolapse include increase in size and length of the stoma; edema of the mucosa; and bleeding; and if ischemic, it may be dusky, cyanotic, or purple in color.

Management and Disposition

Emergent surgical consultation should occur in patients with a gangrenous stoma or if the prolapse is not reducible. To reduce a prolapsed stoma, have the patient lie supine or in slight Trendelenburg to decrease intra-abdominal pressure. Apply continuous gentle pressure on the prolapsed stomal tissues, into the abdominal cavity. If the bowel is edematous, a cold compress or osmotic therapy using table sugar applied for 15 minutes before reduction attempt may reduce the edema. If stomal prolapse reduction is successful, general surgery follow-up should be arranged as soon as possible.

Pearls

1. Oral or intravenous diazepam may help facilitate prolapse reduction.
2. Your hospital stoma nurse may be a great resource to assist with management of stoma complications.
3. Application of table sugar to the prolapsed ileum may facilitate reduction by reducing mucosal edema.



FIGURE 7.85 ■ Prolapsed Stoma. A 32-year-old man with inflammatory bowel disease and ileostomy presents with a prolapsed stoma. Note the bleeding, edema, and dusky appearance of the ileal mucosa. (Photo contributor: Lawrence B. Stack, MD.)

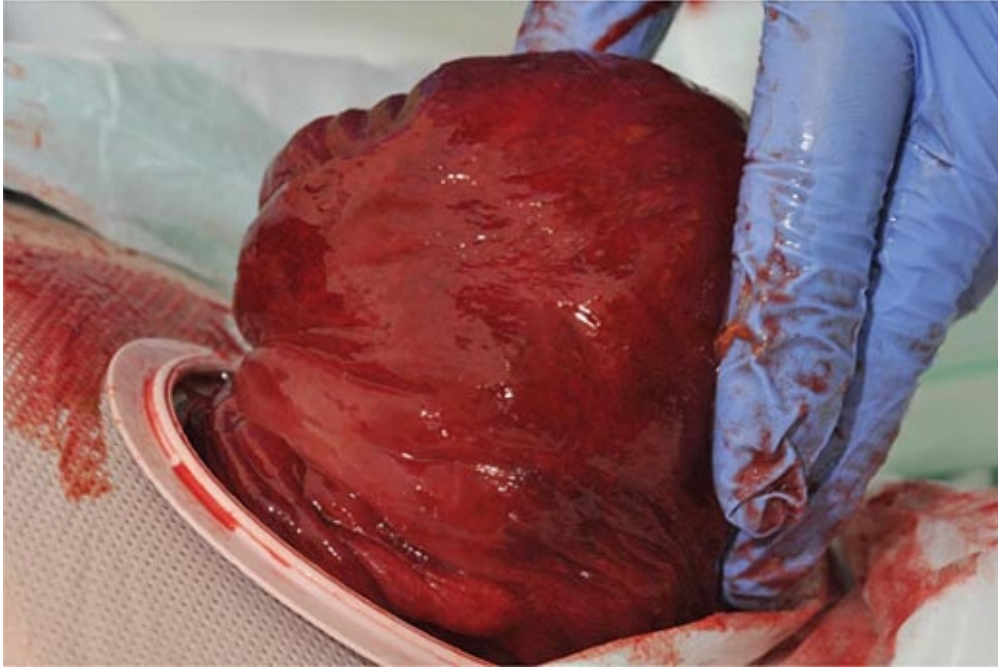


FIGURE 7.86 ■ Prolapsed Stoma. A 48-year-old man with pain and bleeding from stoma that was placed after a partial colectomy for complex diverticulitis. The bowel appears dusky and is oozing blood. (Photo contributor: Nakul Shekhawat, MD, MPH.)

PRUNE BELLY (EAGLE-BARRETT) SYNDROME

Clinical Summary

Prune belly syndrome (PBS) is a congenital disorder characterized by a partial or complete lack of abdominal musculature, severe urinary tract abnormalities, and bilateral cryptorchidism. The syndrome is typically recognized at birth or by ultrasound in utero. Severe renal dysplasia resulting in end-stage renal disease (ESRD) occurs in 50% of patients. ESRD and its complications are the most common reason a patient with PBS comes to the ED. Patients with PBS are at higher risk for recurrent urinary tract infection (UTI), chronic constipation, splenic torsion, and midgut and cecal volvulus.

Management and Disposition

Patients with PBS will most commonly frequent the ED because of complications of ESRD and their hemodialysis, continuous ambulatory peritoneal dialysis, or transplant. Because patients with PBS are at higher risk for

splenic torsion and midgut or cecal volvulus, CT scan should be performed if sudden onset of severe abdominal pain occurs in these patients. Frequent UTIs and chronic constipation may prompt ED visits.

Pearls

1. About one-half of patients progress to ESRD.
2. Chronic constipation is a common symptom in patients with PBS.
3. Gastrointestinal abnormalities are seen in 30% of patients with PBS.

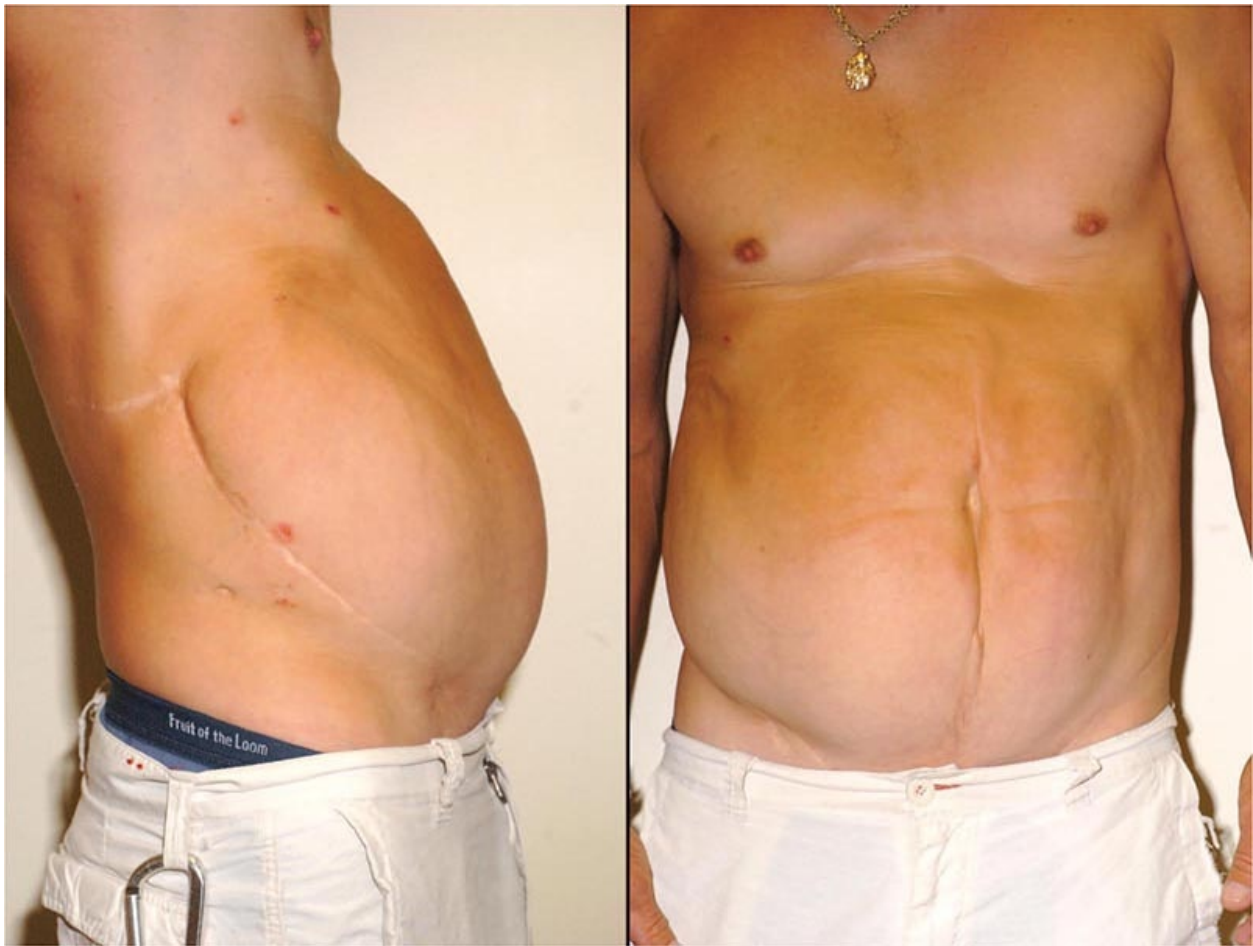


FIGURE 7.87 ■ Prune Belly Syndrome. A 44-year-old man with PBS, status post renal transplant, comes to the ED for abdominal pain and was ultimately diagnosed with constipation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.88 ■ Prune Belly Syndrome. A 2-year-old with PBS on hemodialysis comes to the ED for fever. Patient was bacteremic due to a dialysis catheter infection. (Photo contributor: Lawrence B. Stack, MD.)

STERNOCLAVICULAR JOINT SEPTIC ARTHRITIS

Clinical Summary

Septic arthritis is an acute infection of a joint, most commonly bacterial from

hematogenous seeding as a complication of bacteremia. It is potentially life-threatening and has a mortality rate approaching 10%. An acutely warm, tender, and swollen joint with restricted movement due to pain should be considered septic until proved otherwise. Sternoclavicular joint (SCJ) septic arthritis is a rare process associated with intravenous drug abuse, diabetes, rheumatoid arthritis, and immunosuppressed individuals. *Staphylococcus aureus* is the most common cause.

Management and Disposition



FIGURE 7.89A ■ Sternoclavicular Joint Septic Arthritis. Fever, erythema, tenderness, and fluctuance over the right sternoclavicular joint in a patient with intravenous drug use. Contrast-enhanced chest CT confirms fluid in the joint with overlying abscess. (Photo contributor: Kevin Barlotta, MD.)

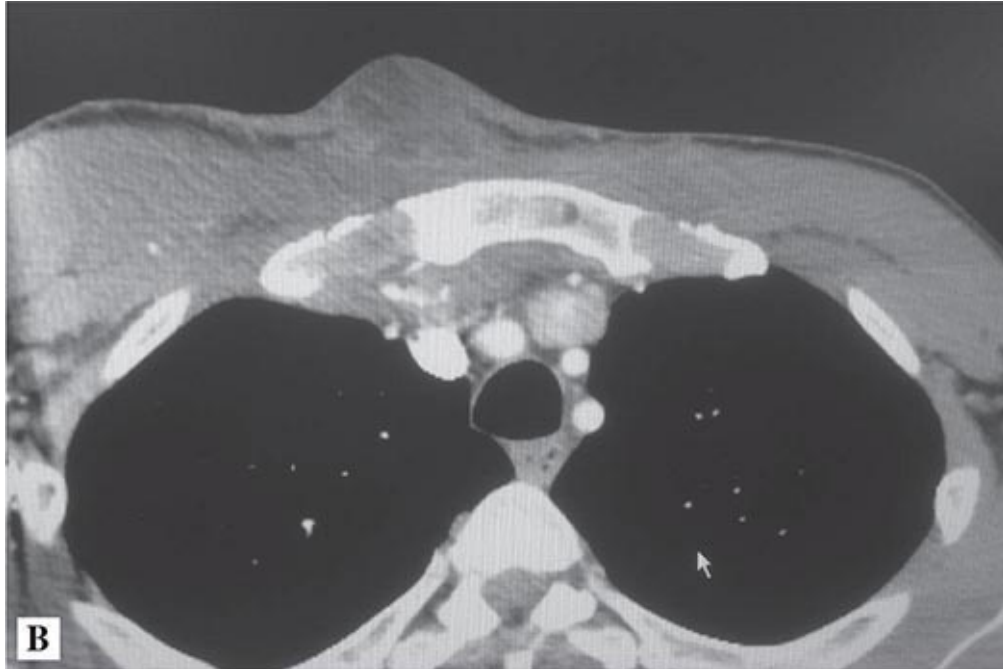


FIGURE 7.89B ■ Sternoclavicular Joint Septic Arthritis-CT. Axial cut of a contrast-enhanced chest CT in patient 7.89A reveals a right SCJ septic arthritis with abscess formation. (Photo contributor: Kevin Barlotta, MD.)



FIGURE 7.90A ■ Sternoclavicular Joint Septic Arthritis. Elderly male with septic arthritis of the right SCJ joint as a remote complication of coronary artery bypass graft surgery. (Photo Contributor: Lawrence B. Stack, MD.)

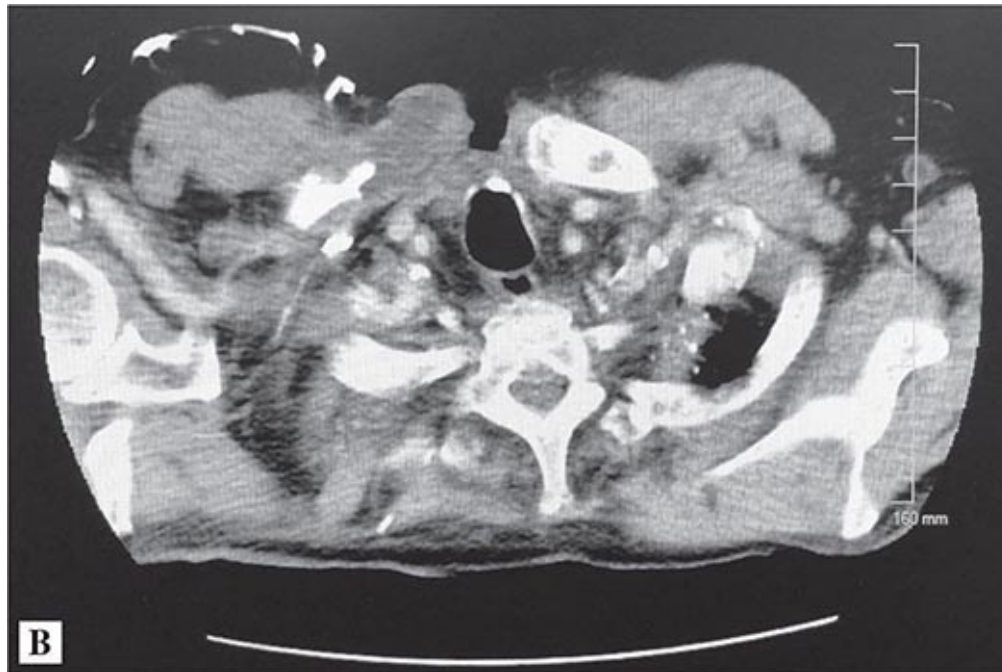


FIGURE 7.90B ■ Sternoclavicular Joint Septic Arthritis. Axial contrast-enhanced CT through the sternoclavicular joint of the patient in Figure 7.90A revealing swelling and abscess formation of the right sternoclavicular joint. (Photo Contributor: Lawrence B. Stack, MD.)

Contrast-enhanced CT scan of the chest may demonstrate if the infection extends beyond the joint. Arthrocentesis is necessary to confirm the diagnosis. Synovial fluid should be sent for cell count and differential, culture and sensitivity, crystals, and Gram stain. Patients with septic arthritis should be admitted for surgical debridement and intravenous antibiotics.

Pearls

1. Sternoclavicular septic arthritis is associated with intravenous drug abuse.
2. Early surgical debridement should follow initiation of appropriate antibiotics.
3. Complications of septic SCJ include mediastinitis and SVCS.

BREAST MASS

Clinical Summary

Female breast cancer is the leading cause of cancer-related mortality of women in the world. Patients seek care at all stages of disease from early (painless

immobile breast lump, bloody nipple discharge, and mammography abnormality) to late (axillary lymphadenopathy, thickening of the breast skin [peau d'orange appearance], necrotic mass, and metastases to bone, liver, or lungs).

Management and Disposition

ED management of a breast mass suspicious for cancer depends on patient stability, level of function, severity of pain, and social and medical resources. Patients with suspected or obvious advanced disease who are reluctant to seek care should be hospitalized to determine disease staging. Stable patients with painless mass or abnormal mammography and medical resources should be referred to breast surgeon for biopsy.

Pearls

1. Approximately 250,000 cases of breast cancer are diagnosed annually in the United States with an associated 40,000 deaths.
2. Mortality rates have decreased over the past 50 years in part due to improved breast cancer screening, patient awareness, and adjuvant therapy.
3. Denial delays time-sensitive treatment.



FIGURE 7.91 ■ Breast Mass. Necrotic breast mass in patient with suspected advanced breast cancer. (Photo contributor: Kevin Barlotta, MD.)



FIGURE 7.92 ■ Breast Mass. Peau d'orange appearance of the breast suggesting underlying breast cancer. (Photo contributor: Michael G. Clark.)

The authors wish to thank Stephen W. Corbett for his contributions to prior editions.

Chapter 8

UROLOGIC CONDITIONS

Sarah Moore
Ryan Pedigo



Priapism. Aspiration of the corpora cavernosa followed by injection of α -adrenergic agents such as phenylephrine through the same needle is demonstrated by combining two syringes with a three-way stopcock. (Photo contributor: David Effron, MD.)

TESTICULAR TORSION

Clinical Summary

Testicular torsion is a twisting of the spermatic cord that leads to testicular ischemia and is a surgical emergency. This condition mainly affects neonates and adolescents, but can occur in older adults as well. Common symptoms include acute-onset testicular pain and swelling with an exam revealing a tender testicle lying in a horizontal plane (bell-clapper deformity). Nausea, vomiting, and abdominal pain may be present.



FIGURE 8.1 ■ Testicular Torsion. Swollen, tender hemiscrotum, with erythema of scrotal skin and retracted testicle. (Photo contributor: Stephen W. Corbett, MD.)

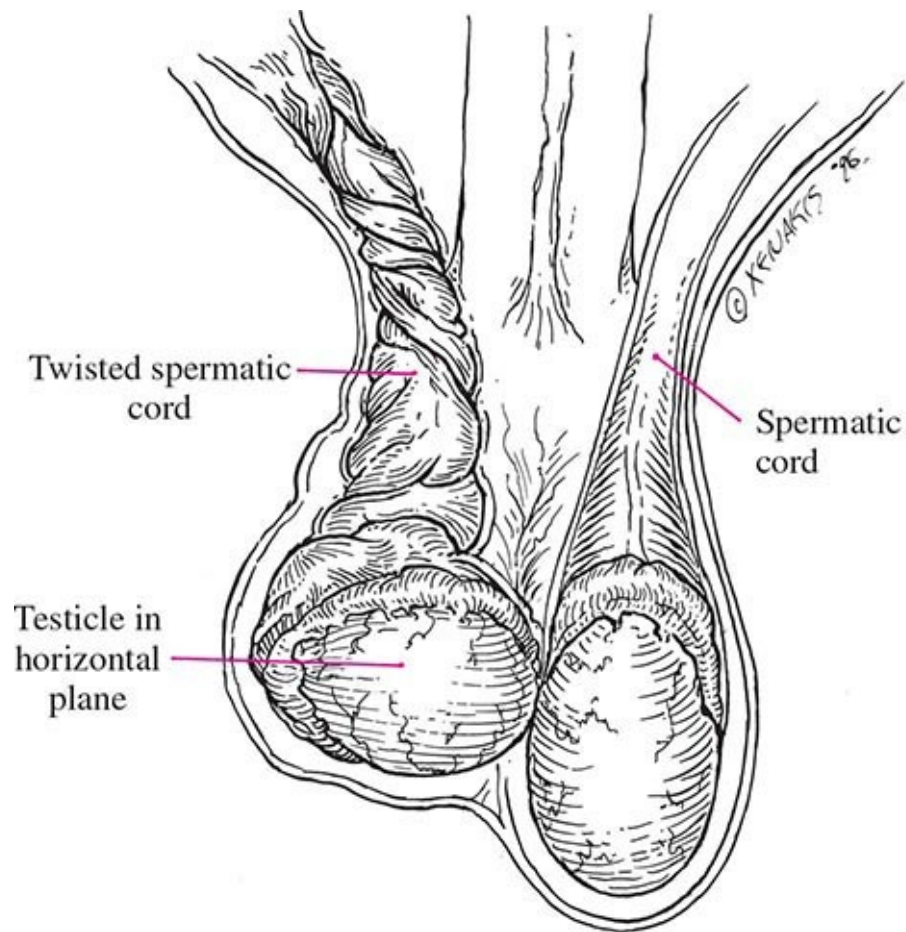


FIGURE 8.2 ■ Bell-Clapper Deformity. Twisting of the spermatic cord causes the testicle to be elevated with a horizontal lie. Lack of fixation to the posterior scrotum predisposes the freely movable testicles to rotation and subsequent torsion. Asymptomatic patients with bell-clapper deformity are at risk for torsion.



FIGURE 8.3 ■ Testicular Torsion. A retracted testicle consistent with early testicular torsion (minimal edema) is seen in both of these patients. (A) (Photo contributor: David W. Munter, MD, MBA.) (B) (Photo contributor: Emergency Medicine Department, Naval Medical Center Portsmouth, VA.)

Management and Disposition

If testicular torsion is suspected, obtain immediate urologic consultation. Ultrasound is diagnostic (unless intermittent torsion is present), but surgical management should not be delayed for confirmatory imaging as the time to operative intervention predicts testicular viability. Manual detorsion can be attempted if operative management is not immediately available. In roughly two-thirds of cases, testicular torsion occurs in the medial direction, so rotating the testicle away from the midline may temporarily improve blood flow. The common explanation of the maneuver is that of “opening a book” where the direction of rotation of the patient’s right testicle is counterclockwise when viewed from below and the left is clockwise.



FIGURE 8.4 ■ Testicular Torsion. Swollen, tender scrotal mass. (Photo contributor: Patrick McKenna, MD.)



FIGURE 8.5 ■ Testicular Torsion. A red, swollen hemiscrotum is seen in this right testicular torsion in a 14-year-old. (Photo contributor: Lawrence Heiskell, MD.)



FIGURE 8.6 ■ Testicular Torsion. Intraoperative image of a testicular torsion which required orchiectomy due to prolonged ischemia and 720 degree torsion. (Photo contributor: Anne Louise Darves-bornoz, MD.)

Pearls

1. The cremasteric reflex is usually absent in testicular torsion but is not pathognomonic.
2. Patients may report similar, less severe episodes that spontaneously resolved in the recent past.
3. Approximately two-thirds of all torsions occur during sleep.
4. Abdominal or inguinal pain is sometimes present without pain to the scrotum.
5. The age of presentation has a bimodal pattern, since torsion is more prevalent during infancy and adolescence.

TORSION OF A TESTICULAR OR EPIDIDYMAL APPENDIX

Clinical Summary

Vestigial remnants in the embryology of the scrotum are often found as appendages on the superior portions of the testicle or the epididymis and can occasionally undergo torsion. This most commonly occurs in boys up to 16 years of age but has been reported in adults. The patient complains of sudden pain around the superior pole of the testicle or epididymis as the appendix undergoes necrosis and inflammation. Early in the course, palpation of a firm, tender nodule in this area will confirm the diagnosis.

Management and Disposition

Obtain urologic consultation immediately. Differentiating from the more emergent testicular torsion is the key responsibility. Ancillary studies are generally not helpful in making this diagnosis unless it presents very early in its course. The characteristic physical signs of a small, tender, upper-pole nodule along with a color Doppler ultrasound showing good flow to the testicle may mitigate the need for emergent surgery. With later presentations or an equivocal ultrasound, the diagnosis may not be made with confidence before surgery. If surgery is not deemed necessary by the urologic consultant, analgesics and rest are all that is required. The appendix will involute and calcify in 1 to 2 weeks.

Pearls

1. Stretching of the scrotal skin across the necrotic nodule will occasionally reveal a bluish discoloration of the nodule, called the “blue-dot sign.” This is pathognomonic for torsion of the appendix.
2. A reactive hydrocele may accompany appendiceal torsion. When the hydrocele is transilluminated, the blue-dot sign may be revealed.



FIGURE 8.7 ■ Blue-Dot Sign. A blue-dot sign is caused by torsion of the testicular appendix. It is best seen with the skin held taut over the testicular appendix. (Photo contributor: Javier A. Gonzalez del Rey, MD.)



FIGURE 8.8 ■ Blue-Dot Sign. A blue-dot sign is seen in an older patient. (Photo contributor: Alan B. Storrow, MD.)

ACUTE EPIDIDYMITIS

Clinical Summary

The onset of scrotal pain typically occurs over hours and is often referred to the ipsilateral inguinal canal or lower abdominal quadrant. Recent urinary tract instrumentation or urinary tract infection is a risk factor. Early in the course, a tender, indurated, edematous epididymis is palpated separately from the nontender testicle. Late presentations will have generalized scrotal swelling and tenderness, making examination and differentiation more difficult. The urinalysis reveals pyuria or bacteriuria half of the time, and the peripheral white blood cell count is frequently elevated. Patients can present with fever and signs of sepsis. Approximately half the time, the epididymis and adjacent testicle will be inflamed, which is termed epididymo-orchitis.

Management and Disposition

Men under the age of 35 can be treated empirically for sexually transmitted infections (STIs), but also should be treated for enteric organisms if they practice insertive anal intercourse. Older men without other risk factors tend to have enteric organisms and not STIs as the cause of infection and are treated with a fluoroquinolone such as levofloxacin. Most patients can be treated as outpatients, but consider admission and intravenous (IV) antibiotics for febrile patients. Ultrasound is not necessary in classic presentations but may be useful for cases that are not straightforward or for cases where more serious conditions such as testicular torsion are on the differential.

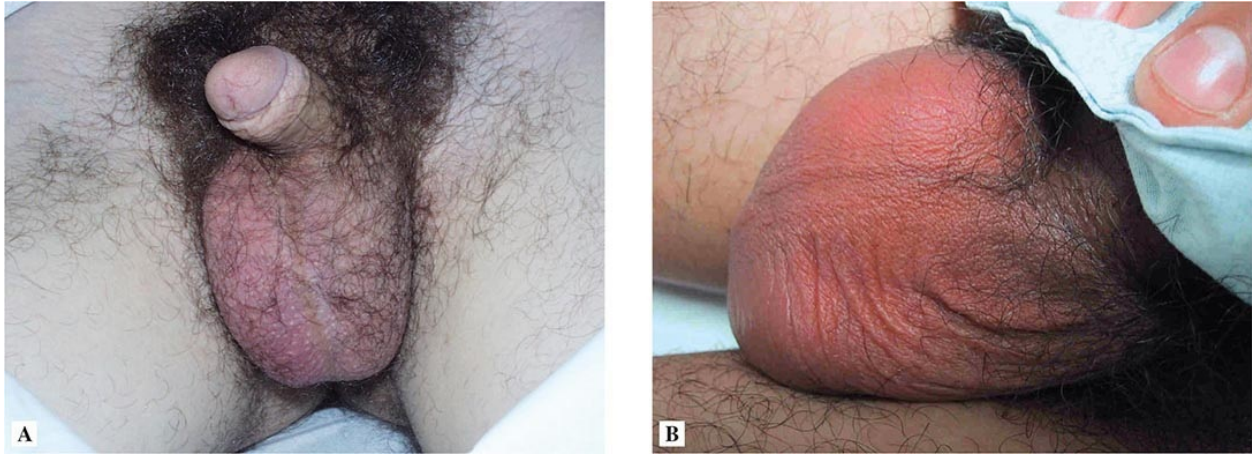


FIGURE 8.9 ■ Acute Epididymitis. (A) Swelling of the right hemiscrotum and tenderness of the inferior posterior portion of the testicle. (Photo contributor: Emergency Medicine Department, Naval Medical Center Portsmouth, VA.) (B) Diffuse and erythema swelling of the entire scrotum. (Photo contributor: Cyril Thomas, PAC.)

Pearls

1. Elevation of the affected hemiscrotum while standing may provide relief of symptoms (Prehn sign).
2. Evaluate older men for urinary retention, as this is a frequent cause of epididymitis.
3. Testicular tumors are most frequently misdiagnosed as epididymitis.
4. The absence of pyuria or bacteriuria does not exclude the diagnosis.
5. Referred pain to the lower quadrants can mimic appendicitis or diverticulitis.

ORCHITIS

Clinical Summary

Orchitis has a variable onset and ranges from mildly uncomfortable to severely painful. It is most frequently a complication of epididymitis, but isolated orchitis without epididymitis can be caused by mumps infection and, more rarely, other viruses. Mumps orchitis occurs 4 to 7 days after parotid symptoms with testicular pain and swelling. It is unilateral 70% of the time with a contralateral infection developing later 30% of the time. The testicle is swollen and tender, sparing the epididymis. The overlying scrotal skin can be edematous and

erythematous. Constitutional symptoms of malaise, headache, myalgias, and fever are common.

Management and Disposition

Supportive care with analgesics, hot or cold packs, and scrotal elevation is sufficient for mumps orchitis. Orchitis in the context of epididymo-orchitis is treated the same as epididymitis. Obtain ultrasound to rule out testicular torsion.

Pearls

1. An enlarged, tender epididymis or boggy, tender prostate supports bacterial epididymo-orchitis and is treated the same as epididymitis.
2. Preceding or concurrent parotid swelling supports mumps orchitis.



FIGURE 8.10 ■ Orchitis. Unilateral testicular pain, swelling, scrotal erythema, and edema are seen in this patient with parotitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 8.11 ■ Orchitis. Swelling and erythema of the right hemiscrotum in a patient with orchitis. (Photo contributor: David Effron, MD.)

HYDROCELE

Clinical Summary

Most hydroceles occur in older patients and develop gradually without significant symptoms. Hydrocele presents as a soft, pear-shaped, fluid-filled cystic mass anterior to the testicle and epididymis that will transilluminate. However, it can be tense and firm and will transilluminate poorly if the tunica

vaginalis is thickened. Almost all hydroceles in children are communicating, resulting from the same mechanism that causes inguinal hernia. A persistent, narrow processus vaginalis acts like a one-way valve, thus permitting the accumulation of dependent peritoneal fluid in the scrotum. Acute symptomatic hydroceles are rarer and can occur in association with epididymitis, trauma, or tumor.

Management and Disposition

Most hydroceles accumulate slowly over time, and many do not require intervention. However, in an acute hydrocele, ultrasound can be helpful in cases of trauma or concern for infection or malignancy. Acute hydroceles should be referred to a urologist. Refer chronic accumulations to a urologist on a more routine basis for elective drainage. Congenital hydroceles in infants should have watchful waiting, as spontaneous resolution can occur.

Pearls

1. Ten percent of testicular tumors have a reactive hydrocele as the presenting complaint.
2. A hydrocele may be mistaken for an inguinal hernia on initial exam; use transillumination or, if still unclear, ultrasound.
3. Consider a reactive hydrocele from an secondary process if acute and/or painful.

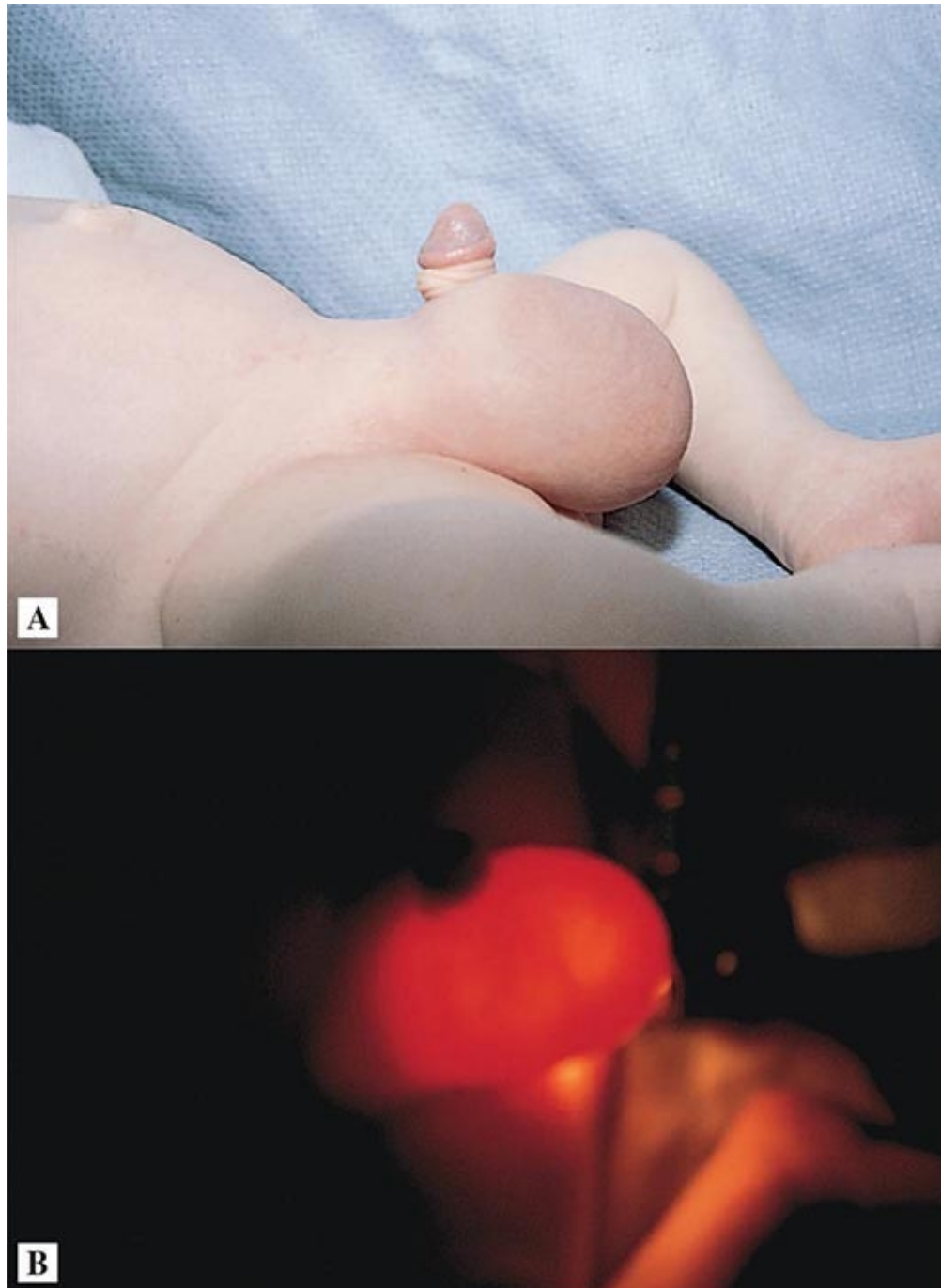


FIGURE 8.12 ■ Hydrocele. Painless swelling in the scrotum of a young boy (**A**). Transillumination of the swelling (**B**) identifies the hydrocele. (Photo contributor: Michael J. Nowicki, MD.)

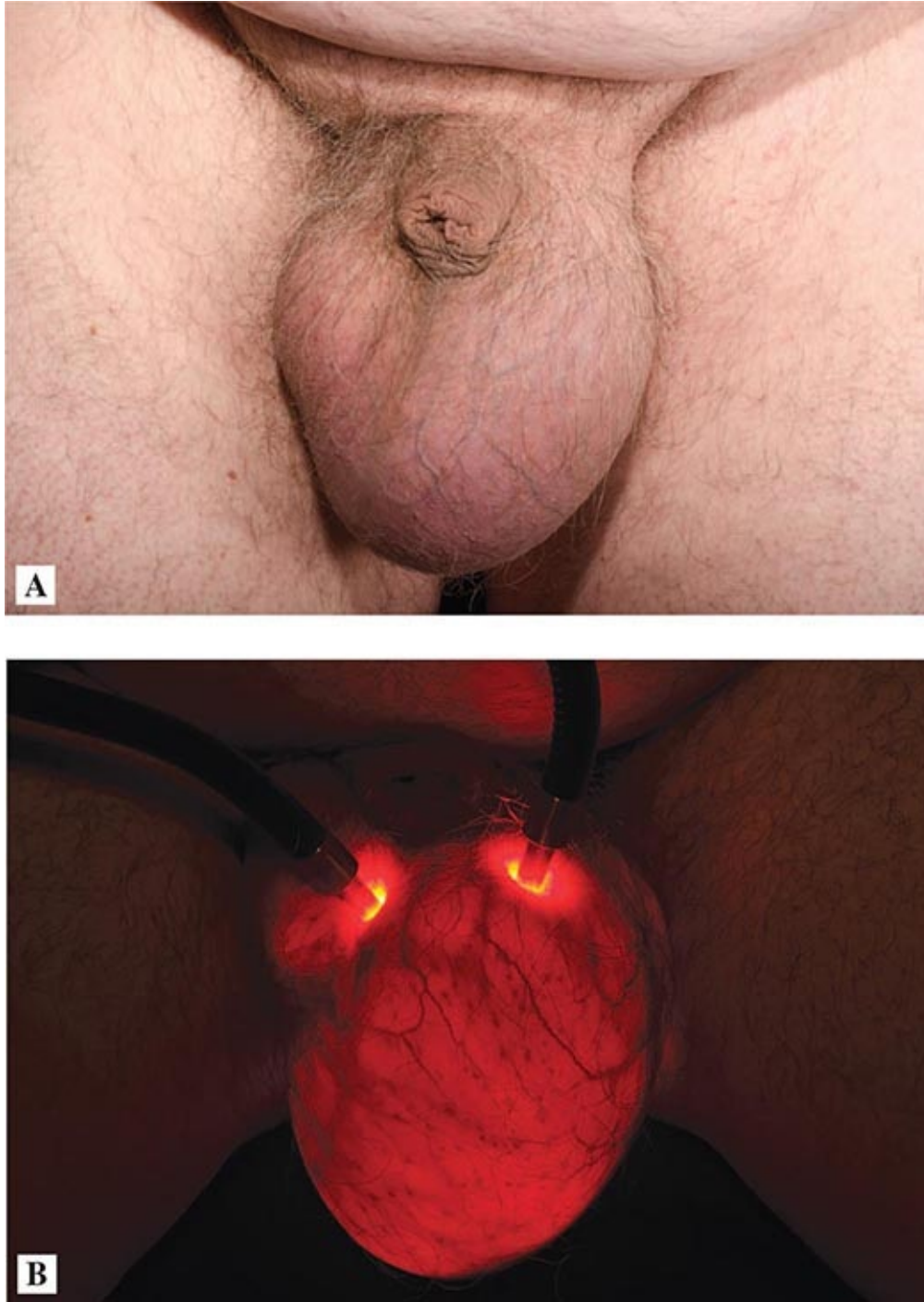


FIGURE 8.13 ■ Hydrocele. Hydrocele without (A) and with (B) transillumination. (Photo contributor: David Bryson. Reproduced from Bryson D. Transillumination of testicular hydrocele. *Clin Med Img Lib.* 2017;3(3):075.)

PYOCLE

Clinical Summary

A scrotal pyocele is a purulent collection of fluid surrounding the testicle in the potential space of the tunica vaginalis and can be distinguished from a hydrocele by presence of internal echoes consistent with debris, septations, or loculations. A scrotal pyocele is typically a sequela of adjacent infection, such as epididymo-orchitis or rupture of a testicular abscess. Less commonly, it can be due to tracking of an intraperitoneal infection through the inguinal canal. The cellular debris may also be blood, such as in a hematocele, but there is usually a history of antecedent trauma in this setting.

Management and Disposition

Urologic consultation should be obtained; surgical drainage and broad-spectrum antibiotics are indicated. Severe cases may compromise viability of the adjacent testicle, and an orchiectomy may be required. In cases where a deeper infection is suspected, such as Fournier gangrene, obtain early IV contrast-enhanced computed tomography (CT) imaging of the pelvis.

Pearls

1. Internal echoes in fluid surrounding the testicle indicate either cellular debris from pus (pyocele) or from blood (hematocele).
2. Obtain urologic consultation for pyoceles.
3. Consider deeper infection such as Fournier gangrene, and obtain CT imaging if suspected.

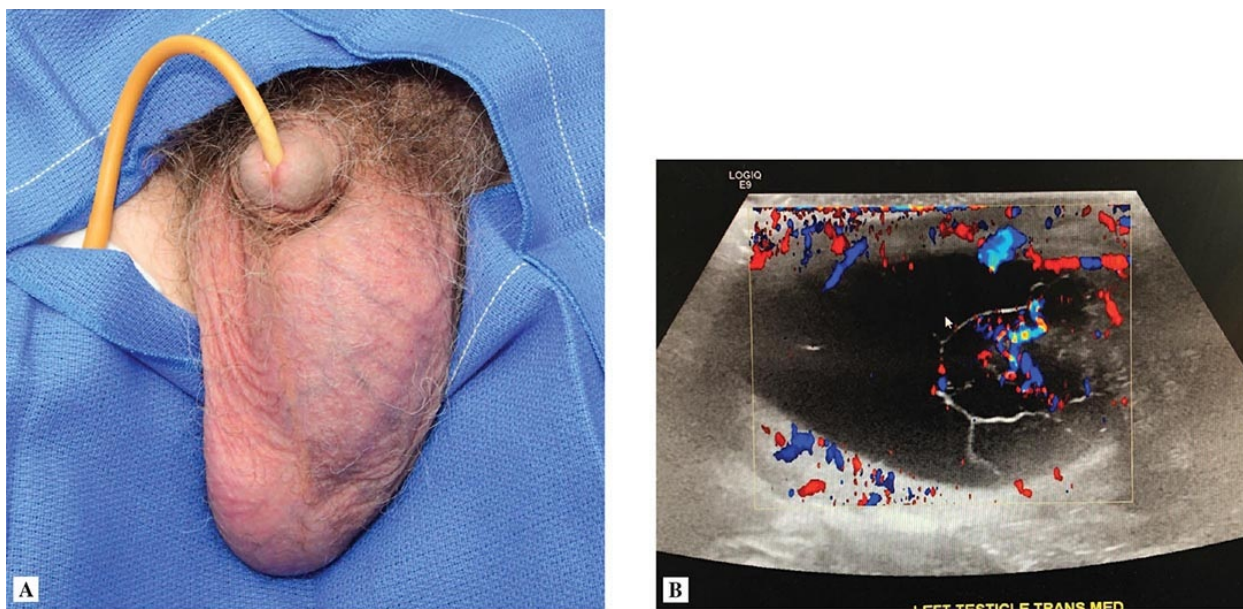


FIGURE 8.14 ■ Scrotal Pyocele. An adult with a pyocele showing (A) swelling of the left hemiscrotum, and (B) ultrasound findings of a fluid collection in the scrotum with internal septations and debris consistent with a pyocele. (Photo contributor: Lawrence B. Stack, MD.)

TESTICULAR TUMOR

Clinical Summary

In testicular tumor, a painless, firm testicular mass is palpated, with the patient often complaining of a “heaviness” of his testicle. If the patient presents early, the mass will be distinct from the testis, whereas later presentations will have generalized testicular or scrotal swelling. These lesions occasionally present with pain due to infarction of the tumor.

Management and Disposition

Refer patients promptly to a urologist for definitive diagnosis and management.

Pearls

1. Acute hydroceles and hematoceles should prompt the physician to consider a tumor as the cause.
2. Pain from tumor infarction is usually not as severe as pain due to torsion or

epididymitis.

3. Findings of an unexplained supraclavicular lymph node, abdominal mass, or chronic nonproductive cough resistant to conventional therapy should prompt a testicular examination for tumor.

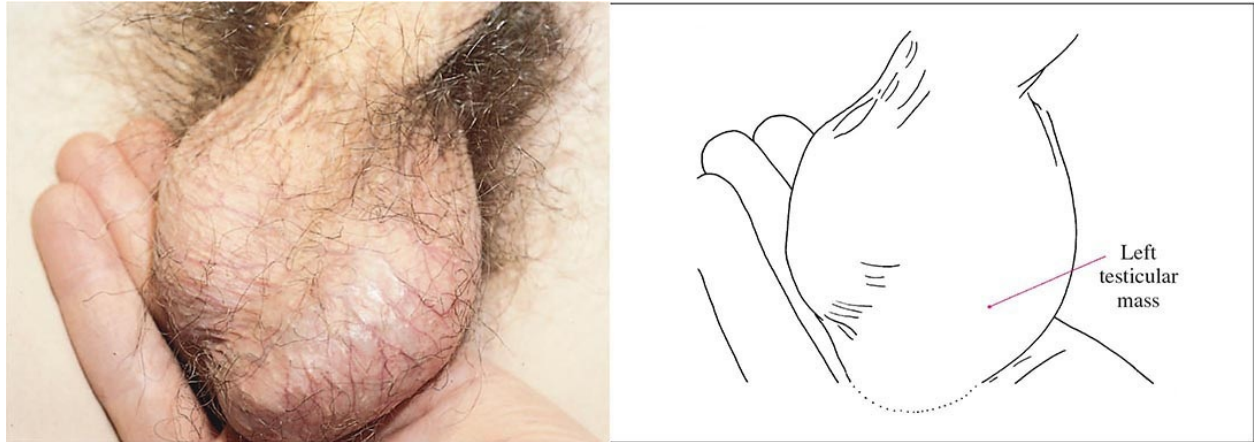


FIGURE 8.15 ■ Testicular Tumor. This painless left testicular mass is highly suspicious for tumor, as proved to be the case in this patient. (Photo contributor: Patrick McKenna, MD.)

SCROTAL ABSCESS

Clinical Summary

A scrotal abscess is a suppurative mass with surrounding erythema involving the superficial layers of the scrotal wall. The usual history given is progressive swelling of a small pustule or papule with increasing pain and induration or fluctuance. Constitutional symptoms and fever are generally absent.

Management and Disposition

For superficial scrotal abscesses of a cutaneous origin, use local anesthesia and simply make a stab incision and drain the abscess using the same technique as for incision and drainage elsewhere. Instruct the patient to use a sitz bath and to change the dressing frequently. Postdrainage antibiotics are not usually indicated. Immunocompromised patients may require IV antibiotics and admission. For more complex abscesses or abscesses thought to be secondary to epididymo-orchitis or associated with Fournier gangrene, consult urology.

Pearl

1. If the patient appears ill out of proportion to the superficial appearance, suspect that this mass is the point of a deep scrotal abscess or Fournier gangrene.



FIGURE 8.16 ■ Scrotal Abscess. Suppurative mass on the scrotum. (Photo contributor: David Effron, MD.)



FIGURE 8.17 ■ Scrotal Abscess. A chronic recurring abscess with intermittent drainage in a patient with diabetes. (Photo contributor: Kevin J. Knoop, MD, MS.)

FOURNIER GANGRENE

Clinical Summary

Fournier gangrene, a form of necrotizing fasciitis, most frequently occurs in a middle-aged diabetic man who presents with swelling, erythema, and severe pain of the entire scrotum, but it is also known to occur in women. In men, the scrotal contents often cannot be palpated because of the marked inflammation. The patient has constitutional symptoms with fever and frequently is in shock. There is often a history of recent urethral instrumentation, an indwelling Foley catheter, or perirectal disease. A localized area of fluctuance usually cannot be appreciated.

Management and Disposition

These patients require aggressive fluid resuscitation and early surgical consultation for immediate debridement and surgical drainage. Give broad-spectrum antibiotics effective against gram-positive, gram-negative, and anaerobic organisms as soon as possible in the emergency department.

Pearls

1. Pain out of proportion to the clinical findings may represent an early presentation of Fournier gangrene.
2. CT imaging can delineate the depth and extent of disease but should not delay operative intervention.
3. Fournier gangrene is usually quite painful but has been known to present with only a mildly uncomfortable necrosis of the scrotal wall and exposed testis.



FIGURE 8.18 ■ Fournier Gangrene. Middle-aged diabetic man with fever and scrotal pain. Final diagnosis is polymicrobial Fournier gangrene. (Photo contributor: Eugene C. Eiland, MD.)

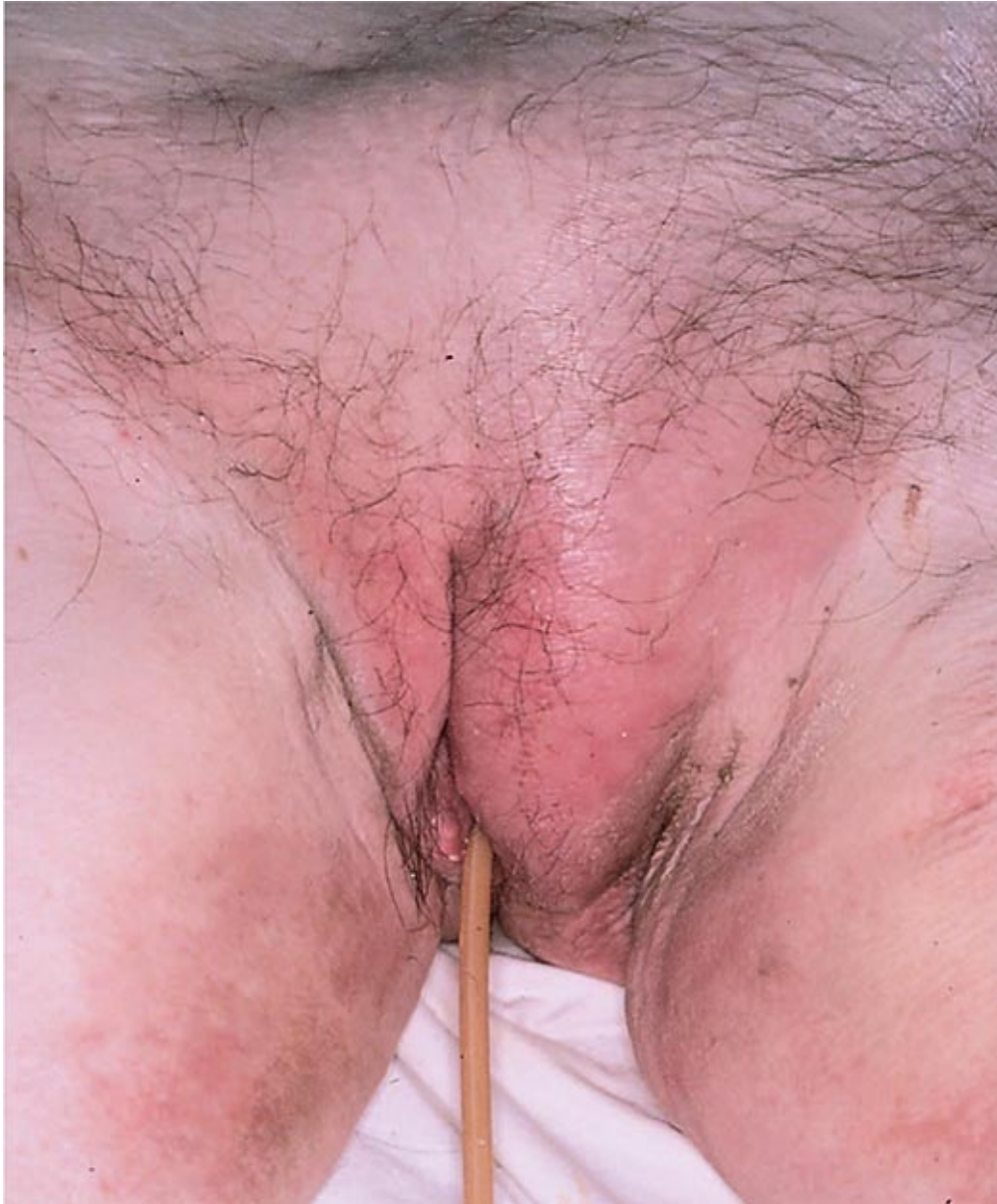


FIGURE 8.19 ■ Fournier Gangrene. Swollen, tender, erythematous labia, perineum, and inner thighs in a female patient with Fournier gangrene. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 8.20 ■ Fournier Gangrene. Necrosis of overlying scrotal skin along with swelling, erythema, high fever, and severe pain was noted in this diabetic patient with Fournier gangrene. (Photo contributor: R. Jason Thurman, MD.)

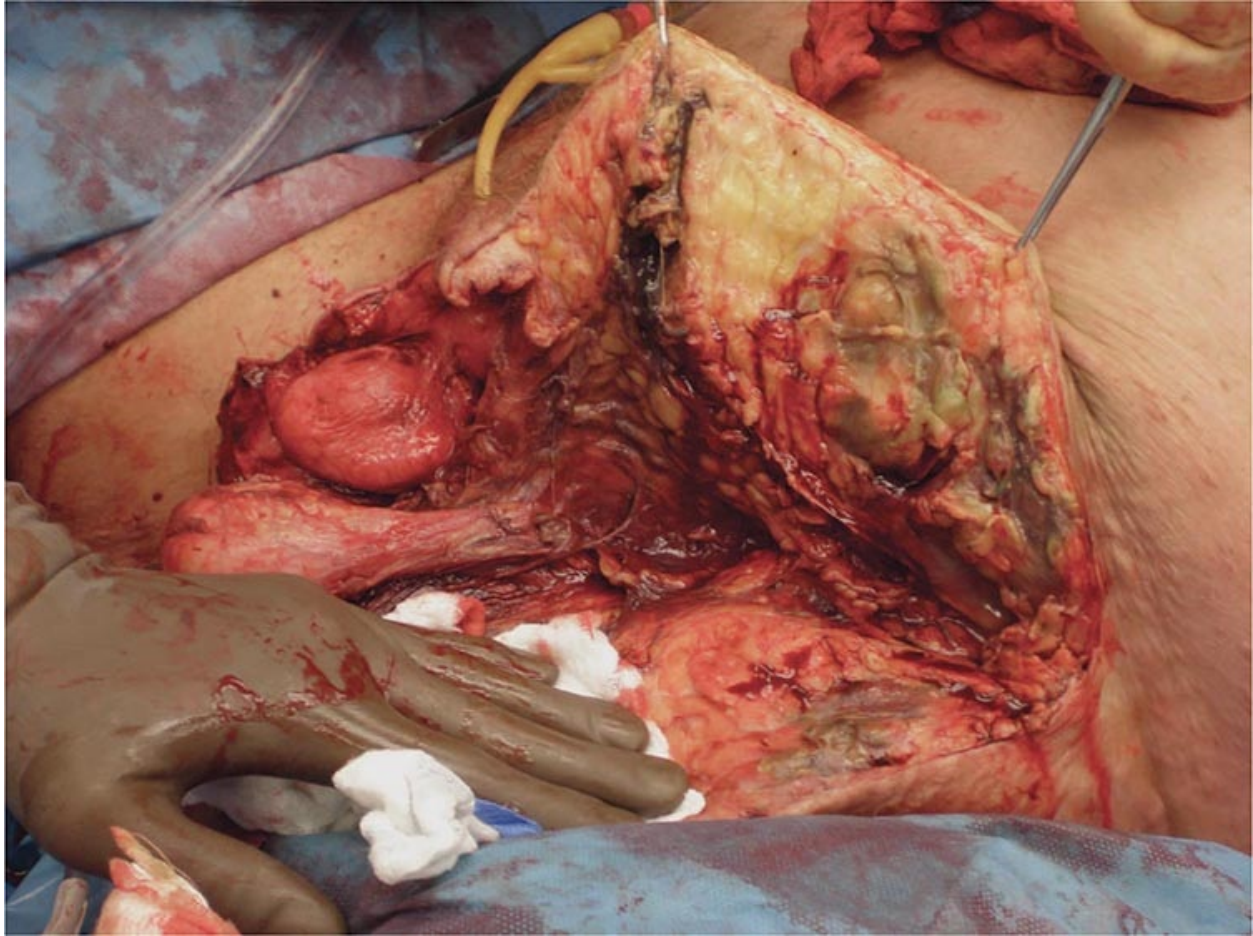


FIGURE 8.21 ■ Fournier Gangrene. The extent of necrosis in Fournier gangrene can be formidable, as seen in this patient. (Photo contributor: R. Jason Thurman, MD.)

PARAPHIMOSIS

Clinical Summary

Paraphimosis is the entrapment of a retracted foreskin that cannot be reduced behind the coronal sulcus. Pain, swelling, and erythema are common. If severe, the constriction causes edema and venous engorgement of the glans, which can lead to arterial compromise with subsequent tissue necrosis. In contrast to paraphimosis, phimosis is the inability to retract the foreskin, which can be physiologic in children.

Management and Disposition

First liberally apply granulated sugar to the glans and foreskin (prepuce). After 15 to 20 minutes, the osmotic effect will help decrease edema and ease reduction. Next squeeze the glans firmly for 5 minutes to reduce the swelling and then reduce the glans into the foreskin by pushing the glans inward with the thumbs while the index fingers pull the prepuce over the coronal sulcus. If manual reduction fails, consult a urologist for local infiltration of anesthesia with vertical incision of the constricting band.

Pearls

1. Reduction of the edema with firm, constant pressure is generally effective.
2. Do not forcibly retract the foreskin in patients with phimosis.



FIGURE 8.22 ■ Paraphimosis. Moderate edema of retracted foreskin, which is entrapped behind the coronal sulcus. (Photo contributor: Lawrence B. Stack, MD.)

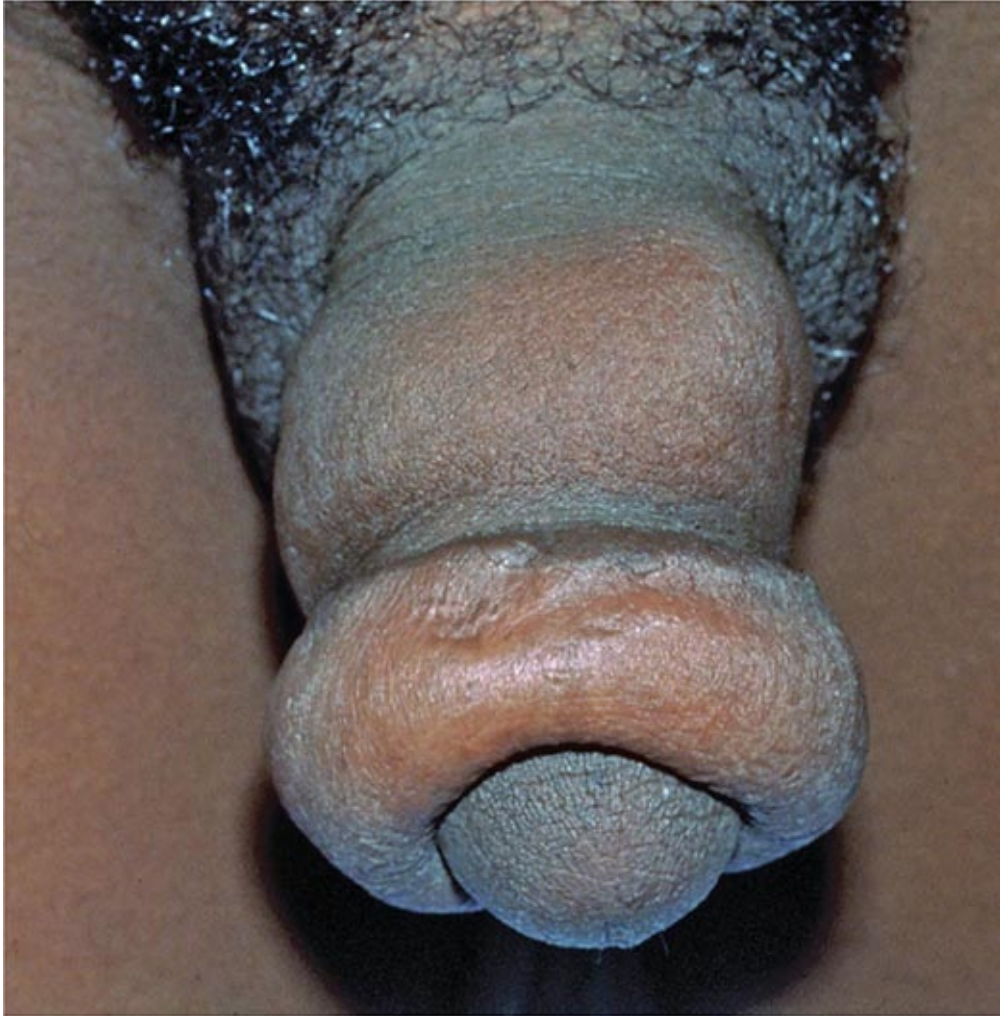


FIGURE 8.23 ■ Paraphimosis. Edema of retracted foreskin entrapped behind the coronal sulcus. (Photo contributor: David Effron, MD.)



FIGURE 8.24 ■ Phimosis. Phimosis in a young patient is physiologic, but also may have obstruction from meatal stenosis and scarring. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 8.25 ■ Phimosis. Phimosis that required catheterization to relieve obstruction. (Photo contributor: David Effron, MD.)

PRIAPISM

Clinical Summary

Low-flow priapism is a compartment syndrome of the penis resulting from a failure of detumescence. Ischemia after only 4 to 6 hours can lead to fibrosis and possible permanent erectile dysfunction. It can be due to sickle cell disease, medications, or illicit drugs. High-flow priapism is often due to a traumatic arteriocavernosal fistula and, although alarming, has a low risk for ischemia due to the preservation of oxygenated blood flow to the penis.

Management and Disposition

In low-flow priapism, the penis is at risk for irreversible ischemic damage after only a few hours and should be considered a urologic emergency. Subcutaneous

terbutaline can be attempted, but 1st-line therapy is intracavernosal injection of phenylephrine with or without aspiration. Mix 1 mg in 9 mL of normal saline to make 100 µg/mL of solution. Up to 2000 µg may be needed, but small aliquots are recommended. Ensure the patient is on the monitor with blood pressure, heart rate, and pulse oximeter. A urologist should be consulted as operative intervention may be required. For high-flow priapism, consult urology; no emergent intervention is needed by the emergency provider.

Pearls

1. Low-flow priapism (think “penile compartment syndrome”) is an emergency, and early therapy and urologic consultation can improve outcomes.
2. Suspect high-flow priapism in a patient with trauma and painless priapism.



FIGURE 8.26 ■ Priapism. A painful persistent erection due to pathologic engorgement of the corpora cavernosa is seen in this patient with sickle cell disease. The glans penis and corpus spongiosum are not engorged. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 8.27 ■ Priapism. Aspiration of the corpora cavernosa is demonstrated. (Photo contributor: David Effron, MD.)



FIGURE 8.28 ■ Traumatic Priapism. A persistent erection is seen in this trauma victim who has sustained a cord injury. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 8.29 ■ Priapism. Before (A) and after (B) aspiration and irrigation with phenylephrine in a patient with recurrent priapism due to risperidone. The involuntary erection had lasted for 10 hours and required 2000 µg of phenylephrine to achieve detumescence. An artificial right testicle also seen. (Photo contributor: Kevin J. Knoop, MD, MS.)

URETHRAL RUPTURE

Clinical Summary

Urethral injury is rarely an isolated event; it is often associated with multiple trauma. Anterior urethral injuries are most often the result of a straddle injury and may present late (many patients are still able to void) with a local infection or sepsis from extravasated urine. Posterior urethral injuries occur in motor vehicle and motorcycle accidents and are usually the result of pelvic fractures. Patients have blood at the urethral meatus, cannot void, and have perineal bruising. In men, the prostate is often boggy or free-floating or may not be palpable at all if there is a retroperitoneal hematoma between the prostate and the rectum.

Management and Disposition

Do not allow urethral instrumentation such as Foley catheterization prior to a retrograde urethrogram with highly concentrated water-soluble contrast. If there is only a partial anterior tear, gently attempt catheterization but abandon it at the 1st sign of resistance. If catheterization is unsuccessful and whenever there is a posterior tear, place a suprapubic catheter if relief of bladder distention is required prior to operative repair.

Pearls

1. Foley catheter insertion is contraindicated in patients with a suspected urethral injury prior to a retrograde urethrogram.
2. Urethral injury should be suspected in the multiple-trauma patient who is unable to void or has blood at the meatus, a high-riding prostate, or perineal trauma.
3. Vaginal lacerations due to trauma in women should prompt consideration of a urethral tear.

4. Occasionally, urine from an anterior urethral tear will extravasate into the scrotum, causing marked swelling.
5. Posterior injuries are frequently associated with other intra-abdominal injury.

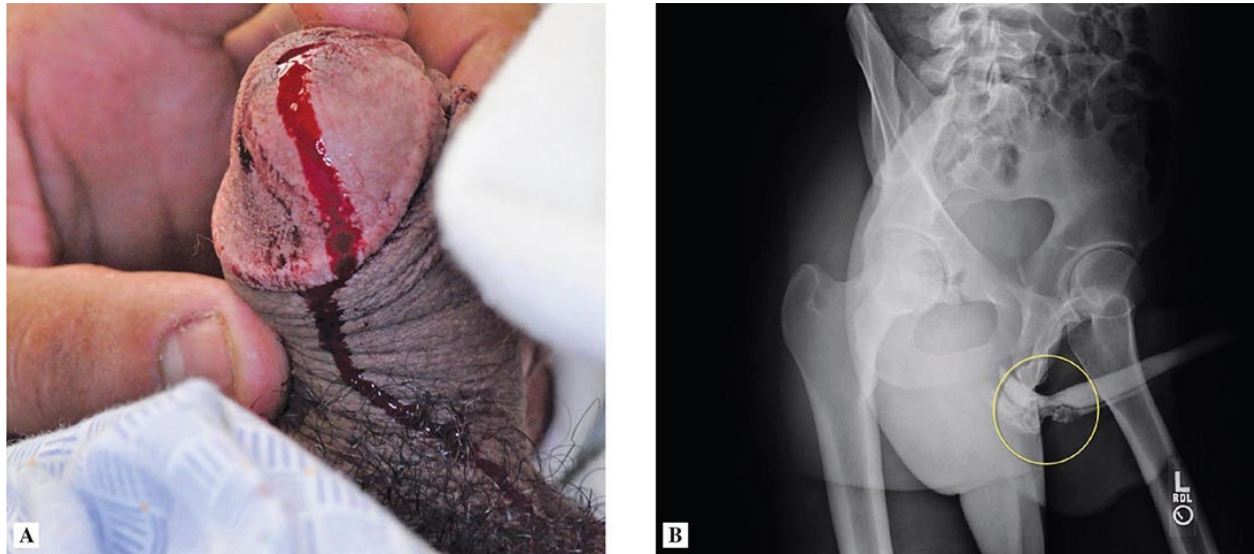


FIGURE 8.30 ■ Urethral Rupture. (A) Blood at the urethral meatus in a patient with an anterior urethral rupture secondary to a straddle injury. (B) A retrograde urethrogram shows extravasation of dye indicating rupture. (Photo contributor: David Effron, MD.)

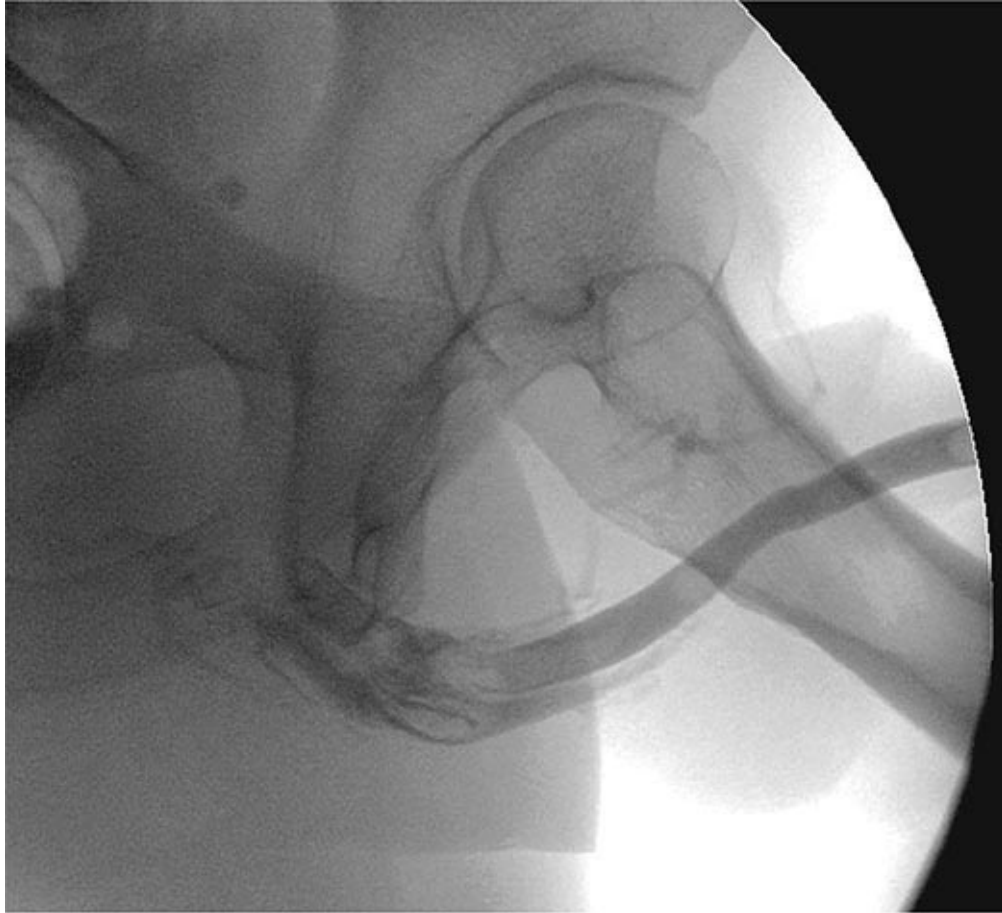


FIGURE 8.31 ■ Urethral Injury. A retrograde urethrogram demonstrates contrast extravasation, signifying a urethral injury. (Photo contributor: Dirk Liebchen, MD.)

FRACTURE OF THE PENIS

Clinical Summary

Patients usually present complaining of trauma during sexual arousal and often relate a sudden “snapping” sound or sensation, pain, and deformity, which is caused by a tearing of the tunica albuginea. The shaft of the penis is swollen and often angulated at the fracture site (called “eggplant deformity”).

Management and Disposition

If the patient cannot urinate, obtain a retrograde urethro-gram to rule out urethral injury. Obtain urologic consultation as patients are frequently taken immediately

to the operating room for repair.

Pearls

1. Patients sometimes concoct elaborate stories that are not sexually related surrounding the circumstances of injury, but penile fracture most commonly occurs during sexual arousal.
2. Penile implants are also subject to injury in a similar fashion.



FIGURE 8.32 ■ Penile fracture. Penile fracture with ecchymosis and angulation. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 8.33 ■ Penile fracture. Penile fracture with ecchymosis and edema without angulation. (Photo contributor: Emergency Medicine Department, Naval Medical Center Portsmouth, VA.)



FIGURE 8.34 ■ Penile Fracture. A penile fracture showing classic “eggplant deformity.” (Reproduced with permission from Cooper B, Beres K. Penile fracture. *J Emerg Med.* 2017;52(2):238-239. © Elsevier Inc. 2016.)

PENILE TOURNIQUET

Clinical Summary

A hair or filament that wraps around the penis can lead to a surgical emergency. The constricting band will impair distal venous and lymphatic drainage causing more edema and further impairing drainage. The ultimate sequela is arterial compromise and amputation. This is most commonly seen in young children, although it has also been reported in men who use penile rings or constrictors.

Management and Disposition

Immediate release of the constriction generally gives relief and restores any impaired circulation. Take care not to injure any underlying structures. If the edema is so great that the constricting band cannot be easily released, obtain emergent uro-logic consultation.

Pearls

1. Edema may obscure the hair or filament and bury it subcutaneously.
2. Measures to decrease the swelling, such as direct pressure and ice packs, may facilitate visualization and incision of the tourniquet.
3. Clitoral tourniquets have been described.
4. Penile tourniquet should be considered when presented with a fussy infant.

STRADDLE INJURY

Clinical Summary

In straddle injury, the patient has pain, swelling, contusion, and hematoma of the perineum or scrotum following direct blunt trauma. This injury is commonly caused by a fall onto a bicycle frame top tube, playground equipment, or a toilet seat. Swelling can be severe enough to interfere with urination. Scrotal contents can also be contused or crushed with this injury.

Management and Disposition

Treatment is supportive and includes cold packs, elevation, rest, and analgesics. If unable to void, catheterize the patient, but first obtain a retrograde urethrogram if concomitant urethral injury is suspected.



FIGURE 8.35 ■ Penile Tourniquet. Penile engorgement due to self-inflicted tourniquet in a prepubescent boy. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 8.36 ■ Straddle Injury. Marked ecchymosis, swelling, and contusion of the perineum in an adolescent girl. Examination under anesthesia was required to determine the extent of this patient's injuries. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 8.37 ■ Straddle Injury. Contusion of the scrotum and lower abdomen in a young boy consistent with a straddle injury. (Photo contributor: David W. Munter, MD.)



FIGURE 8.38 ■ Straddle Injury. This unfortunate patient suffered a vaginal tear as a result of slipping forward off the seat of her bicycle and landing on its top tube. (Photo contributor: R. Jason Thurman, MD.)

Pearls

1. Perineal laceration can be obscured by swelling if a careful examination is not performed.
2. Pelvic radiographs should be obtained in all perineal injuries.
3. Men and women are at high risk for urethral injuries with this type of injury.
4. Straddle injury is differentiated from abuse with a good history from a reliable caregiver that matches the injury.

BALANOPOSTHITIS

Clinical Summary

Balanoposthitis is an infection and inflammation of the glans penis that also involves the overlying foreskin (prepuce). *Balanitis* is isolated to the glans, whereas *posthitis* involves only the prepuce. Pain, erythema, and edema of the affected parts of the penis are typically present. Patients may refrain from urination secondary to dysuria, or the edema may induce meatal occlusion, leading to urinary retention or obstruction. Common etiologies include overgrowth of normal bacterial flora secondary to poor hygiene (pediatric patients), STIs (adolescents and adults), and candidal infections (the elderly or immunocompromised).

Management and Disposition

Treatment is directed at the suspected etiology. Warm soaks and topical antibiotics (mupirocin) are the mainstay of therapy for infectious etiologies owing to poor hygiene. Counsel parents about proper cleansing and handling of the prepuce. Oral or IV antibiotics may be indicated if there is an accompanying cellulitis or urinary tract infection. If urinary obstruction is present, attempt catheterization using a small catheter. If catheterization is unsuccessful, urologic consultation for emergent surgical correction of the prepuce is required. Candidal infections are treated with meticulous hygiene and topical antifungal agents.

Pearls

1. The inability to retract the foreskin completely is normal in boys up to the teens. Counsel parents that the prepuce should never be forcibly retracted. Attempting to do so could cause a paraphimosis, a true emergency.
2. Placing the child in a bathtub with warm water will help alleviate difficulty with micturition assuming that no obstruction is present.
3. Candidal balanitis or balanoposthitis may be indicative of an undiagnosed immunocompromised state, including diabetes.



FIGURE 8.39 ■ Balanoposthitis. Note the erythema, localized edema, and significantly constricted preputial orifice of the distal penis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 8.40 ■ Balanitis. Candidal balanitis in an elderly patient with no other complaints. New-onset diabetes was diagnosed. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 8.41 ■ Balanoposthitis. This toddler presented with physiologic phimosis, inflamed distal foreskin, dysuria, and swelling of the glans. (Photo contributor: James Palma, MD.)



FIGURE 8.42 ■ Balanoposthitis. Candidal balanoposthitis in an uncircumcised man. (Photo contributor: Alan Storrow, MD.)

PENILE ZIPPER INJURY

Clinical Summary

Zipper injury is usually seen in young children. The foreskin becomes entrapped in the teeth of the zipper or between the fastener and the zipper teeth as the zipper is being opened (downward). Accessibility to the median bar of the zipper facilitates removal.

Management and Disposition



FIGURE 8.43 ■ Penile Zipper Injury. Entrapment of the foreskin between the fastener and the zipper teeth is seen. The median bar is exposed and easily accessible for cutting. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 8.44 ■ Penile Zipper Injury. Separation of the zipper by cutting the median bar of the zipper with a bone cutter allows release. (Photo contributor: Kevin J. Knoop, MD, MS.)

Treatment is directed at removing the zipper and freeing the entrapped penile foreskin or prepuce while minimizing trauma and pain. A penile block with local injection at the base of the penis eases removal but might not be necessary if the child is cooperative and removal can be effected atraumatically. Several methods are described depending on the mechanism.

Pearls

1. Zipper entrapment of the penis is one of the most common genital injuries in prepubertal boys.
2. When tissue is entrapped by the zipper teeth only, release can be effected by cutting the cloth of the zipper either between the teeth or below the point of entrapment.
3. Cutting the median bar with a bone cutter allows the whole zipper to fall apart and release the entrapped skin.
4. Lateral compression of the distal zipper fastener with pliers may immediately release the tissue without need for anesthesia. This method requires application of equal pressure to both the anterior and posterior fastener plates simultaneously.
5. After removal, ensure that the urethra is patent and the child can void.



FIGURE 8.45 ■ Penile Zipper Injury. Crushed tissue after zipper removal did not obstruct urine flow in this patient. (Photo contributor: Kevin J. Knoop, MD, MS.)

VARICOCELE

Clinical Summary

A varicocele forms when the veins along the spermatic cord become engorged due to valve incompetence and is usually asymptomatic. Patients may complain of a dragging sensation or heaviness of the testicle, or notice the painless mass of

veins itself (bag of worms). It usually develops slowly by the third decade of life and is on the left side approximately 90% of the time.

Management and Disposition

Varicocele is generally harmless, but severe ones can affect fertility and cause testicular atrophy. Obtain an ultrasound if diagnosis is unclear. Refer to a urologist and, if symptomatic, prescribe scrotal support.

Pearls

1. Rapid appearance or presentation on the right side or at an age greater than 40 years should raise concern for extrinsic compression from an abdominal/pelvic mass or renal tumor.
2. Superior mesenteric artery compression of the left renal vein against the aorta (nutcracker syndrome) can present as a varicocele.
3. Varicocele forms on the left side because the left spermatic vein anastomosis to the renal vein is at 90 degrees, whereas the right-side anastomosis is considerably less acute and goes directly to the much larger inferior vena cava.



FIGURE 8.46 ■ Varicocele. Palpation of the nontender, twisted mass along the spermatic cord feels like a “bag of worms.” (Photo contributor: Dr. Paul Turek, www.TheTurekClinic.com.)



FIGURE 8.47 ■ Varicocele. A varicocele that led to infertility. (Photo contributor: Sandro Esteves, MD.)

PENILE IMPLANT COMPLICATIONS

Clinical Summary

Penile implants are typically undertaken when more conservative therapy for erectile dysfunction is either unacceptable to the patient or fails. The two main types are semi-rigid rods, which result in a permanent erection, and inflatable prostheses, which include cylinders placed in the corpora cavernosa and a pump in the scrotum. Erosion rarely can occur months or years after the placement of the prosthesis, and the rods or cylinders can erode through the urethral meatus or the pump and tubes can erode through the scrotum.

Management and Disposition

Any erosion of the device requires complete removal of the device and potential replacement. A urologist should be contacted in the emergency department, although the removal may not be emergent unless an infection is present. Infection may coexist, either as the etiology of the erosion or as a result of the erosion. Antibiotics and more urgent removal are indicated if an infection is present.

Pearls

1. The inflatable prosthesis typically has cylinders in the corpora cavernosa and the pump in the scrotum.
2. Any visible erosion of the device requires removal of the device.
3. Evaluate for concomitant infection as the etiology of or resulting from the erosion.



FIGURE 8.48 ■ Penile Implant Erosion. Inflatable prosthesis from penile implant eroding through the corpora cavernosa and out of the urethral meatus after eroding into the urethra. (Photo contributor: R. Jason)

Thurman, MD.)



FIGURE 8.49 ■ Penile Implant Erosion. A displaced penile implant with erosion of the pump and tubing through the scrotum is seen. (Photo contributor: Alan B. Storrow, MD.)

The authors acknowledge Jeffery D. Bondesson, MD, for his contributions in authoring the previous four editions of this chapter.

Chapter 9

SEXUALLY TRANSMITTED INFECTIONS AND ANORECTAL CONDITIONS

Suzanne Dooley-Hash
Nicholas W.C. Herrman



Acute Thrombosed External Hemorrhoid. Young male suffered sudden onset of rectal pain and bleeding after a bowel movement. (Photo contributor: Lawrence B. Stack, MD.)

SYPHILIS

Clinical Summary

Syphilis is caused by the spirochete *Treponema pallidum* and occurs in three distinct stages. Primary syphilis usually presents within 2 to 6 weeks of exposure as a single papule that develops at the site of inoculation. This papule turns into a painless ulcer with indurated margins and a nonexudative base known as a chancre. Longer incubation periods and multiple primary ulcers are seen less commonly. Regional bilateral lymphadenopathy is common. Ulcers resolve spontaneously within several weeks, but without treatment, the infection progresses to the secondary stage. Treatment will prevent progression of disease.



FIGURE 9.1 ■ Primary Chancre—Male. This dry-based, painless ulcer with indurated borders is typical for a primary chancre in a male patient. (Reproduced with permission from Wisdom A. *A Colour Atlas of Sexually Transmitted Diseases*. London, United Kingdom: Mosby-Wolfe Ltd; 1992. Copyright © Elsevier.)



FIGURE 9.2 ■ Primary Chancre—Male. Painless, solitary penile ulcer in a sexually active male with multiple partners. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 9.3 ■ Primary Chancre—Female. A solitary, painless genital chancre with a clean base in a patient with primary syphilis. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)



FIGURE 9.4 ■ Secondary Syphilis—Trunk. Rash on trunk due to secondary syphilis. (Photo contributor: Andrew Wong, MD.)

Secondary syphilis involves systemic dissemination of *T pallidum* and manifests several weeks to months after the primary chancre as a diffuse nonpruritic eruption consisting of pink, violaceous, or copper-colored macule and papules primarily on the trunk, palms, and soles. Mucous patches, condyloma lata, and patchy alopecia may also be present. Diffuse painless lymphadenopathy is common. These symptoms resolve within months, but if untreated, syphilis will persist as latent disease and/or tertiary syphilis may develop. Tertiary syphilis has serious cardiac and neurologic sequelae, a discussion of which is beyond the scope of this text.



FIGURE 9.5 ■ Secondary Syphilis—Palms. Papulosquamous eruption of secondary syphilis. Note the annular appearance of the palmar scales. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH, ed. *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)



FIGURE 9.6 ■ Secondary Syphilis—Palms. Resolving palmar eruption of secondary syphilis. (Photo contributor: Andrew Wong, MD.)



FIGURE 9.7 ■ Secondary Syphilis—Soles. Hyperkeratotic plan-tar rash in a patient with secondary syphilis. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH, ed. *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)



FIGURE 9.8 ■ Secondary Syphilis—Mucous Patches. Mucous patches seen on the proximal tongue are a manifestation of secondary syphilis and are highly infectious. Microscopic dark-field examination often reveals spirochetes. (Image appears with permission of VisualDx, www.visualdx.com.)

Management and Disposition

Diagnosis is established by serologic testing, both nontreponemal (rapid plasma reagin [RPR] and Venereal Disease Research Laboratory [VDRL]) and treponemal (detection of IgM and IgG antibodies) tests. Test for other sexually transmitted infections (STIs). Parenteral benzathine penicillin G is the preferred treatment for every stage. Use one dose of 2.4 million units intramuscularly (IM) for primary and secondary stages and 2.4 million units IM once weekly for 3 weeks for latent and tertiary syphilis. Doxycycline 100 mg orally twice daily for

14 days can be used for primary syphilis in penicillin-allergic patients. Pregnant patients with penicillin allergy should be referred for desensitization and subsequent treatment with penicillin G.

Refer for repeat testing in 6 to 12 months to confirm adequate response to treatment. Empirically treat any sexual partners from within 90 days and recommend serologic testing for any beyond 90 days. All cases must be reported to the appropriate public health agency.

Pearls

1. Consider dark-field microscopy examination of scrapings from lesions of the primary or secondary stage to rapidly confirm the diagnosis.
2. Cutaneous lesions of primary and secondary syphilis are highly infectious.
3. Warn patients of the Jarisch-Herxheimer reaction, an acute febrile illness that develops within the first 24 hours of treatment that is due to release of endotoxins from bacteria lysis. It is self-limited, and pretreatment with nonsteroidal anti-inflammatory drugs may attenuate the reaction.



FIGURE 9.9 ■ Secondary Syphilis—Condyloma Lata. Typical appearance of the smooth, broad-based hypopigmented flat plaques of condyloma lata, a manifestation of secondary syphilis. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH, ed. *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)



FIGURE 9.10 ■ Secondary Syphilis—Perianal Condyloma Lata. Painless, smooth, broad-based hypopigmented flat plaques of condyloma lata in a patient having unprotected anal sex. (Photo contributor: Jennifer Himmel Salch, DO).

GONORRHEA

Clinical Summary

Infection with *Neisseria gonorrhoeae* may occur at any exposed site and typically manifests within 2 to 7 days. Up to 10% of infections in men and 80% in women are asymptomatic. Urethritis, characterized by purulent urethral discharge and dysuria, is the most common manifestation in men. Although women may develop urethritis, cervicitis is more common. Symptoms may include vaginal irritation, discharge, or spotting, particularly with intercourse. Examination reveals a friable cervix, and a mucopurulent endocervical exudate may be present.

Disseminated gonococcal infection (DGI) occurs in 1% to 3% of patients and most commonly in females. Forty percent of DGI presents as monoarticular septic arthritis of the knee, wrist, ankle, or elbow. The other 60% of DGI presents as the arthritis-dermatitis syndrome where, in addition to arthritis, crops of pustules on a purpuric base are also present. Complications of DGI include myocarditis, hepatitis, endocarditis, and meningitis.

Gonococcal conjunctivitis, characterized by chemosis and copious purulent exudate, is most often seen in neonates but may occur at any age. In neonates, symptoms develop within 2 to 6 days of exposure at birth. Complications if untreated include endophthalmitis and perforation of the globe with permanent loss of vision.

Management and Disposition

Urethritis and cervicitis: Treat with single doses of both ceftriaxone 250 mg IM and oral azithromycin 1000 mg. Single-agent treatment is no longer recommended.

Conjunctivitis: Treat adults with ceftriaxone 1 g IM and oral azithromycin 1 g once. Oral doxycycline 100 mg twice daily for 7 days is an alternative. Treat infants and children over 2 months of age with ceftriaxone 25 to 50 mg/kg IM once. For infants less than 2 months old, use cefotaxime 100 mg/kg once. All neonates should receive empiric treatment with erythromycin ophthalmic ointment at birth.

DGI: Treat initially with ceftriaxone 1 g intravenously (IV) plus a single dose of azithromycin 1 g orally. Treat suspected endocarditis or meningitis with higher doses of ceftriaxone (1-2 g twice daily). Admit all patients with DGI for continued parenteral treatment for a minimum of 7 to 10 days followed by 7 to 10 days of an oral third-generation cephalosporin. All sexual partners should be treated empirically. Gonorrhea is a reportable disease.



FIGURE 9.11 ■ Male Gonococcal Urethritis. Purulent urethral discharge from a patient with gonococcal urethritis. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 9.12 ■ Female Gonococcal Urethritis. Gonococcal urethritis in a female patient. Note the purulent urethral discharge. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA, eds. *Atlas of Sexually Transmitted Diseases and AIDS*. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)

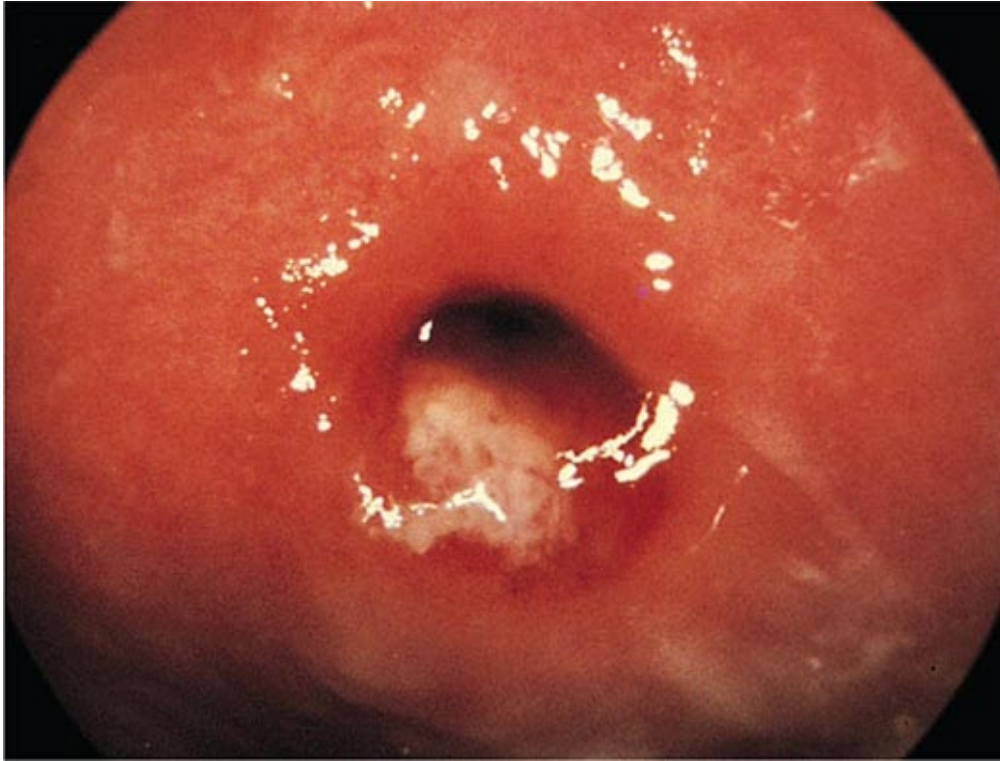


FIGURE 9.13 ■ Gonococcal Cervicitis. Endocervical purulent exudate in an asymptomatic patient with gonococcal cervicitis. The cervix is very friable. (Photo contributor: King K. Holmes, MD. From Handsfield HH, ed. *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)

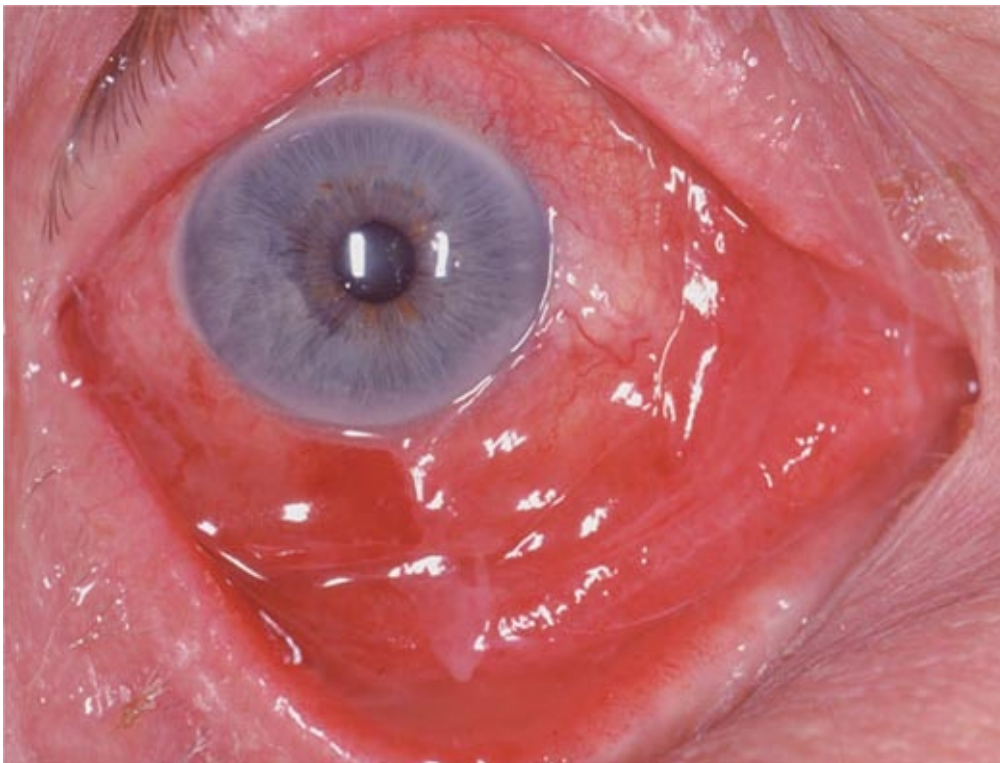


FIGURE 9.14 ■ Gonococcal Hyperacute Conjunctivitis. Chemotic conjunctiva and copious purulent

exudate in a patient with gonococcal conjunctivitis. (Photo contributor: Lawrence B. Stack, MD.)

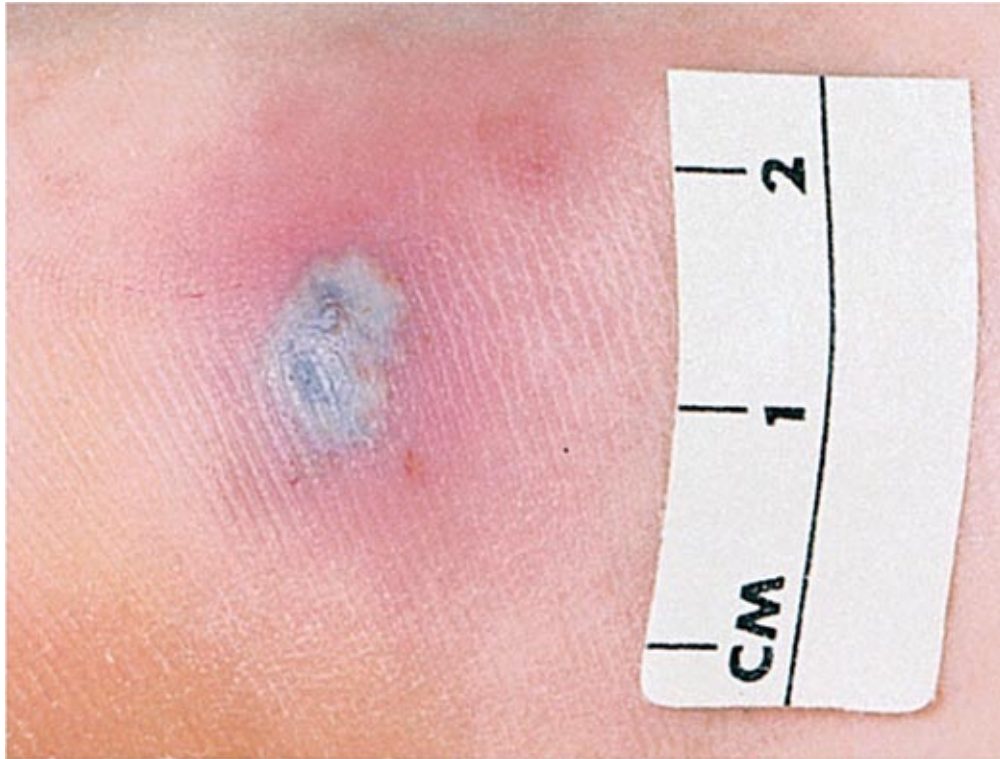


FIGURE 9.15 ■ Disseminated Gonococcal Infection—Skin Eruption. Hemorrhagic pustules suggestive of disseminated gonococcal infection. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH, ed. *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)



FIGURE 9.16 ■ Gonococcal Bartholin Abscess. Enlarged, fluctuant, tender Bartholin abscess of the labia, usually but not always a result of gonorrhea. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Coinfection with chlamydia is seen in 30% of men with urethritis and 50% of women with cervicitis. Treat patients with gonorrhea empirically for chlamydia.
2. Gonococcal arthritis is the most common cause of monoarticular arthritis in young, sexually active patients.
3. Cultures are the gold standard for confirming the diagnosis of DGI. Selective media should be used when specimens are obtained from the cervix, pharynx, urethra, or rectum. A nonselective medium (blood agar) should be used in culturing joint fluid, blood, or cerebrospinal fluid.

CHLAMYDIA

Clinical Summary

Chlamydia trachomatis infections are often asymptomatic. The most common manifestations are cervicitis in women and urethritis in men. Urethritis presents with dysuria and urethral discharge. Complications include epididymitis, which can cause infertility if untreated. Symptoms of cervicitis include vaginal irritation, discharge, or spotting, particularly with intercourse, and the cervix may appear friable. If untreated, pelvic inflammatory disease (PID) may develop, which can be complicated by tubo-ovarian abscess (TOA) formation. Fallopian tube scarring from TOA increases risk for future ectopic pregnancy and infertility. Exposed neonates may develop chlamydial conjunctivitis within 5 to 14 days of delivery. Symptoms include mild swelling and watery to mucopurulent discharge.

Management and Disposition

Preferred treatment for urethritis and cervicitis is a single dose of oral azithromycin 1 g. Doxycycline 100 mg orally twice daily for 7 days is equally effective but should not be used if there are concerns about medication compliance. Alternative regimens include 7 days of oral ofloxacin 300 mg twice daily or levofloxacin 500 mg once daily.

Treat uncomplicated PID with a 14-day course of oral doxycycline 100 mg twice daily in conjunction with a single IM dose of ceftriaxone 250 mg IM. Complicated PID with TOA or other sequelae requires gynecologic consultation for possible surgical intervention and admission for parenteral antibiotics. Treat neonatal chlamydial conjunctivitis with erythromycin 50 mg/kg per day divided four times daily for 14 days. All sexual partners should be notified and treated empirically. Chlamydia is a reportable disease.

Pearls

1. Due to common coinfection, empirically treat any patient with chlamydia for gonorrhea.
2. Chlamydia is not detected by routine urinalysis or culture. Therefore, dysuria with pyuria but without bacteriuria should prompt consideration of chlamydial infection in sexually active patients.



FIGURE 9.17 ■ Male Chlamydial Urethritis. Thin urethral discharge of chlamydial urethritis. (Photo contributor: Walter Stamm, MD. From Handsfield HH, ed. *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)

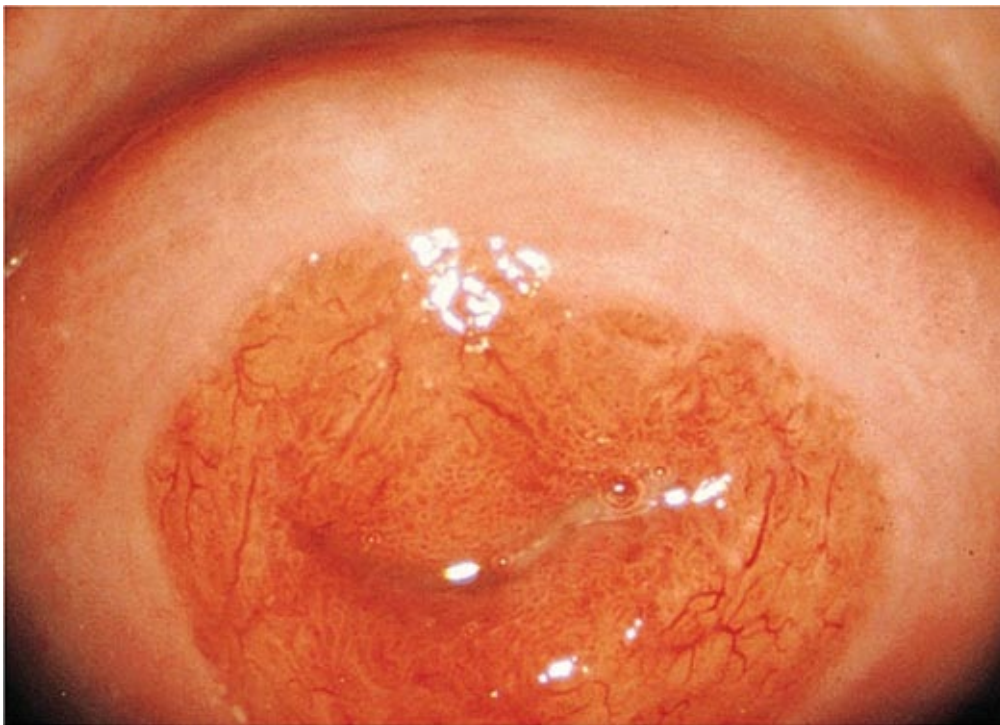


FIGURE 9.18 ■ Chlamydial Cervicitis. Mucopurulent cervicitis from chlamydial infection. (Photo contributor: Claire E. Stevens, PA. From Handsfield HH, ed. *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)



FIGURE 9.19 ■ Chlamydia Conjunctivitis. Follicles (focal areas of conjunctival inflammation) and a thin watery discharge are seen typical of chlamydia conjunctivitis. This is usually an indolent course lasting weeks to months. Follicles and inflammation are best seen on the upper tarsal conjunctiva. (Photo contributor: Jeffrey Goshe, MD.)

GENITAL HERPES

Clinical Summary

Genital herpes is caused by both herpes simplex virus (HSV) type 1 and 2. Primary infections are often asymptomatic but may present with a prodrome of fever, headache, myalgias, and dysuria followed by development of an eruption within a few days. The typical eruption consists of clusters of small vesicles that ulcerate into shallow painful ulcers with an erythematous base. The eruption can last for several weeks. Most primary infections are self-limited, but complications may include acute urinary retention, aseptic meningitis, encephalitis, hepatitis, and pneumonitis. Nonprimary infections occur when patients with preexisting antibodies to one HSV type are infected by the other type. Recurrent episodes are common. Both nonprimary first and recurrent episodes tend to have a milder course than primary infections. Immunocompromised patients are at risk for disseminated herpes and other complications. Perinatal transmission of genital herpes is associated with high

rates of infant morbidity and mortality.

Management and Disposition

Diagnosis of primary or nonprimary first infection is confirmed by viral culture from lesions or serum polymerase chain reaction testing. Recurrent episodes are diagnosed clinically.

Antiviral agents can decrease length and duration of symptoms and may be used for suppressive therapy, but no curative treatment is available. Treat *primary genital herpes* with oral acyclovir 400 mg three times daily or 200 mg five times daily for 7 to 10 days. Alternatives include famciclovir 250 mg three times daily or valacyclovir 1000 mg twice daily for 7 to 10 days.



FIGURE 9.20 ■ Herpetic Ulcerations—Female. Multiple coalescing vesicles and ulcers causing a deep ulcer. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.21 ■ Primary Genital Herpes—Female. Multiple superficial ulcerations of primary genital herpes. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.22 ■ Primary Lesions—Male. Multiple genital vesicles of primary genital herpes. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH, ed. *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)



FIGURE 9.23 ■ Primary Lesions—Male. Confluence of ulcerations on an erythematous base in a patient with primary herpes simplex type II. (Photo contributor: David Effron, MD.)



FIGURE 9.24 ■ Recurrent Genital Herpes Type 2—Male. Multiple umbilicated vesicles on an erythematous base adjacent to genital warts on the mons pubis of a male who typically wears condoms during sex. (Photo contributor: Lawrence B. Stack, MD).

For *recurrent genital herpes*, treatment is most effective if started within 24 hours of symptom onset. Multiple antiviral treatment regimens are appropriate, with the most commonly used being 3- to 5-day courses of oral acyclovir 400

mg three times daily or 800 mg twice daily, famciclovir 125 mg twice daily, or valacyclovir 1000 mg once daily.

Admit patients with disseminated herpes infections and other complications for treatment with IV antiviral medications.

All patients should be counseled on the chronic, recurrent nature of the illness and the risk to sexual partners. Women should be advised to inform their obstetricians if they become pregnant.

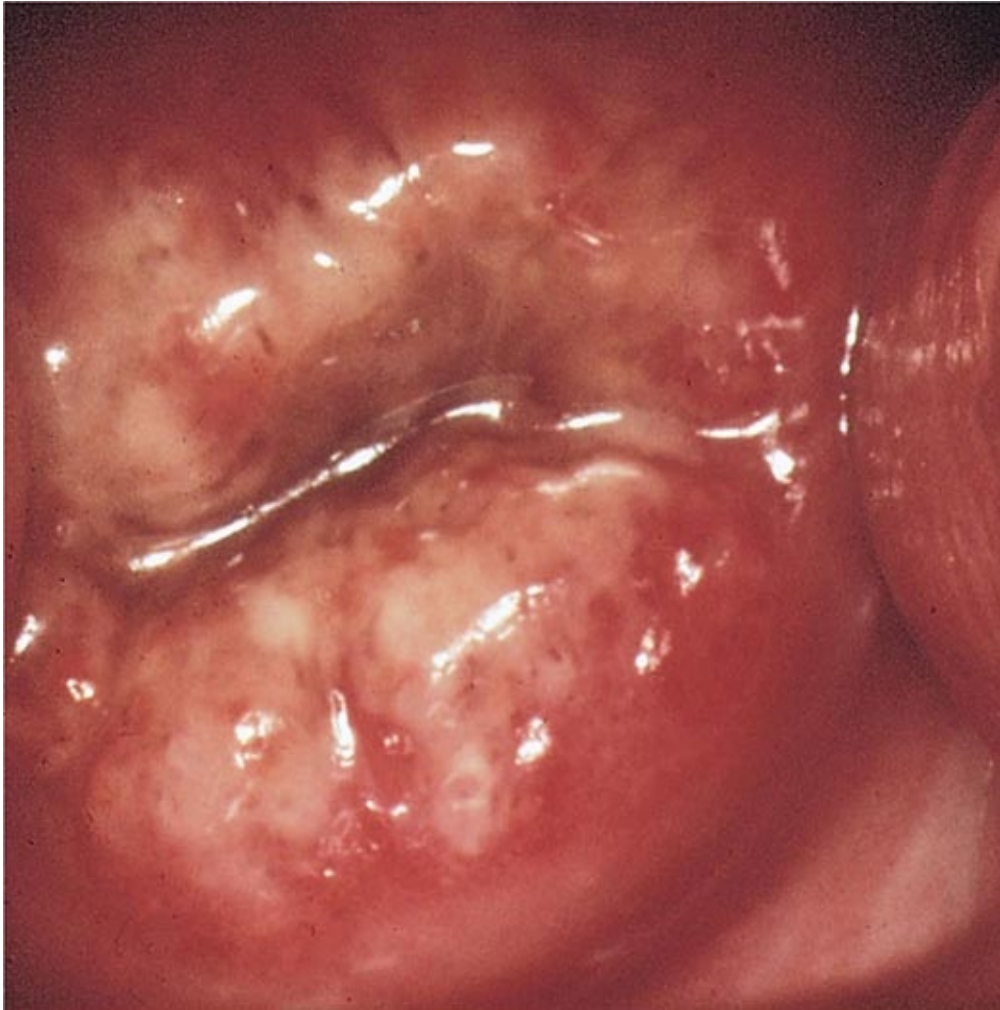


FIGURE 9.25 ■ Herpes Simplex Virus—Cervix. Erosive ulcerations of the cervix in a patient with genital herpes infection. This patient may be completely asymptomatic and may transmit the disease. (Reproduced with permission from Wisdom A. *A Colour Atlas of Sexually Transmitted Diseases*. London, United Kingdom: Mosby-Wolfe Ltd; 1992. Copyright © Elsevier.)

Pearls

1. Genital herpes is the most common cause of ulcerating genital lesions in the

- developed world.
2. The Tzanck smear has limited utility due to low sensitivity and specificity.
 3. Topical antiviral agents are not effective against genital herpes.

LYMPHOGRANULOMA VENEREUM

Clinical Summary

Lymphogranuloma venereum (LGV) is caused by the L1, L2, and L3 serotypes of *C trachomatis* and primarily affects lymphatic tissue. Primary LGV causes a self-limited painless genital ulceration that may not be noticed by the patient. The presence of pain and/or surrounding erythema, warmth, or tenderness of the area should raise concerns for an alternative diagnosis. Secondary LGV occurs several weeks later and causes painful femoral or inguinal lymphadenopathy. This may occur both above and below the inguinal ligament, causing the “groove sign” suggestive of LGV. Enlarged lymph nodes, or buboes, may spontaneously rupture. Patients with rectal exposure may present with rectal pain, drainage, and tenesmus due to proctocolitis, which may be mistaken for inflammatory bowel disease. Untreated disease can cause sinus tracts to skin, anogenital fibrosis, and strictures.

Management and Disposition

Treat with doxycycline 100 mg orally twice daily for 21 days. Alternatives are erythromycin 500 mg orally four times daily for 21 days or azithromycin 1 g orally once weekly for 3 weeks. Buboes should be aspirated or incised and drained to prevent rupture and subsequent fistula formation. Send sample of drainage for culture if diagnosis is in question. Asymptomatic sexual partners should be empirically treated with a single dose of azithromycin 1000 mg orally or doxycycline 100 mg orally twice daily for 7 days. Symptomatic sexual partners should undergo the full treatment course.



FIGURE 9.26 ■ Lymphogranuloma Venereum. Unilateral left lymphadenopathy in a patient with lymphogranuloma venereum. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.27 ■ Lymphogranuloma Venereum—Groove Sign. The appearance of the “groove sign” is due to inguinal node enlargement above and femoral node enlargement below Poupart’s ligament and is seen in 33% of patients with LGV. (Image appears with permission of VisualDx, www.visualdx.com.)

Pearls

1. LGV is typically a clinical diagnosis initially; although *C trachomatis* testing is widely available, specific serotype testing is not.
2. Concurrent infection with other STIs is common.
3. Delayed diagnosis and treatment can result in sequelae such as genital elephantiasis, anal fistulae and strictures, and infertility.

CHANCROID

Clinical Summary

Chancroid is caused by *Haemophilus ducreyi*. Following a 4- to 10-day incubation period, patients develop erythematous papules that evolve into pustules. The pustules then erode forming painful ulcers. Differential diagnosis

should include other causes of genital ulcerations including genital herpes and syphilis. Painful, suppurative inguinal lymphadenopathy develops in almost 50% of cases. Large infected lymph nodes may spontaneously rupture. Systemic symptoms are uncommon.

Management and Disposition

Treat with a single dose of ceftriaxone 250 mg IM or oral azithromycin 1000 mg. Ciprofloxacin 500 mg orally twice daily for 3 days is an alternative. Refer for follow-up examination in 7 days to evaluate response to treatment. Aspirate or incise and drain large fluctuant buboes to prevent spontaneous rupture and fistula tract formation. Sexual partners should be empirically treated.

Pearls

1. Chancroid is a clinical diagnosis as culture is technically difficult and not widely available.
2. The ulcerative lesions of chancroid are very tender and usually multiple.
3. Chancroid is less common than genital herpes and syphilis. Consider concurrent treatment for these infections as definitive testing for chancroid may not be available.
4. Methicillin resistant *Staphylococcus aureus* (MRSA) penile shaft infections may mimic STIs.



FIGURE 9.28 ■ Chancroid Lesions. Multiple painful, deep ulcerations of chancroid. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH, ed. *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York: McGraw Hill; 2011.)



FIGURE 9.29 ■ Chancroid. Painful ulcerations on an erythematous base of the glans and penile shaft in a sexually active male. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.30 ■ Methicillin-Resistant *Staphylococcus aureus* (MRSA) Infection of the Penis. Lesion from MRSA infection of penis mimics LGV, syphilis, herpes, and chancroid. (Photo contributor: Adam Nicholson, MD.)

CONDYLOMA ACUMINATA (GENITAL WARTS)

Clinical Summary

Genital warts are caused by human papillomavirus (HPV). Typical eruptions are flesh-colored with a cauliflower-like appearance, but they may also be flat, sessile, or pedunculated. They can develop anywhere in the anogenital area.

Although usually asymptomatic, they may be pruritic. They range in size from a few millimeters to several centimeters.

Management and Disposition

Genital warts typically do not require emergent treatment, and patients may be referred for outpatient follow-up. Almost half of lesions will resolve without treatment, usually over the course of several months. Topical caustic agents can be prescribed if treatment is initiated in the emergency department. Options include imiquimod cream (applied three times weekly for up to 16 weeks), podophyllotoxin (applied twice daily for 3 days followed by no therapy for 4 days; repeat cycle up to four times), and sinecatechins (applied three times daily for up to 16 weeks).



FIGURE 9.31 ■ Genital Warts—Female. Verrucous lesions of the posterior fourchette in a patient with condyloma acuminata. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH, ed. *Atlas of*



FIGURE 9.32 ■ Genital Warts—Male. Typical appearance of condyloma acuminata of the penis. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA, eds. *Atlas of Sexually Transmitted Diseases and AIDS*. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)



FIGURE 9.33 ■ Genital Warts—Male. Cauliflower-like appearance of condyloma acuminata of the foreskin of this uncircumcised male. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.34 ■ Perianal Condyloma Acuminata. Multiple peri-anal pedunculated warts are seen in this patient. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 9.35 ■ Giant Warts—Female. Extensive condyloma acuminata in a female. (Photo contributor: Hope Haefner, MD.)



FIGURE 9.36 ■ Genital Warts—Child. Condyloma in female child should raise suspicion for sexual abuse. (Photo contributor: Hope Haefner, MD.)



FIGURE 9.37 ■ Giant Warts—Male. Giant warts in a male patient with extensive condyloma acuminata. (Reproduced with permission from Wisdom A. *A Colour Atlas of Sexually Transmitted Diseases*. London, United Kingdom: Mosby-Wolfe Ltd; 1992. Copyright © Elsevier)



FIGURE 9.38 ■ Genital Wart. Single verrucous eruption of condyloma acuminata in the perianal area. (Photo contributor: Lawrence B. Stack.)

Advise patients that recurrence is common and multiple treatments may be needed to completely eradicate the warts. Refer for appropriate follow-up care and consideration of other therapies, which include cryotherapy, electrocautery, trichloroacetic acid, and laser ablation for extensive disease. Patients should be evaluated for other STIs. HPV infection is associated with increased risk of cervical cancer. Refer women with genital warts for gynecologic evaluation.

Pearls

1. The incidence of HPV is decreasing in the United States due to immunization of young women against HPV, although it remains the most common STI in the world.
2. Genital warts are considered a sexually transmitted condition. Their presence in a child should raise suspicion for child sexual abuse.
3. Trichloroacetic acid and cryoablation are the preferred treatments in pregnant patients.

PEDICULOSIS PUBIS

Clinical Summary

Pediculosis pubis is caused by the crab louse, *Pthirus pubis*. The lice are 0.8 to 1.2 mm long and difficult to see. The egg form, the nit, adheres to hair and clothing. Transmission occurs both by sexual contact and less frequently by fomites, such as clothing and towels. Pruritis due to hypersensitivity is typically the primary clinical manifestation. Small blue macules (maculae ceruleae) may also develop secondary to louse anticoagulant saliva injection during feeding. Patients may also notice small blood stains on the undergarments secondary to bleeding from louse bites.

Management and Disposition

Diagnosis is made by demonstration of lice or nits. This can be done either visually or by microscopic examination if the diagnosis is uncertain. Treat with topical permethrin 1% cream or pyrethrins 0.33% with piperonyl butoxide 4%. Either agent should be washed off after 10 minutes. Nits should be removed with a comb or tweezers. Repeat treatment in 1 week should be considered but is not uniformly recommended. Alternative regimens include topical malathion 0.5% lotion washed off after 8 to 12 hours or oral ivermectin 250 µg/kg once weekly for 2 weeks. Advise patients to wash all clothing and linens used in the preceding 24 hours and to avoid sexual contact until cured. All sexual partners should be screened.

Pearls

1. Nits are easier to find on examination than are mature lice; the average number of lice in an infestation is only 10.
2. Although an effective treatment, lindane is no longer recommended as first-line therapy due to concerns regarding toxicity.
3. Evaluate patients with pediculosis pubis for other STIs.



FIGURE 9.39 ■ *Pthirus pubis*—On Pubic Hair. *Pthirus pubis*, or the crab louse, in the pubic hair (pediculosis pubis) of a patient complaining of itching. Note the nits attached to the hair. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA, eds. *Atlas of Sexually Transmitted Diseases and AIDS*. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)

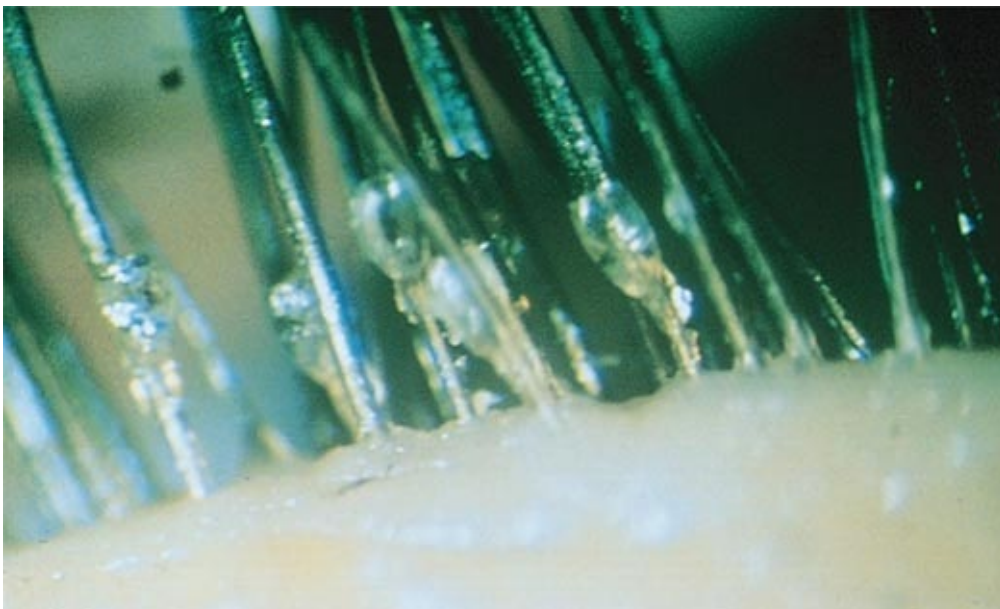


FIGURE 9.40 ■ *Pthirus pubis*—On Eyelashes. *Pthirus pubis* lice noted in the eyelashes. (Reproduced with permission from Spalton DJ, Hitchings RA, Hunter PA, eds. *Atlas of Clinical Ophthalmology*, 2nd ed.

ANAL FISSURE

Clinical Summary

An anal fissure is a longitudinal tear of the skin of the anal canal that extends from the dentate line to the anal verge. It is the most common cause of painful rectal bleeding. Fissures are thought to be caused by the passage of hard or large stools with constipation, but may also be seen with diarrhea. Incidence follows a bimodal distribution with fissures most commonly seen in infants and middle-aged adults. Patients present with intense sharp, burning pain during and after bowel movements. Pain may be accompanied by bright red blood. Gentle examination with separation of the buttocks usually provides adequate visualization. Anal fissures are typically a few millimeters wide and most commonly occur in the posterior midline where the skeletal muscle fibers encircling the anus are weakest.

Management and Disposition

Treat initially with supportive measures, including increased dietary fiber and fluids, sitz baths, topical analgesics such as lido-caine 2% jelly, and topical vasodilators such as nifedipine 0.2% ointment or nitroglycerin 0.2% ointment. Second-line therapies include topical diltiazem 2% gel as well as oral nifedipine or diltiazem. Most simple anal fissures resolve in 2 to 4 weeks, and surgical management is typically reserved for refractory cases.

Pearls

1. Pain and involuntary sphincter spasm may preclude bedside digital or anoscopic examination, and anesthesia may be required for adequate evaluation.
2. An anal fissure off the midline should prompt consideration of secondary causes, such as inflammatory bowel disease, STI, or rarely leukemia, sickle cell disease, or anal neoplasm.
3. Anal fissures are the most common cause of painful rectal bleeding.



FIGURE 9.41 ■ Anal Fissure. A typical anal fissure located in the posterior midline. (Photo contributor: Paul J. Kovalcik, MD.)



FIGURE 9.42 ■ Anal Fissure. An anal fissure is seen at the superior midline in a patient with 2 weeks of constipation. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 9.43 ■ Anal Fissure. This adolescent patient complains of bloody, painful, hard stools and has an anal fissure in the midline toward the perineum. (Photo contributor: Lawrence B. Stack, MD.)

ANORECTAL ABSCESS

Clinical Summary



FIGURE 9.44 ■ Perianal Abscess. Swelling and erythema around the anus consistent with a perianal abscess. (Photo contributor: The American Society of Colon and Rectal Surgeons.)



FIGURE 9.45 ■ Perirectal Abscess—CT. Hypodense fluid collection surrounded by enhancing ring in the right perirectal tissue suggests an abscess. (Photo contributor: Brett Bechtel, MD.)

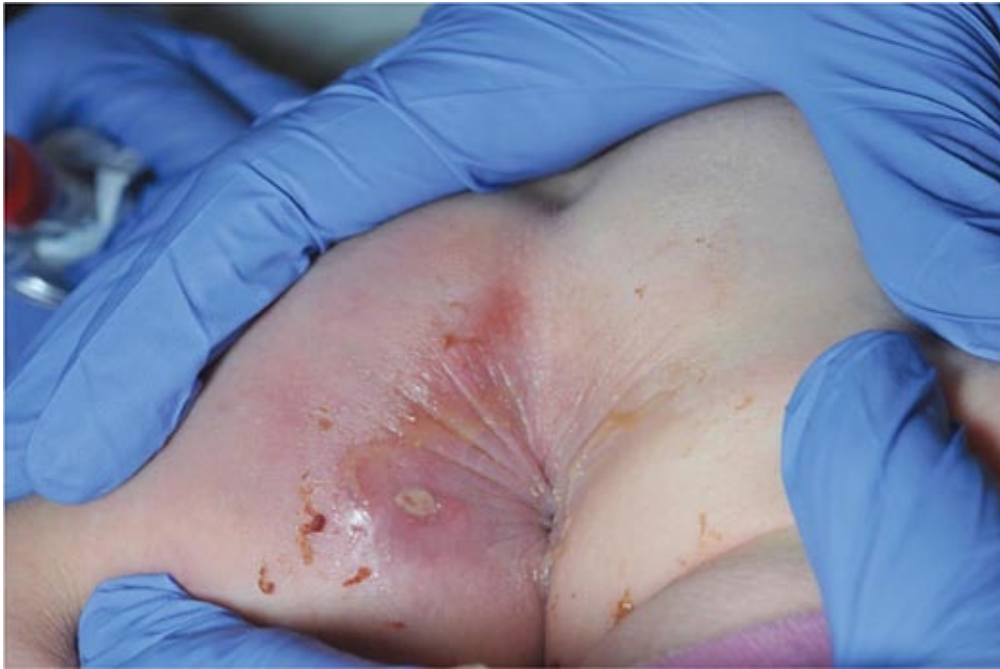


FIGURE 9.46 ■ Draining Perianal Abscess. A draining, left peri-anal abscess in a febrile infant. (Photo contributor: Lily Yu, MD.)



FIGURE 9.47 ■ Perineal Abscess and Cellulitis. A large perineal abscess is seen extending from the perianal tissues to the right labia in a female infant. (Photo contributor: Lawrence B. Stack, MD.)

Anorectal abscesses include perianal and perirectal abscesses, which are named according to the involved space: ischiorectal, intersphincteric, supralelevator, and horseshoe. These are most commonly found in males aged 30 to 50 years old

and occur due to occlusion of the mucus-producing anal crypt glands. Patients typically present with severe pain in the perianal area. Constitutional symptoms such as fever and malaise may also be present. Examination will often reveal an area of erythema and perianal fluctuance. Deeper perirectal abscesses may only exhibit tenderness and fluctuance with digital rectal examination. Predisposing conditions include Crohn's disease, chronic steroid use, diabetes mellitus, malignancy, radiation fibrosis, and trauma.

Management and Disposition

Incise and drain perianal abscesses using a small cruciate incision lateral to the external sphincter as close to the anal verge as possible. For an uncomplicated abscess, this can be accomplished under local anesthesia. All patients require outpatient follow-up. Antibiotic therapy is advised in immunosuppressed patients and those with systemic symptoms or overlying cellulitis. Commonly used antibiotic regimens are amoxicillin-clavulanate or the combination of ciprofloxacin and metronidazole. Obtain contrast-enhanced computed tomography for suspected deep perirectal abscesses that are not palpable on rectal examination, especially in patients with systemic symptoms. Obtain surgical consultation for large or complicated abscesses or those requiring examination and treatment under anesthesia.

Pearls

1. Consider underlying Crohn's disease in cases of recurrent anorectal abscesses.
2. All patients warrant surgical follow-up due to the high incidence of fistula formation.



FIGURE 9.48 ■ Perirectal Abscess. This large, spontaneously draining perirectal abscess required further surgical debridement in the operating room. (Photo contributor: Lawrence B. Stack, MD.)

HEMORRHOIDS

Clinical Summary

In the rare case of severe life-threatening bleeding, initiate fluid resuscitation and clamp or ligate the bleeding vessel. Otherwise, conservative treatment is

recommended for initial management. Options include increased dietary fiber and fluid intake, warm sitz baths, and topical analgesics. Advanced cases may require surgical consultation and treatment. Emergency department treatment of thrombosed external hemorrhoids includes an elliptical excision and extrusion of the clot under local anesthesia.

Management and Disposition

Internal hemorrhoids originate proximal to the dentate line and have visceral innervation. They commonly present with painless, bright red rectal bleeding after defecation. This results from the passage of stool over the thin-walled venules, causing abrasions and bleeding. Patients may also complain of a sensation of anal fullness from prolapse of the vessel.

External hemorrhoids originate distal to the dentate line, and since they have somatic innervation, they often cause significant pain. External hemorrhoids can also present with bright red rectal bleeding. Symptoms may also include swelling and pruritis. Thrombosis is much more common with external hemorrhoids and results in severe pain in the acute phase.

Factors contributing to hemorrhoid formation include constipation, obesity, increased intra-abdominal pressure from pregnancy or ascites, and family history. Differential diagnosis includes anorectal abscess, inflammatory bowel disease, malignancy, local trauma, herpes or other STI, rectal polyp, and rectal prolapse.

Pearls

1. Patients with other serious anorectal conditions may complain of “hemorrhoids.” Careful consideration of other potential causes of symptoms is critical.
2. Having the patient strain during the examination may reveal bleeding or prolapse of an internal hemorrhoid that might otherwise go unnoticed. Anoscopy can also be helpful.
3. Thrombosed hemorrhoids should not be excised in pregnant, immunocompromised, or pediatric patients.



FIGURE 9.49 ■ External Hemorrhoids. Multiple engorged, thrombosed (dark blue hue) external hemorrhoids are seen in this patient with anal pain and bleeding. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.50 ■ Evacuation of Thrombosed Hemorrhoid. (A) Painful 1.5-cm thrombosed hemorrhoid. (B) Infiltration with marcaine till blanches. (C) Clot delivery after incision. (D) Collapsed hemorrhoid after clot evacuation. (Photo contributor: Lawrence B. Stack, MD.)

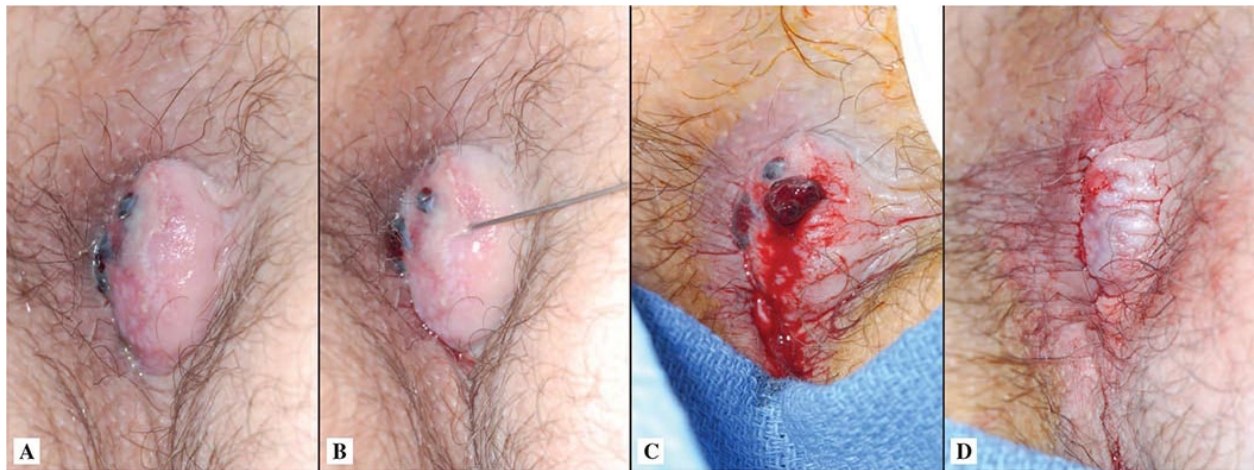


FIGURE 9.51 ■ Bleeding External Thrombosed Hemorrhoid. Engorged painful bleeding thrombosed hemorrhoids in a patient with chronic constipation. (Photo contributor: Department of Emergency Medicine, Naval Medical Center, Portsmouth, VA.)

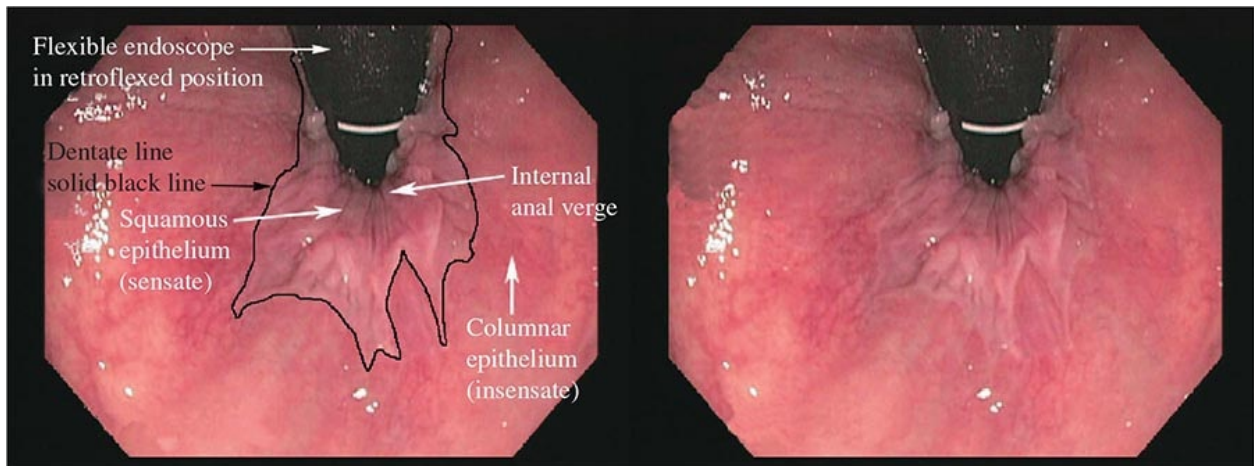


FIGURE 9.52 ■ Normal Distal Rectum Surface Anatomy. Columnar epithelium lines the colon (rectum), while squamous epithelium lines the anal canal and extends into the distal rectum. The columnar-squamous junction (interface) is termed the dentate line. The columnar epithelium is insensate, while the squamous epithelium is sensate. (Photo contributor: Philip E. Stack, MD.)

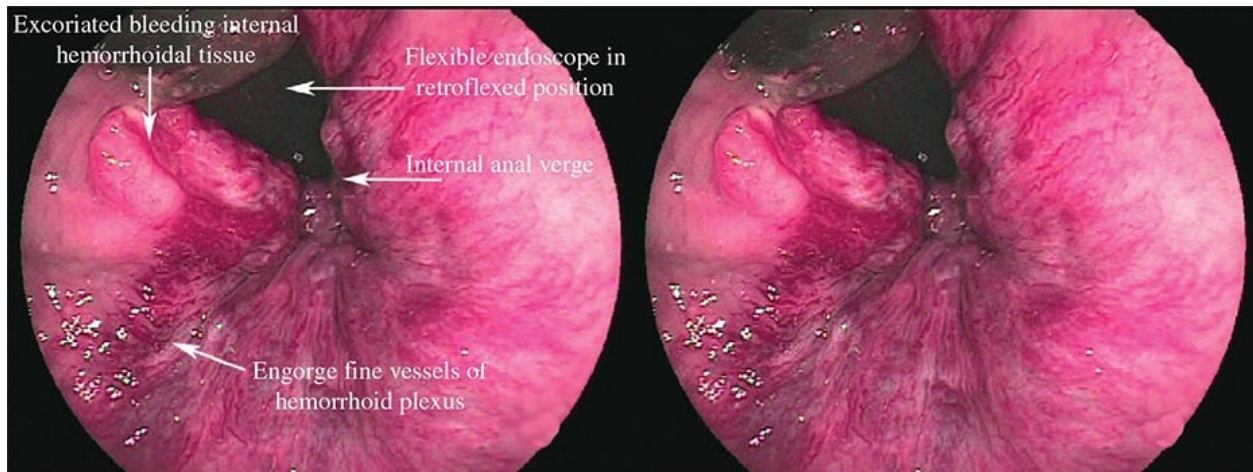


FIGURE 9.53 ■ Bleeding Internal Hemorrhoids. Middle-aged male with painless hematochezia. Internal hemorrhoidal bleeding is usually painless with a bleeding character that is described as “dripping or like a spray.” Moderate hematochezia outside of anticoagulation rarely results in any hemodynamic compromise. Treatment of predisposing underlying condition(s)—constipation, diarrhea, straining, poor dietary fiber intake, prolonged sitting and lifting, etc—is paramount; otherwise, hemorrhoids are likely to recur or become chronic. (Photo contributor: Philip E. Stack, MD.)

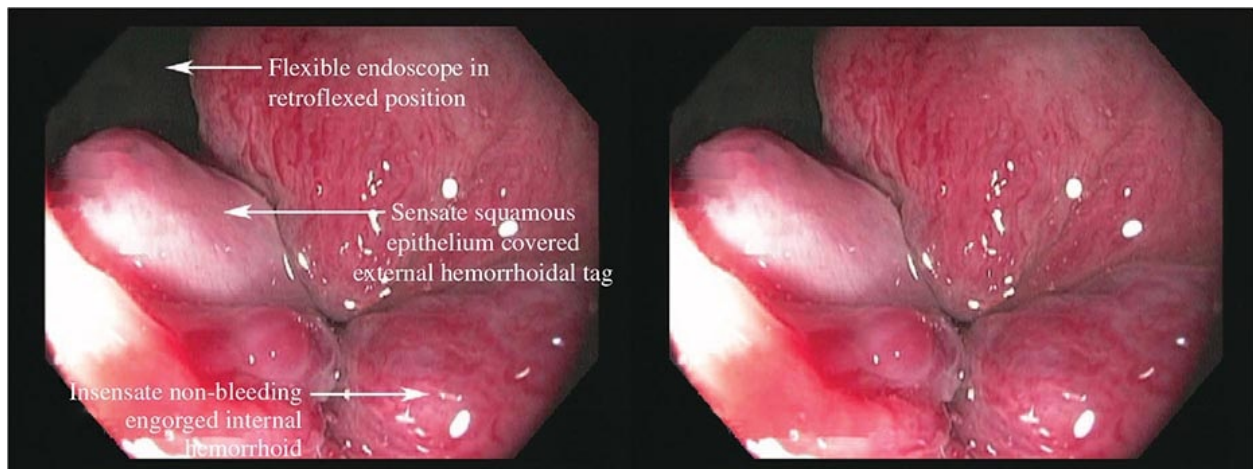


FIGURE 9.54 ■ External and Internal Hemorrhoids. Middle-aged woman with hemorrhoids and intermittent bloody stools and tender pro-lapsing small mass after bowel movements, which she manually reduces. Acute engorgement of external hemorrhoids can result in residual redundant tissue that is prone to re-engorgement. This sequence can result in the formation of a “tag or tissue” of squamous epithelium, which can be tender. This tissue, once formed, is not amenable to medical therapies and would require endoscopic or surgical removal if deemed necessary. (Photo contributor: Philip E. Stack, MD.)

RECTAL PROLAPSE

Clinical Summary

Rectal prolapse, also called rectal procidentia, occurs when anorectal tissue protrudes through the anus. Prolapse may be partial, involving only the mucosa, or complete, involving all layers of the rectal wall. Prolapse may result from laxity of the pelvic floor, weak anal sphincters, or lack of mesorectal fixation. Patients are often at extremes of age and may present with altered bowel habits, rectal mass, straining with bowel movements, and mucus discharge. Risk factors include multiparity, vaginal delivery, chronic constipation, and cystic fibrosis. Rectal prolapse is typically painless. Pain as a presenting symptom should prompt consideration of other diagnoses, such as rectal foreign body, neoplastic process, anorectal abscess, rectal polyp, or external hemorrhoids.

Management and Disposition

Firm persistent manual pressure will reduce most cases. Granulated sugar may be applied to the prolapsed tissue as an osmotic agent to reduce edema to aid reduction. If manual reduction fails, surgical consultation and operative reduction are indicated. Surgical consultation is also indicated with a complete prolapse. Refer all patients for anoscopic and sigmoidoscopic examination to evaluate for secondary causes.

Pearls

1. Rectal prolapse is commonly seen in children with cystic fibrosis. All children with rectal prolapse should undergo a sweat chloride test.
2. Examination of rectal prolapse reveals concentric mucosal rings and a sulcus between the anal canal and the rectum. Prolapsed hemorrhoids are separated by radial grooves and the sulcus is absent.

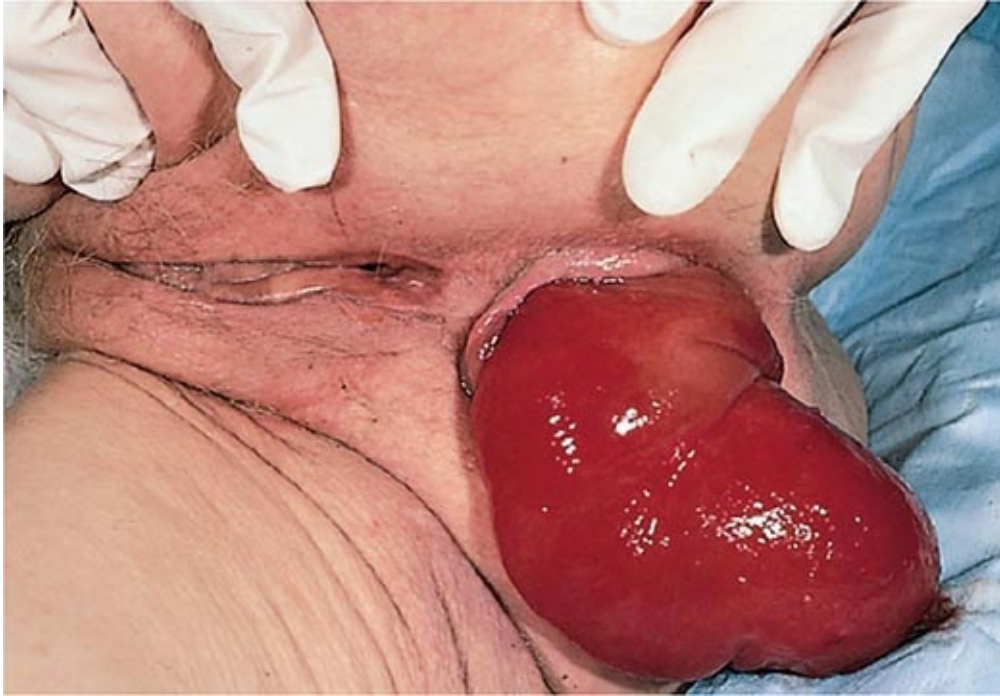


FIGURE 9.55 ■ Prolapsed Rectum. The rectum is completely pro-lapsed in this elderly patient. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 9.56 ■ Prolapsed Rectum. Recurrent rectal prolapse due to chronic constipation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.57 ■ Prolapsed Rectum—Infant. Two-month-old with rectal prolapse after repeated episodes of straining at stool. Rectal biopsy performed because of abnormal barium enema. Biopsy results negative for Hirschsprung disease. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.58 ■ Prolapsed Rectum. Large, complete, nonedematous rectal prolapse in a nursing home patient with recurrent prolapse. (Photo contributor: Kevin J. Knoop, MD, MS.)

PILONIDAL ABSCESS

Clinical Summary

Pilonidal abscesses typically occur at the superior aspect of the gluteal fold and

are more common in teenage and young adult males. Patients present with pain, swelling, and drainage, but usually do not have systemic symptoms. They are thought to occur when bacteria enter the hair follicle and cause edema that obstructs the opening to the skin surface. Eventually the follicle may rupture, which allows spread into the subcutaneous fatty tissue and abscess formation. Acute abscesses typically contain mixed organisms, including *Staphylococcus aureus* and *Streptococcus* species. Anaerobes and gram-negative organisms may also be present. Cellulitis in the sacrococcygeal area may result from a simple abscess or furuncle. Consider alternative causes such as anal fistulae, hidradenitis, inflammatory bowel disease, or tuberculosis.



FIGURE 9.59 ■ Pilonidal Abscess. Redness, fluctuance, and tenderness in the gluteal cleft seen with a pilonidal abscess. (Photo contributor: Louis La Vopa, MD.)

Management and Disposition

An acutely fluctuant abscess requires incision and drainage under local anesthesia. Instruct the patient on careful wound care and sitz baths. Antibiotic therapy is indicated in immunocompromised patients or those with significant surrounding cellulitis or systemic symptoms. Pilonidal abscesses have a high recurrence rate. Refer chronic or recurrent cases to a surgeon for evaluation for operative management.

Pearls

1. Pilonidal abscesses almost always occur in the midline but can have sinus tracts extending off the midline.
2. Pilonidal disease is three times more common in men than in women.



FIGURE 9.60 ■ Pilonidal Abscess. Close-up of the cutaneous manifestations of a large pilonidal abscess. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.61 ■ Pilonidal Abscess with Cellulitis. Middle-aged male with recurrent pilonidal abscess and surrounding cellulitis. (Photo contributor: Lawrence B. Stack, MD.)

RECTAL FOREIGN BODY

Clinical Summary

Diagnosis of rectal foreign body is usually made by history and confirmed by digital examination. The foreign body is usually directly inserted into the rectum, but an ingested foreign body may also become trapped in the rectum. The most common and serious complication of a rectal foreign body is perforation of the rectum or sigmoid colon. Perforation superior to the peritoneal reflection is associated with intraperitoneal free air and peritoneal signs. Perforation inferior to the peritoneal reflection often causes retroperitoneal injuries and presents with more nonspecific complaints. Determine the size, shape, and number of objects to assess the risk of perforation. In children, rectal foreign bodies usually present as rectal bleeding. Imaging is indicated if there are significant concerns for perforation.

Management and Disposition

The patient must be carefully evaluated for evidence of perforation by detailed examination and radiographic studies as indicated. Obtain an immediate surgical consultation if perforation is present or suspected. Without perforation blunt objects may be manually removed in the emergency department with adequate local anesthesia and sedation. Surgical consultation for removal of sharp objects under proctoscopic or sigmoidoscopic visualization is recommended. If the risk of perforation appears high or adequate relaxation and anesthesia cannot be obtained, removal should proceed under general anesthesia.

Pearls

1. A urinary catheter or small endotracheal tube may be passed proximal to some foreign bodies. This may release the vacuum effect of the rectum and the balloon may be inflated to aid in removal.
2. A rectal foreign body in a child should raise the suspicion of abuse.
3. Imaging after removal is needed to rule out perforation resulting from the procedure.
4. Pelvis imaging is recommended prior to rectal exam in incarcerated patients to prevent injury from sharp objects often hidden in this location.



FIGURE 9.62 ■ Rectal Foreign Body. This foreign body (a 7-oz beer bottle) required removal in the operating room. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 9.63 ■ Rectal Foreign Body. This victim of assault had a stick forced into his rectum. After removal, sigmoidoscopy revealed a retained small broken stick. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 9.64 ■ Rectal Foreign Body. Ventriculoperitoneal shunt tubing seen protruding from the anus after eroding through the trans-verse colon. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.65 ■ Ventriculoperitoneal Shunt Tubing in Colon. Ventriculoperitoneal shunt tubing seen in the transverse colon and proceeding down the distal colon and exiting the anus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.66 ■ Rectal and Genitourinary Foreign Body. One handcuff is encircling the genitalia (**A**), whereas the other is placed into the rectum (**B**). (Photo contributor: Christopher L. Stark, DO.)

RECTAL CANCER

Clinical Summary

Rectal cancer is often asymptomatic, especially in early stages. It may also present with rectal mass, pain or bleeding, pain with passage of stool, anemia, or weight loss. More than 90% of cases occur in people over 50 years, and risk increases with age. Males are affected more commonly than females. Other risk factors include inflammatory bowel disease, family history of colorectal cancer or polyps, hereditary cancers such as familial adenomatous polyposis or hereditary nonpolyposis colorectal cancer (Lynch syndrome), low-fiber diet, and tobacco and alcohol use. There are multiple types of rectal cancers, with adenocarcinoma being the most common.

Management and Disposition

Management of rectal cancer is beyond the scope of this book. In the emergency department, if active bleeding is present, place two large-bore IVs and apply pressure to any visible bleeding lesions. Obtain abdominal x-rays if symptoms of bowel obstruction (vomiting, abdominal pain, obstipation) are present. Unstable patients and those with active bleeding should be admitted. For those with the incidental finding of rectal mass or less severe symptoms, consider computed tomography to further evaluate. If stable for discharge, it is imperative to ensure rapid outpatient follow-up with a gastroenterologist for colonoscopy and further evaluation.

Pearls

1. Colorectal cancer is the 3rd most common cancer in adults in the United States. Rectal cancer accounts for one-third of all colorectal cancers.
2. Rectal polyps are potentially precancerous. All patients with polyps identified in the emergency department should be referred for rapid outpatient gastrointestinal follow-up regardless of symptoms.



FIGURE 9.67 ■ **Rectal Cancer.** Signet ring cell carcinoma of the rectum. (Photo contributor: Christopher L. Stark, DO.)

The authors wish to thank Diane M. Birnbaumer, Lynn K. Flowers, and Brian R. Sharp for their contributions to prior editions.

Chapter 10

GYNECOLOGIC AND OBSTETRIC CONDITIONS¹

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Nuchal Cord. Hand placement prior to applying downward traction to deliver the anterior shoulder. A loose nuchal cord is seen. (Photo contributor: William Leininger, MD.)

Gynecologic Conditions

VAGINITIS

Clinical Summary

Candidal vaginitis may present with vulvar erythema, pruritus, and/or burning and is characterized by a thick, clumping, white discharge. Risk factors include oral contraceptive, antibiotic, or corticosteroid use; pregnancy; and diabetes or other immuno-compromising conditions. It is uncommon in postmenopausal women and is not typically a sexually transmitted infection (STI). Diagnosis is confirmed with a wet mount slide prepared with 10% potassium hydroxide (KOH) that shows characteristic branched chain hyphae and spores.

Trichomonas vaginitis presents as a gray/green, thin, foul-smelling discharge with varying amounts of erythema and inflammation. Diagnosis by rapid antigen tests and nucleic acid amplification has generally replaced saline wet mount microscopy, which is less than 60% sensitive. Multiple petechiae on the vaginal wall or cervix (strawberry spots/strawberry cervix) are pathognomonic but not common.

Bacterial vaginosis (BV) also presents with a gray, thin, mal-odorous discharge, typically without other vaginal symptoms. Diagnosis is made based on clinical findings, an amine (fishy) odor accentuated by the addition of a drop of KOH, vaginal pH greater than 4.5, and the presence of clue cells on normal saline wet mount. BV is often associated with other STIs and increases the risk of pregnancy loss and other complications.

The majority of vaginitis is caused by infectious etiologies, as described above. However, other possible etiologies that should be considered include local chemical irritants or allergens, vaginal foreign bodies, and atrophic vaginitis.

Management and Disposition

For *Candida* vaginitis, treatment with a single dose of oral fluconazole (150 mg) is very effective and is the treatment of choice if treatment compliance is a concern. Topical antifungal medication, such as clotrimazole or miconazole, should be considered in pregnant patients or for those with allergy to or difficulty tolerating fluconazole.

For *Trichomonas* vaginitis or other BV, oral metronidazole (500 mg orally twice daily for 7 days or 2 g once) is the treatment of choice. Patients should be advised of the potential for a disulfiram-like reaction when taken with alcohol and other potential adverse effects. Alternatives include topical metronidazole (5 g of 0.75% gel) or 2% clindamycin once daily for 7 days. Treatment for

asymptomatic sexual partners is not generally recommended.

TABLE 10.1 ■ CHARACTERISTICS OF INFECTIOUS VAGINITIS

Disease	Discharge Qualities	Vaginal pH	Microscopic Findings
<i>Candida</i> spp.	White, thick, clumping	< 4.5	Hyphae and spores in KOH
<i>Trichomonas</i> vaginitis	Green, thin, frothy, foul	> 4.5	Flagellates in wet mount
Bacterial vaginosis	Thin, white, “fishy” odor in KOH	> 4.5	Clue cells

Pearls

1. Undiagnosed diabetes mellitus or immunosuppression should be considered in refractory or recurrent cases of candidal vaginitis.
2. *Trichomonas* and BV should be considered STIs. Test for *Neisseria gonorrhoeae* and *Chlamydia trachomatis* and consider serologic testing for syphilis, HIV, and hepatitis B.
3. Both BV and *Trichomonas* vaginosis increase the risk of transmission of other STIs.

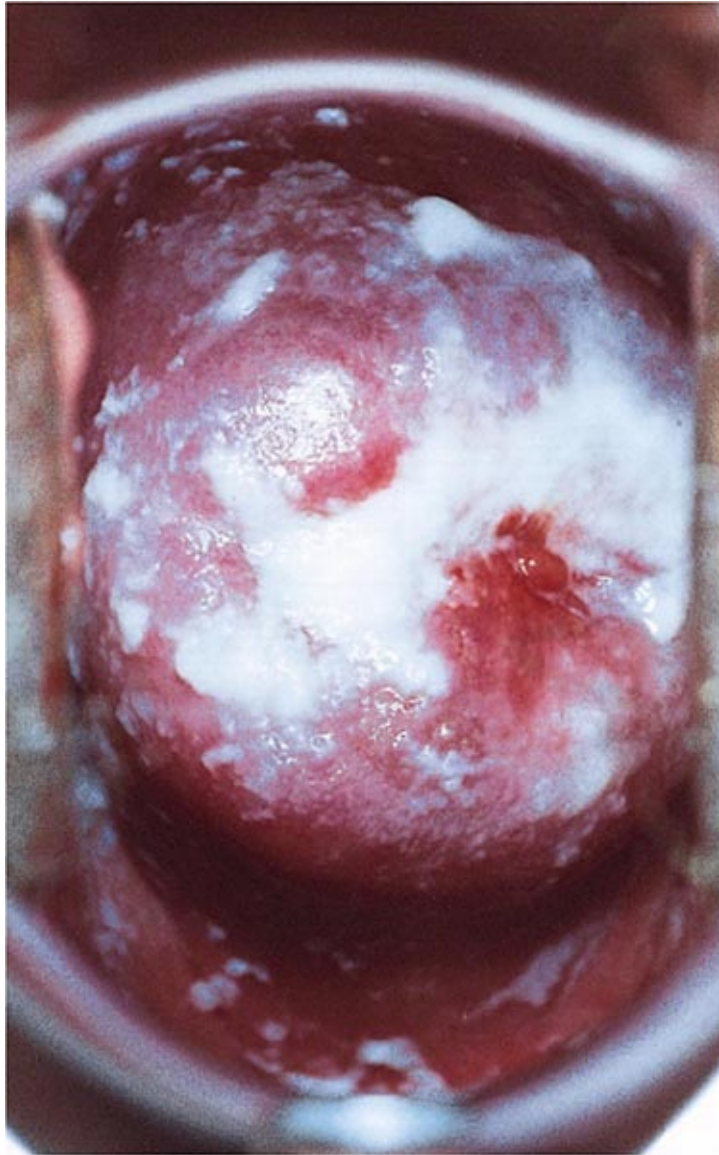


FIGURE 10.1 ■ Candidal Vaginitis. Thick, curdy white discharge secondary to candidal vaginitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

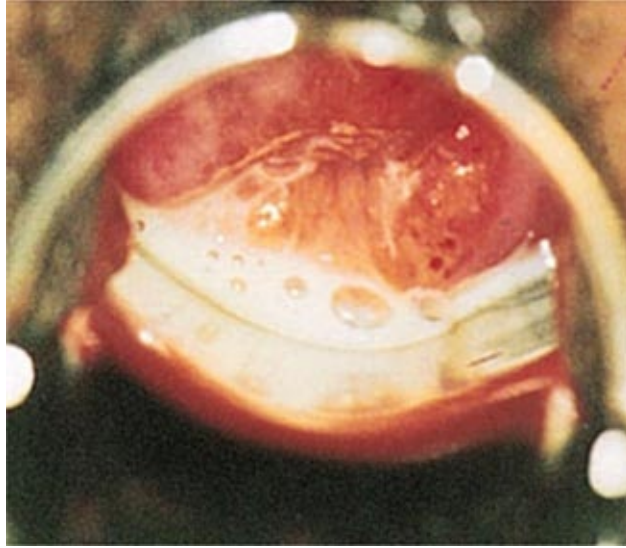


FIGURE 10.2 ■ *Trichomonas Vaginitis*. Thin vaginal discharge suggestive of trichomonas vaginitis. (Photo contributor: H. Hunter Handsfield. *Atlas of Sexually Transmitted Diseases*. New York, NY: McGraw Hill; 1992.)



FIGURE 10.3 ■ *Gardnerella Vaginosis*. Thin, milky white discharge suggestive of *Gardnerella* vaginosis. (Photo contributor: Curatek Pharmaceuticals.)

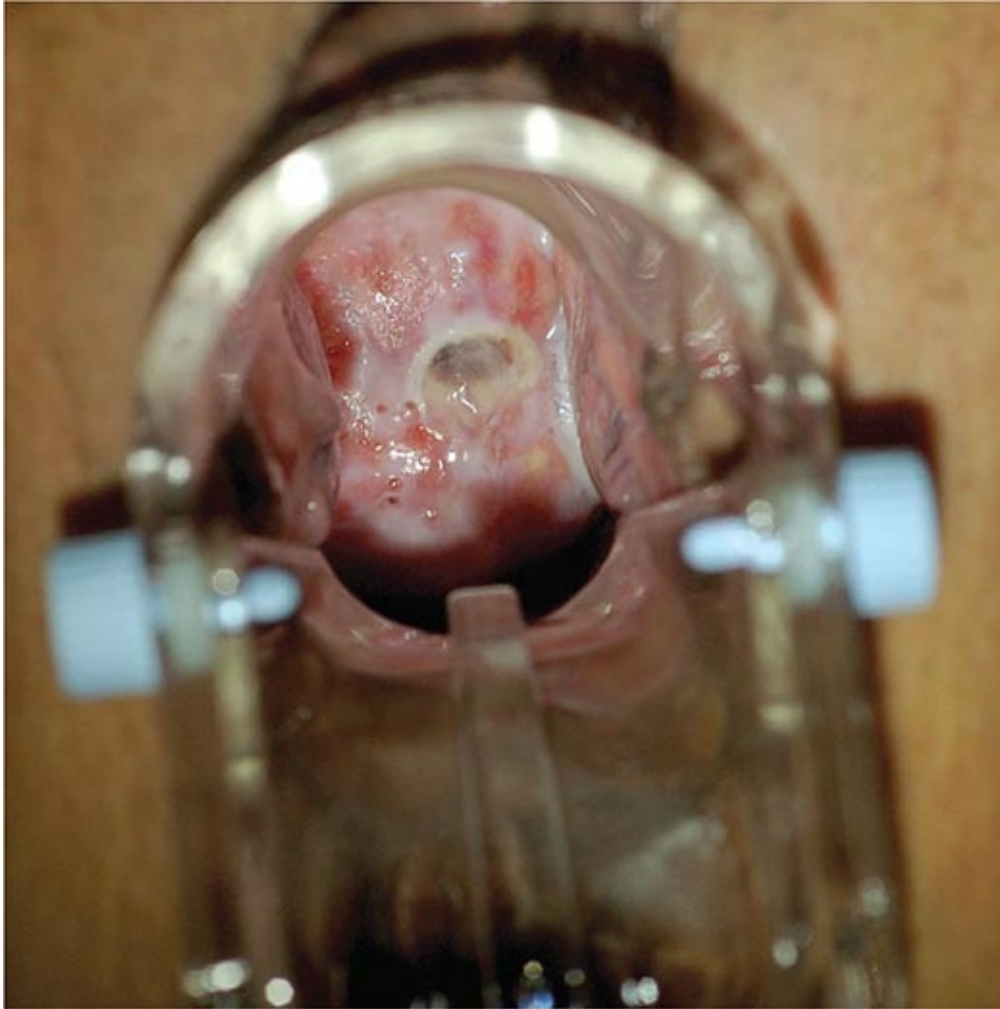


FIGURE 10.4 ■ Bacterial Vaginosis. Thin, milky white discharge suggestive of bacterial vaginosis. (Photo contributor: David Effron, MD.)



FIGURE 10.5 ■ *Candida* Vulvovaginitis. Intense vulvar erythema with thick white clumping discharge consistent with *candida* vulvovaginitis. (Photo contributor: David Effron, MD.)

CERVICAL POLYPS

Clinical Summary

Cervical polyps are friable, fingerlike growths that usually originate in the endocervical canal. They are typically asymptomatic but may bleed with minimal trauma such as intercourse or douching. Polyps may also become infected and cause purulent discharge. Single polyps are more common than multiple polyps. Polyps are most commonly caused by infection, chronic inflammation, or excess estrogen.

Management and Disposition

No treatment is necessary in the emergency department (ED). Consider alternative causes of vaginal bleeding such as infection and pregnancy. Refer all patients with incidentally discovered polyps for outpatient gynecologic

evaluation.

Pearls

1. Cervical polyps are the most common benign neoplasm of the cervix.
2. Only polyps that are bleeding or infected are usually removed.

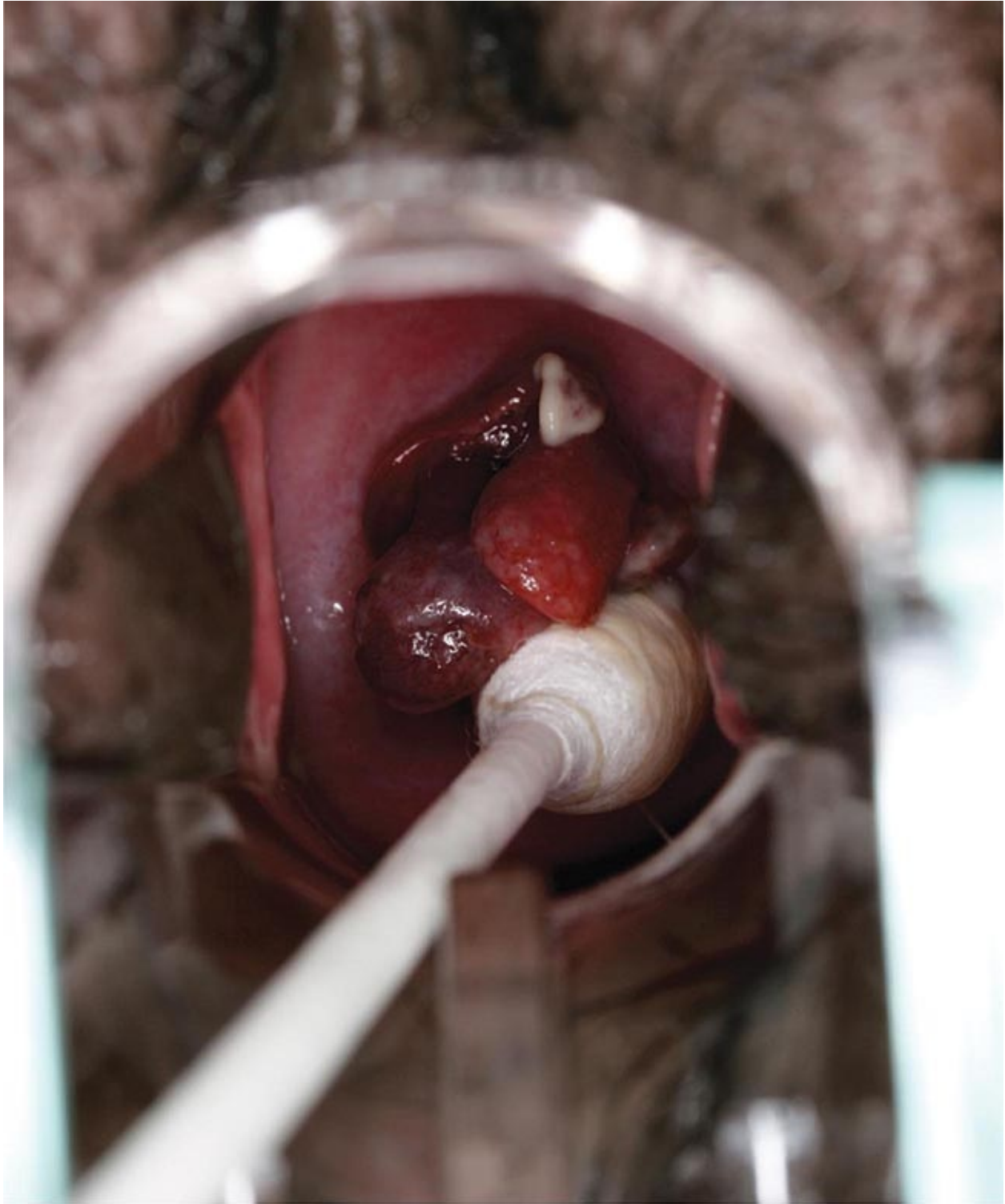


FIGURE 10.6 ■ Cervical Polyps. Several fleshy fingerlike growths are seen protruding from the cervical os. (Photo contributor: Kevin J. Knoop, MD, MS.)

BARTHOLIN GLAND ABSCESS

Clinical Summary

Bartholin gland cysts are mucus-filled and found within the labia majora, posterolateral to the vaginal opening. Cysts form secondary to Bartholin gland duct obstruction and can become infected, leading to abscess. Infection is usually with mixed vaginal or fecal flora (*Escherichia coli*) but may also contain *N gonorrhoeae* and *C trachomatis*. Cysts may be asymptomatic, whereas abscesses are usually erythematous and swollen and may cause dyspareunia. A tender, fluctuant, cystic mass with surrounding labial edema is easily appreciated on examination. Differential should include epidermal inclusion cysts and sebaceous cysts of the labia majora, hidradenitis suppurativa, vulvar hematomas, leiomyomas, lipomas, and fibromas.

Management and Disposition

Simple incision and drainage followed by sitz baths is the most effective immediate treatment, but recurrence of cysts is common. Placement of a Word catheter into the cyst cavity decreases the incidence of reocclusion. The catheter, however, must remain in place up to 6 weeks to ensure epithelialization.

Pearls

1. Antibiotics are usually not required after incision and drainage.
2. Incise the internal (medial) surface of the cyst or abscess rather than the external (lateral) aspect, as the incision site may become the new drainage tract.
3. Refer recurrent abscesses to gynecologist for definitive treatment, which may involve marsupialization of the gland.



FIGURE 10.7 ■ Bartholin Gland Cyst. Bartholin gland cyst that is not infected. The labia minoris lays over the middle of the cyst. (Photo contributor: Hope Haefner, MD.)



FIGURE 10.8 ■ Bartholin Gland Abscess. Bartholin gland abscess with the labial fluctuance pointing medially. (Photo contributor: Medical Photography Department, Naval Medical Center, San Diego, CA.)



FIGURE 10.9 ■ Bartholin Gland Abscess. Medial incision of the cyst yielding purulent fluid, consistent with a Bartholin gland abscess. (Photo contributor: Medical Photography Department, Naval Medical Center, San Diego, CA.)



FIGURE 10.10 ■ Bartholin Gland Abscess. Insertion and inflation of a Word catheter into the cyst cavity. The free end of the catheter can be tucked into the vagina for long-term placement, allowing for epithelialization of the incision site. (Photo contributor: Medical Photography Department, Naval Medical

SPONTANEOUS ABORTION

Clinical Summary

Spontaneous abortion most often presents with vaginal bleeding in early pregnancy (< 20 weeks), with or without pelvic pain. Severe pain, heavy bleeding, passage of clots or tissue, and hypotension are possible. Symptoms that are not accompanied by passage of tissue or cervical dilation constitute a *threatened abortion*. Uterine cramping with progressive cervical dilation indicates an *inevitable abortion*. Partial passage of products of conception (POC) with intrauterine retention of some tissue is an *incomplete abortion*. Fever, leukocytosis, pelvic tenderness, and malodorous cervical discharge suggest a *septic abortion*. *Completed abortion* is characterized by the passage of confirmed POC, followed by resolution of bleeding and closure of the cervical os.

Management and Disposition

Immediately obtain large-bore intravenous access and institute aggressive fluid resuscitation for any patient with severe pain, heavy bleeding, or hypovolemia. Also request cross-matched blood and urgent gynecologic consultation. Stable patients can undergo routine ED evaluation with complete blood count (CBC), human chorionic gonadotrophin (hCG), pelvic exam, and ultrasound (US). Send all identified tissue to pathology for definitive identification. *Ectopic pregnancy* must be ruled out by US, close clinical follow-up, and serial hCG testing. Administer anti-Rh immunoglobulin (RhoGAM) in all cases of vaginal bleeding where the mother is Rh negative.

Pearls

1. Large blood clots or intrauterine decidual casts may be mistaken for POC, and their presence cannot be used to rule out *ectopic pregnancy*.
2. The passage of large clots usually indicates rapid, heavy bleeding.
3. Consider heterotopic pregnancy in patients with significant ongoing

symptoms despite loss of previously identified viable early intrauterine pregnancy (IUP).

4. *Septic abortion* can lead to septic shock, acute respiratory distress syndrome, disseminated intravascular coagulation, and group A *Streptococcus*–induced toxic shock syndrome.

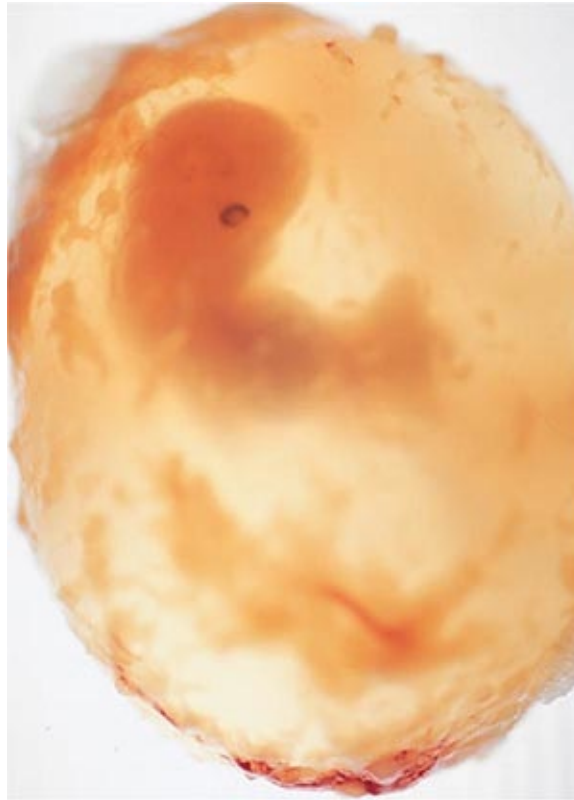


FIGURE 10.11 ■ Spontaneous Abortion. Passage of tissue in a spontaneous abortion at 4 weeks. (Photo contributor: Lawrence B. Stack, MD.)

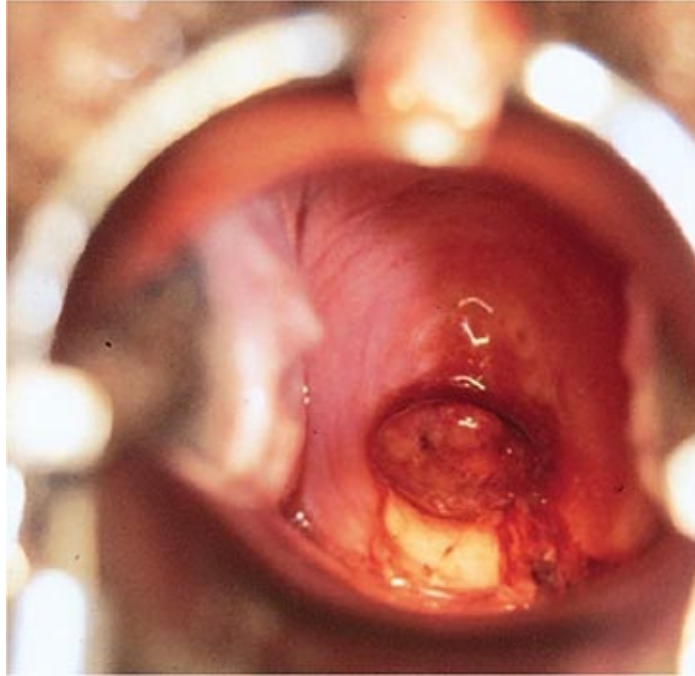


FIGURE 10.12 ■ Spontaneous Abortion. Dilation of the cervical os with partial extrusion of tissue in the setting of an inevitable abortion. (Photo contributor: Robert Buckley, MD.)



FIGURE 10.13 ■ Decidual Cast. A decidual cast or organized clot may occasionally be mistaken for products of conception. (Photo contributor: Medical Photography Department, Naval Medical Center, San Diego, CA.)

GENITAL TRAUMA AND SEXUAL ASSAULT

Clinical Summary

Comprehensive care of victims of sexual assault must address both acute medical *and psychosocial* needs of the patient and must happen in concert with forensic and legal requirements. Ideally, this care is provided by a specially trained sexual assault examiner when available. Thorough collection and preservation of all potential evidence and careful documentation of any physical findings are imperative. Chain of custody must be established and maintained for all samples collected in order for them to be used in later legal proceedings. Meticulous general and genital examination may reveal injuries to the perineum, rectum, vaginal fornices, vagina, and cervix as well as associated injuries to other areas of the body. Toluidine staining and colposcopy are useful in enhancing less apparent injuries such as those to the posterior fourchette and perianal area. Tears to the posterior fourchette are most commonly found in the distribution between the 3- and 9-o'clock positions when the patient is examined in dorsal lithotomy. Perianal lacerations are more evident with toluidine staining and appear as linear tears.

Management and Disposition

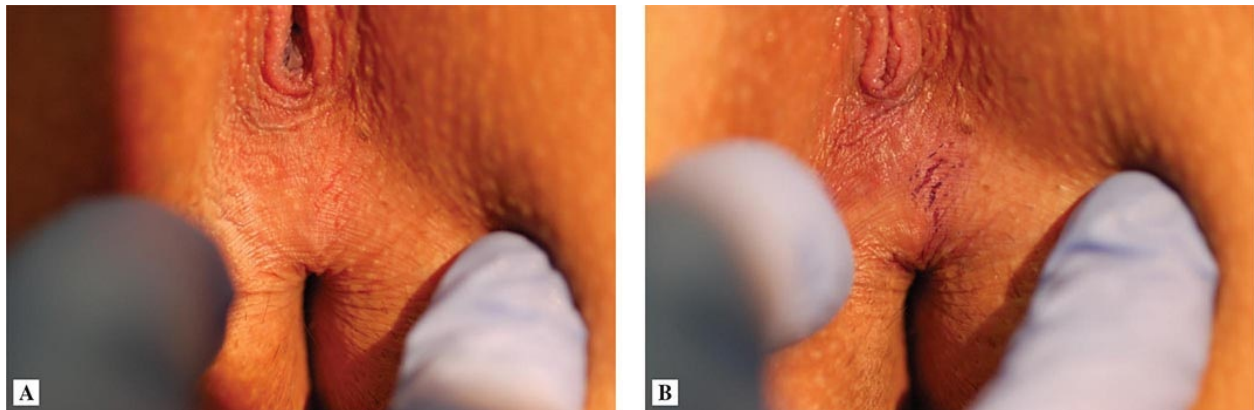


FIGURE 10.14 ■ Genital Trauma (Posterior Fourchette). (A) Linear tears of the posterior fourchette consistent with sexual assault. (B) Findings enhanced by toluidine blue staining. (Photo contributors: Hillary J. Larkin, PA-C, and Joshua Luftig, PA-C.)

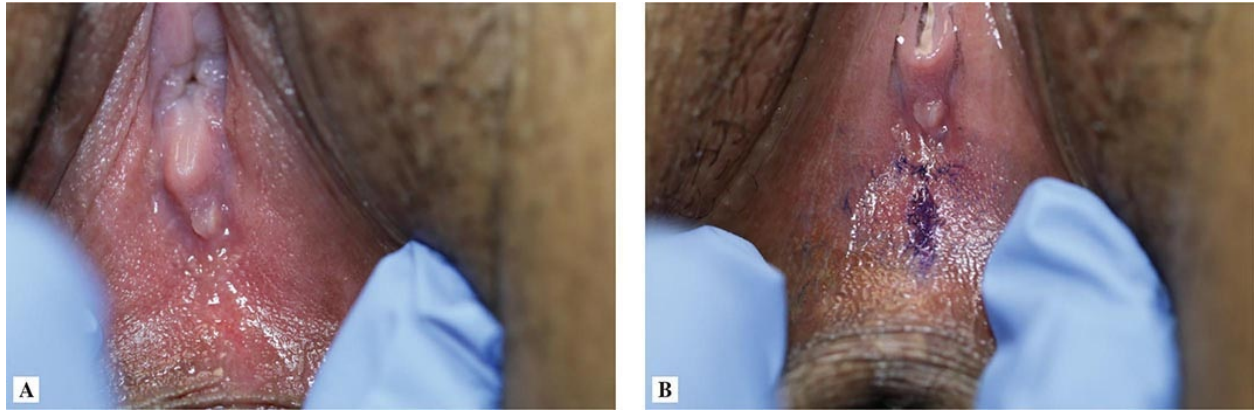


FIGURE 10.15 ■ Genital Trauma (Posterior Fourchette). (A) Linear midline tear of posterior fourchette. (B) Findings enhanced by toluidine blue staining. (Photo contributors: Hillary J. Larkin, PA-C, and Joshua Luftig, PA-C.)

Forensic evidence collection should precede treatment other than pain relief. Forensic evaluation should include Wood lamp examination to identify semen for collection, pubic hair sampling and combing, vaginal and cervical smears (air-dried), a cervical and vaginal wet mount to identify sperm, vaginal aspirate to test for acid phosphatase, and rectal or buccal swabs for sperm. A prepackaged kit with directions may be available to facilitate the collection of evidence. Consult your state's laws for requirements.

Obtain cervical cultures for *Chlamydia* and *N gonorrhoeae* as well as serum testing for syphilis, hepatitis, and HIV. Provide empiric antibiotic coverage against STIs and offer an oral contraceptive to prevent unwanted pregnancy where applicable.

Pearls

1. Victims of sexual assault may initially be reluctant to disclose their history and often present with an unrelated chief complaint. A thorough sexual history is necessary for proper identification and treatment.
2. Normal physical examination and lack of sperm on wet preparation do *not* exclude the possibility of assault.
3. Perineal injuries from accidental trauma may be indistinguishable from those of sexual assault and should be interpreted in the context of the history.



FIGURE 10.16 ■ Genital Trauma (Posterior Fourchette and Perineum). Multiple linear tears of posterior fourchette and perineum enhanced with toluidine blue stain. (Photo contributors: Hillary J. Larkin, PA-C, and Joshua Luftig, PA-C.)

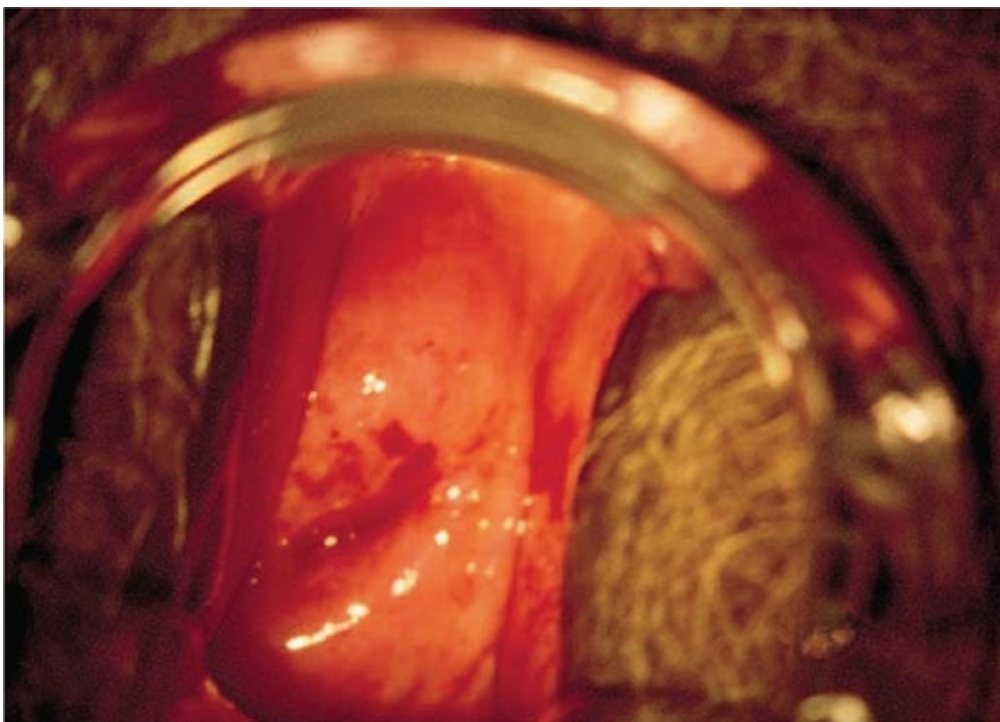


FIGURE 10.17 ■ Genital Trauma (Cervix). Cervical trauma in an elderly victim of sexual assault. Petechiae and freshly bleeding abrasions are noted from 10- to 3-o'clock position. (Photo contributors: Hillary J. Larkin, PA-C, and Lauri A. Paolinetti, PA-C.)

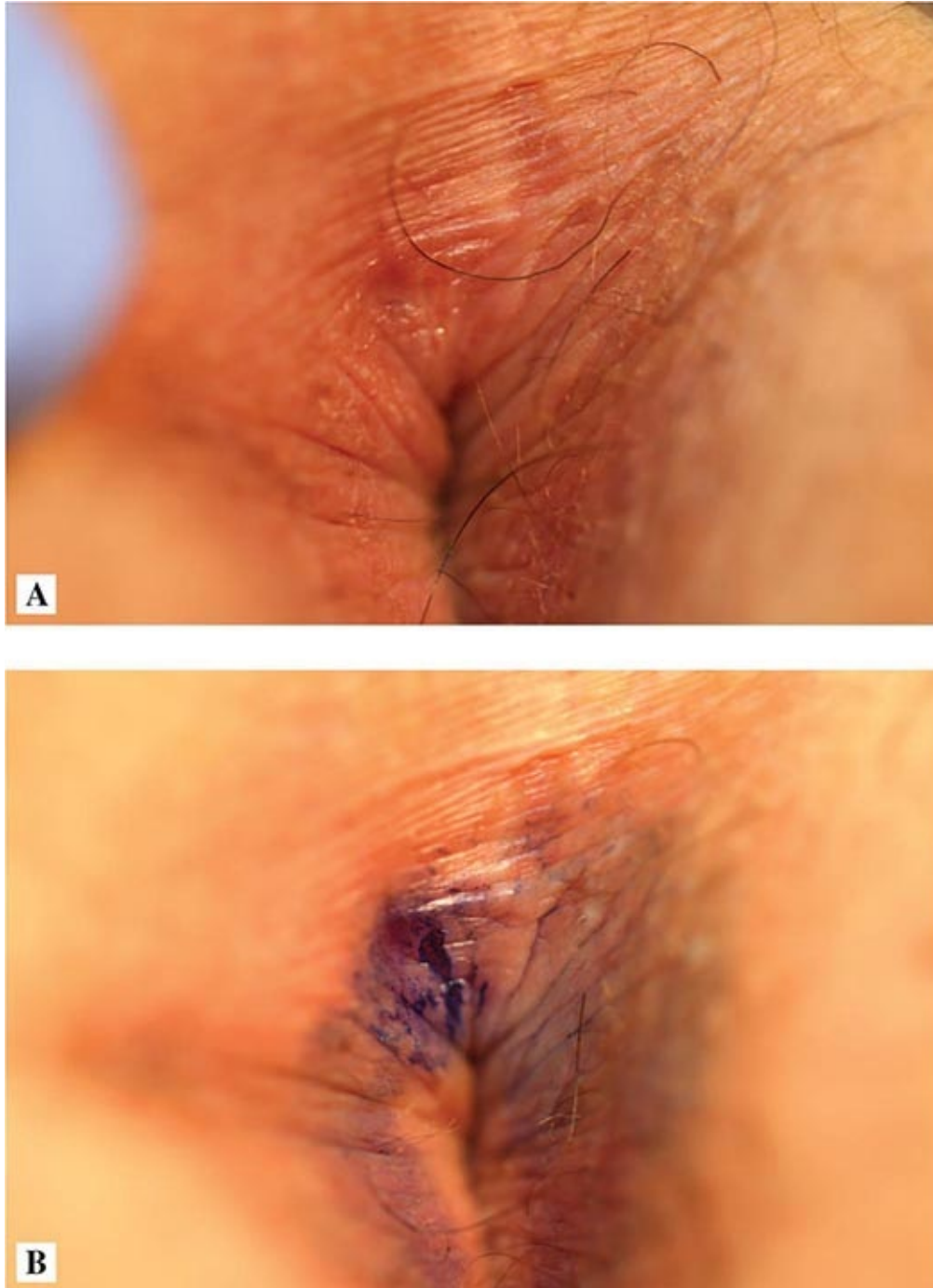


FIGURE 10.18 ■ Anal Trauma (Perianal, Toluidine Blue). Multiple perianal tears without toluidine blue stain (**A**) and with stain (**B**). Note specific stain uptake enhancing two tears at 11-o'clock position in contrast to nonspecific uptake elsewhere. (Photo contributors: Hillary J. Larkin, PA-C, and Joshua Luftig, PA-C.)

VULVAR HEMATOMA

Clinical Summary

Because of the rich vascular supply, trauma to the perineum can produce bleeding, commonly resulting in a vulvar hematoma. It is important to ascertain the mechanism and to examine for internal injuries, which are more difficult to detect. A common mechanism is a straddle injury, but this can also be a result of forceful consensual or nonconsensual sexual activity. Patients are often reluctant to reveal the nature of the mechanism, making those at risk harder to identify. The accumulation of the hematoma is usually rapid and painful. If there is an associated laceration, bleeding can be brisk.

Management and Disposition

Large or expanding hematomas require urgent consultation for further management to include examination under anesthesia for internal injury, further observation, pain control, and rarely, incision and drainage, which is best avoided due to the risk of introducing bacteria and difficult hemorrhage control. Minor hematomas can be managed as an outpatient providing pain is controlled (ice packs and analgesics) and follow-up arranged. Disposition to a short-stay observation unit may be appropriate.

Pearls

1. Sexual abuse or assault should be considered in cases that do not have a straight forward history or mechanism.
2. Vulvar hematomas are the most common sequelae of vulvar trauma.
3. Translabial ultrasound may be useful to discern between hematoma and soft tissue swelling.
4. Place a foley catheter early to prevent bladder obstruction for large or expanding hematomas.



FIGURE 10.19 ■ Vulvar Hematoma. This large vulvar hematoma from coitus-related activity required surgical drainage. A laceration is noted at the 6-o'clock position of the labia. (Photo contributor: Zachary Wohlgemuth, MD.)

LICHEN SCLEROSUS

Clinical Summary

Lichen sclerosus (LS) is a benign, chronic, inflammatory condition of unclear etiology that results in epithelial atrophy or hyperplasia, and scarring. The condition typically occurs in the anogenital region (85%-98%), but can develop on any skin surface. Female-to-male ratio is 10:1 with onset often perimenopausal. It occurs more frequently in women in low-estrogen states with autoimmune conditions such as diabetes mellitus type 1, vitiligo, and thyroid disorders.

Patients may be asymptomatic or may have intense pruritis and pain in the affected areas, bleeding, dyspareunia, or dysuria. Lesions are white, atrophic papules and patches and most often affect the labia. Excoriations may be present and may lead to secondary lichenification (thickening of the epidermis with exaggeration of normal skin lines). The disease is often progressive, and scarring may lead to loss of the vulvar architecture with fusion of the labia. Extragenital lesions may occur on the thighs, breasts, wrists, shoulders, neck, and, rarely, the oral cavity. They are usually not as pruritic as genital lesions.

Management and Disposition

No emergent treatment is necessary. Advise patients on good vulvar hygiene and cessation of scratching to prevent secondary infections. Consider oral antihistamines for pruritis. Refer patients for outpatient gynecologic evaluation, which may include biopsy for definitive diagnosis. Potential treatments include topical steroids, hormonal treatments, and/or immunosuppressants.

Pearls

1. Lichen sclerosis is associated with an increased risk of vulvar squamous cell cancer. Refer all patients for biopsy and histologic evaluation.
2. Advise patients that although LS is not an infectious condition, the presence of open excoriations and fissures can increase risk of acquiring STIs.
3. The vagina and cervix are not involved in lichen sclerosis.



FIGURE 10.20 ■ Lichen Sclerosus. Early lichen sclerosus appearing as white, atrophic papules and patches of the labia minora and labia majora. (Photo contributor: Hope Haefner, MD.)

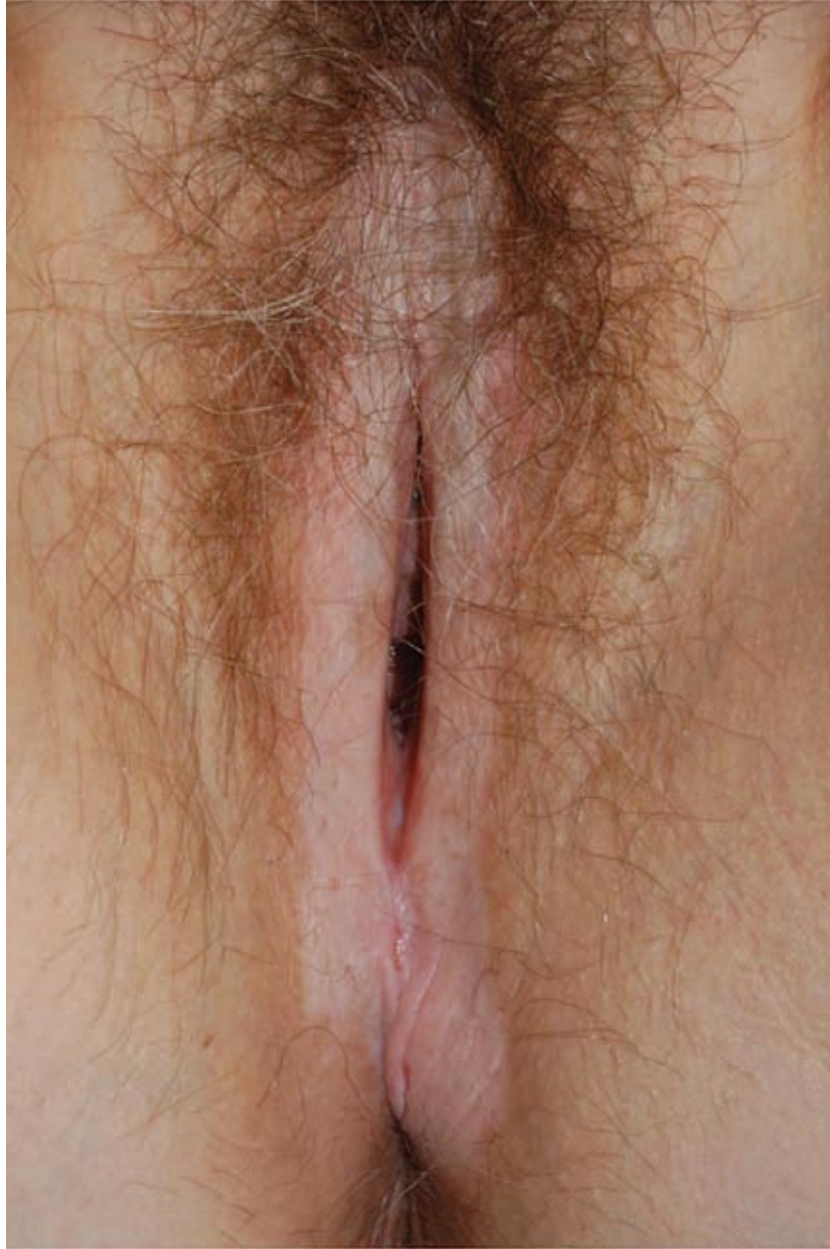


FIGURE 10.21 ■ Lichen Sclerosus—Chronic. Atrophy and scarring of chronic lichen sclerosus with early fusion of the anterior labia. (Photo contributor: Hope Haefner, MD.)



FIGURE 10.22 ■ Lichen Sclerosus—Advanced. Extensive fusion of the labia minora in advanced lichen sclerosus over the vaginal opening. (Photo contributor: Hope Haefner, MD.)

LICHEN PLANUS

Clinical Summary

Lichen planus is a recurrent inflammatory condition that can affect any area of skin, mucosa, nails, and scalp. Although the etiology is unknown, it is often associated with autoimmune diseases suggesting an autoimmune cause.

Symptoms of lichen planus may include intense vulvar pruritus, pain, and/or dysuria. Vaginal involvement is seen in 70% of cases and may result in vaginal discharge, dyspareunia, and occasionally postcoital bleeding. Coexisting oral or other cutaneous lesions are common. Vulvar lichen planus is characterized by violaceous, glassy, erosive, or papular lesions. Desquamation may be present. Recurrent lesions may lead to extensive scarring with resultant loss of the vulvar architecture including stenosis of the vaginal introitus and urethral obstruction.

Lichenoid drug eruptions can be clinically indistinguishable from lichen planus. Medications including β -blockers, methyldopa, penicillamine, quinidine, nonsteroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme (ACE) inhibitors, sulfonamide agents, carbamazepine, gold, lithium, quinidine, or hydrochlorothiazide (HCTZ) have been associated with lichenoid drug eruptions.

Management and Disposition

ED management of lichen planus includes antihistamines for pruritis and gynecologic referral. If a lichenoid drug eruption is suspected, recommend discontinuation of medications that may be causative. Advise patients on good vulvar hygiene including cessation of scratching to prevent secondary infections. Sitz baths may help alleviate symptoms. Definitive diagnosis is made by biopsy.

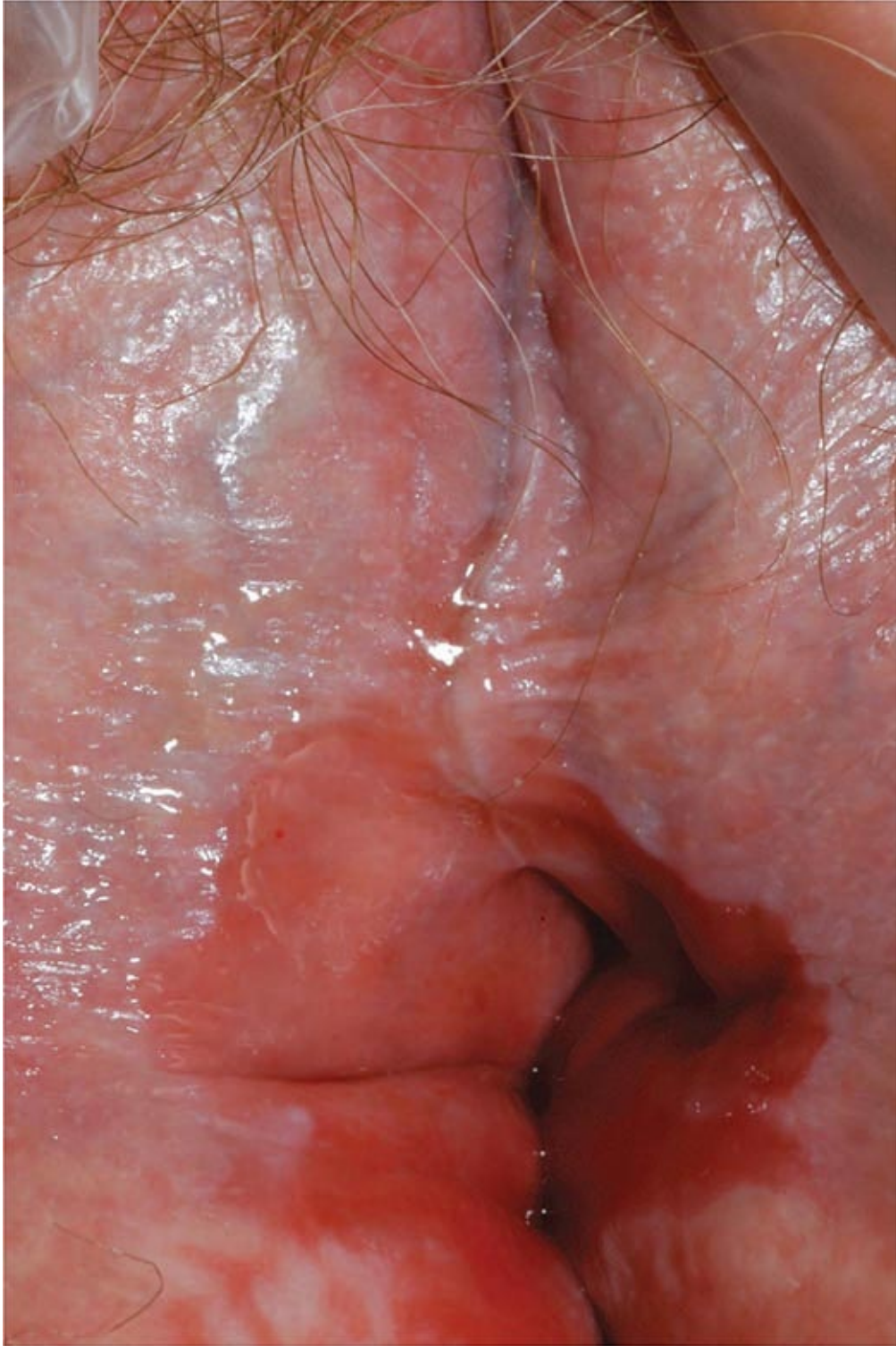


FIGURE 10.23 ■ Erosive Lichen Planus. Glassy erosive lesions of lichen planus with “lichenification,” or thickening of the epidermis, in peripheral areas. (Photo contributor: Hope Haefner, MD.)



FIGURE 10.24 ■ Erosive Lichen Planus. Glassy erosive lesions of lichen planus. (Photo contributor: Hope Haefner, MD.)



FIGURE 10.25 ■ Erosive Lichen Planus—Oral. Lichen planus can affect any area of skin, mucosa, nails, and scalp. (Photo contributor: Hope Haefner, MD.)

Pearls

1. HCTZ and NSAIDs are common triggers for recurrent lichen planus.
2. Lichen planus does not involve the perianal region, whereas lichen sclerosis may.

CERVICAL CANCER

Clinical Summary

Cervical cancer was once one of the most common causes of death from cancer in US women; however, deaths have decreased significantly with the use of routine Pap smears, which can detect precancerous changes and prompt early treatment. It is most commonly diagnosed between the ages of 35 and 44, but risk increases with age, and more than 15% of new cases are in women older than age 65. It is rare in women younger than 20. Squamous cell carcinoma accounts for 90% of cases, with adenocarcinoma being the second most common. Risk factors include infection with human papillomavirus (HPV), smoking, chlamydial infections, immunosuppression, intrauterine device (IUD) use, family history, multiple full-term pregnancies, early age at 1st pregnancy, and long-term oral contraceptive use. Most cervical cancer or precancerous lesions are found incidentally or during routine gynecologic exam. More advanced cases may present with pelvic discomfort, vaginal bleeding or discharge, bleeding after intercourse, and dyspareunia. Speculum examination with careful visual inspection of the cervix is key to identification. Definitive diagnosis is made by biopsy.

Management and Disposition

With the exception of rapid bleeding from a large lesion, emergent treatment is not typically warranted for cervical cancer. If heavy active bleeding is present, apply pressure manually or by packing the vagina to tamponade the bleeding. Consult gynecologist immediately. Refer all stable patients with suspicious lesions for outpatient gynecologic evaluation.

Pearls

1. Cervical cancer is considered by some to be an STI because almost all cases

are caused by infection with HPV.

2. The increasing use of HPV vaccinations is reducing the incidence of infection with resultant decreased incidence of cervical cancer.



FIGURE 10.26 ■ Cervical Cancer. An early cervical cancer is seen. Note the inflamed cervix with friable mucosa. (Photo contributor: Loyd A. West, MD.)



FIGURE 10.27 ■ Cervical Cancer. An irregular inflamed and friable-appearing cervix biopsy positive for cervical cancer is seen. (Photo contributor: Loyd A. West, MD.)

VULVAR CANCER

Clinical Summary

Vulvar cancer accounts for 5% of gynecologic malignancies and occurs most commonly in postmenopausal women. Many vulvar neoplasms are asymptomatic and are discovered incidentally during gynecologic examination. Patients with symptoms most commonly complain of vulvar pruritis and may have noticed a vulvar mass. In advanced cases, vulvar bleeding or discharge, dysuria, or inguinal lymphadenopathy may be present. A single plaque, ulcer, or mass (fleshy, nodular, or warty) is most commonly found on the labia majora, but other areas may be affected. Less than 5% of cases have multifocal lesions.

Squamous cell carcinomas account for 90% of vulvar cancers, whereas melanoma is the second most common type. Risk factors include smoking, chronic inflammatory conditions such as LS, and infection with HPV 16, 18, or 33.

Management and Disposition

No treatment is necessary in the ED. Refer all patients with suspicious vulvar lesions for outpatient gynecologic evaluation. Definitive diagnosis is made by biopsy.

Pearl

1. Coexisting cervical neoplasia is found in more than 20% of patients with a vulvar malignancy.



FIGURE 10.28 ■ Vulvar Melanoma. A melanoma is seen in the proximal vaginal mucosa. (Photo contributor: Loyd A. West, MD.)



FIGURE 10.29 ■ Squamous Cell Carcinoma. An area of lichenification, with an ulcerated erythematous lesion at the 6-o'clock position on the vaginal introitus is seen. (Photo contributor: Loyd A. West, MD.)



FIGURE 10.30 ■ Squamous Cell Carcinoma. A large erythematous ulceration in the midportion of the left labia majora is seen. An area of invasive cancer is seen at the superior margin of the labia majora. (Photo contributor: Loyd A. West, MD.)

URETHRAL PROLAPSE

Clinical Summary

Urethral prolapse is rare. It occurs most commonly in prepubertal black females and postmenopausal white women. Symptoms such as vaginal bleeding and urinary complaints are common in postmenopausal women, but children are frequently asymptomatic. A large prolapse may become strangulated and result in pain, bleeding, and urinary symptoms. Examination reveals a protrusion of the distal urethra through the meatus that appears as a donut-shaped mass at the

anterior vaginal wall. Diagnosis is confirmed on identification of the urethral meatus as the central opening in the prolapsed tissues. Urethral catheterization may be required if direct observation during voiding is not possible.

Management and Disposition

Rule out urinary tract infection, which is frequently associated with urethral prolapse. Consult urology emergently for signs of urethral strangulation. Medical therapy consists of sitz baths and topical estrogen cream applied two to three times to the urethra for 2 weeks. Surgical therapy is reserved for strangulation or failed medical therapy. Refer all patients with confirmed or suspected urethral prolapse to a urologist for follow-up.

Pearls

1. Topical estrogen cream may be contraindicated in patients with a history of breast cancer.
2. Urethral prolapse in children may be mistaken for sexual abuse (see [Chapter 15](#)).
3. Urethral masses may be misdiagnosed as urethral prolapse.



FIGURE 10.31 ■ Urethral Prolapse. Note the protrusion of the distal urethra, seen as a donut-shaped mass at the anterior vaginal wall. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 10.32 ■ Urethral Prolapse. Protrusion of the distal urethra is seen as a donut-shaped mass just superior to the vaginal introitus in a patient complaining of “vaginal bleeding.” (Photo contributor: Lawrence B. Stack, MD.)

UTERINE PROLAPSE

Clinical Summary

Uterine prolapse is defined as the extrusion of the uterus through the pelvic floor

or vaginal introitus. Prolapse is caused by weakened pelvic floor muscles and is common in older, multigravid women. In first-degree prolapse, the cervix descends into the lower third of the vagina, while in second-degree prolapse, the cervix usually protrudes through the introitus. Third-degree prolapse, or procidentia, is characterized by externalization of the entire uterus with eversion of the vagina. Symptoms include a sensation of inguinal traction, low back pain, urinary incontinence, and the presence of a vaginal mass.

Management and Disposition

Refer patients with first- or second-degree prolapse to a gynecologist for pessary placement or surgical correction. Urgent gynecologic consultation is indicated for procidentia. Temporize with manual reduction of the uterus into the vaginal vault and place the patient on complete bed rest.

Pearls

1. With procidentia, the exposed uterus is prone to abrasion and secondary infection.
2. Valsalva maneuver may reproduce or increase prolapse, cystocele, and enterocele.
3. Uterine prolapse may be confused with a cystocele (discussed below), enterocele, or soft-tissue tumor.



FIGURE 10.33 ■ Third-Degree Uterine Prolapse. Note the protrusion of the entire uterus with the cervix visible through the vaginal introitus. (Photo contributor: Lawrence B. Stack, MD.)

CYSTOCELE

Clinical Summary

A cystocele is a herniation of the posterior bladder wall and trigone through the anterior wall of the vagina. It is caused by weakening of the pelvic floor during childbirth. Patients complain of vaginal bulging or fullness. Symptoms are worsened by Valsalva maneuvers and improved with positioning (recumbent). Cystoceles are often associated with urinary incontinence and incomplete emptying of the bladder. Rectoceles, uterine prolapse, and soft tissue tumors should also be considered.

Management and Disposition

ED management is reassurance and outpatient referral to a gynecologist. Advise

the patient to avoid heavy lifting or straining. Definitive treatment for larger cystoceles or those associated with significant urinary symptoms, pain, or bothersome bulging may require pessary placement or surgery.

Pearl

1. Most cystoceles are asymptomatic and are detected incidentally at the time of pelvic examination.



FIGURE 10.34 ■ Cystocele. Cystocele with bulging of the posterior bladder wall into the vagina. (Photo contributor: Matthew Backer, Jr., MD.)



FIGURE 10.35 ■ Cystocele. Cystocele worsening with Valsalva. (Photo contributor: Matthew Backer, Jr., MD.)

RECTOCELE

Clinical Summary

A rectocele is the herniation of the rectum through the posterior vaginal wall. Most small rectoceles are asymptomatic, but symptoms of introital bulging, constipation, and incomplete rectal evacuation may occur. Physical examination may reveal bulging of the posterior vaginal wall through the introitus. A thin-walled protrusion of the rectovaginal septum into the lower part of the vagina is seen on internal vaginal examination.

Management and Disposition

ED management is symptomatic with hydration, laxatives, and stool softeners. Refer patients with large or symptomatic rectoceles to a gynecologist for possible surgical repair.

Pearl

1. Patients may complain of the need for manual reduction of the rectocele when defecating.



FIGURE 10.36 ■ Rectocele. This is characterized by bulging of the posterior vaginal wall at the introitus. (Photo contributor: Matthew Backer, Jr., MD.)



FIGURE 10.37 ■ Rectocele. Worsening of the rectocele with Valsalva. (Photo contributor: Matthew Backer, Jr., MD.)

IMPERFORATE HYMEN

Clinical Summary

The hymen is normally a perforate membrane seen at the vaginal introitus where it separates the vestibule externally from the vagina internally. Imperforate

hymen refers to the congenital absence of a hymenal orifice. This condition may present in infants or young children as a smooth, glistening membrane protruding from the introitus due to the buildup of vaginal secretions known as a *mucocolpos*. More commonly, it presents in adolescent girls with the accumulation of menstrual blood and secretions behind the hymen, known as a *hematocolpos*. The fluid collection can become large enough to compress the bladder neck and cause urinary retention. Occasionally, the accumulated blood spilling into the peritoneal cavity through the fallopian tubes results in free pelvic fluid and signs of peritonitis. On examination, hematocolpos is evident as a smooth, dome-shaped, bluish-red bulging membrane at the introitus. A large, smooth, cystic mass can often be palpated anteriorly on digital rectal examination.

Management and Disposition

Refer to a gynecologist for definitive treatment of imperforate hymen and other abnormalities of the vaginal outlet. Incision of the hymen to allow drainage of the hematocolpos may be needed emergently in patients with severe pain and/or signs of peritonitis.

Pearls

1. An imperforate hymen commonly presents in adolescent girls with primary amenorrhea and recurrent abdominal pain.
2. Refer preadolescent patients to a pediatric gynecologic practitioner when possible.



FIGURE 10.38 ■ Imperforate Hymen. A bulging mass at the introitus is seen in this patient with abdominal distention and amenorrhea. The imperforate hymen was diagnosed, with subsequent incision and drainage of the hematocolpos. (Photo contributor: Mark Eich, MD.)

Obstetric Conditions

ECTOPIC PREGNANCY

Clinical Summary

Ectopic pregnancy is one that occurs outside of the uterus and is the leading cause of first-trimester maternal obstetric morbidity. It can be asymptomatic or present with symptoms ranging from mild vaginal bleeding and lower abdominal pain to shock secondary to massive hemorrhage. Menstrual history may reveal a missed or recent abnormal menses. Pelvic examination may be normal or may include vaginal bleeding, cervical motion tenderness, or adnexal tenderness. An abnormally low or slowly rising hCG may be seen. Risk factors include use of an IUD, prior ectopic pregnancy, and a history of pelvic inflammatory disease,

tubal ligation, and other abdominal or pelvic surgeries.

The visualization of an IUP on US excludes the diagnosis of ectopic pregnancy with the exception of a rare heterotopic pregnancy (both intrauterine and ectopic). A gestational sac (GS) may be seen at about 5 weeks. This is *suggestive* of an IUP; however, definitive diagnosis of IUP requires identification of a yolk sac. The double decidual sac sign is evidence of a true GS and should be differentiated from the single pseudogestational sac formed from a decidual cast in ectopic pregnancy. When no GS is visualized (“empty uterus”), ectopic pregnancy cannot be distinguished from an early IUP or from recently completed spontaneous abortion. Free fluid on US is concerning but not specific.

Management and Disposition

Unstable patients require aggressive resuscitation with fluid and blood, followed by surgery. Stable patients warrant immediate gynecologic consultation. Observation of an asymptomatic patient with repeat measurement of hCG and outpatient follow-up may be considered after gynecologic consultation. Medical management with methotrexate may be used in stable patients, whereas immediate surgical intervention is indicated in unstable patients. US has diminished diagnostic accuracy at lower hCG levels, and ectopic pregnancy has been observed at hCG level less than 100 mIU/mL. Therefore, obtain gynecologic consultation if there is a clinical suspicion for ectopic pregnancy regardless of the US findings or hCG level.

Pearls

1. Ectopic pregnancy should be considered in all women of reproductive age presenting with syncope, vaginal bleeding, abdominal pain or tenderness, and missed menstrual period.
2. Failure to visualize an IUP by transvaginal ultrasonography when the serum hCG level is *above* the discriminatory zone of the machine and examiner (typically 1500-2000 mIU/mL) is highly suggestive of ectopic pregnancy.

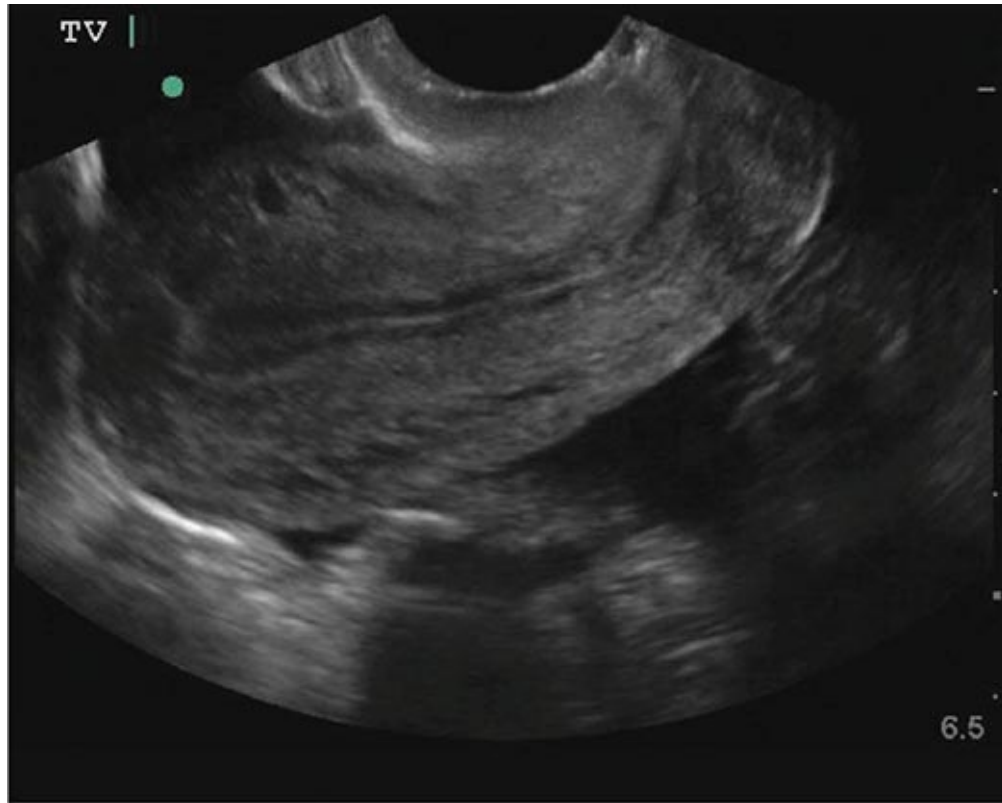


FIGURE 10.39 ■ Empty Uterus. Free fluid seen in the pelvis in this patient with an empty uterus is concerning if ectopic pregnancy is suspected. Ectopic pregnancy should be strongly suspected if a transvaginal ultrasound reveals an empty uterus in the setting of a serum quantitative hCG level above the institution's discriminatory zone. (Photo contributor: Lauren Oliveira, DO.)

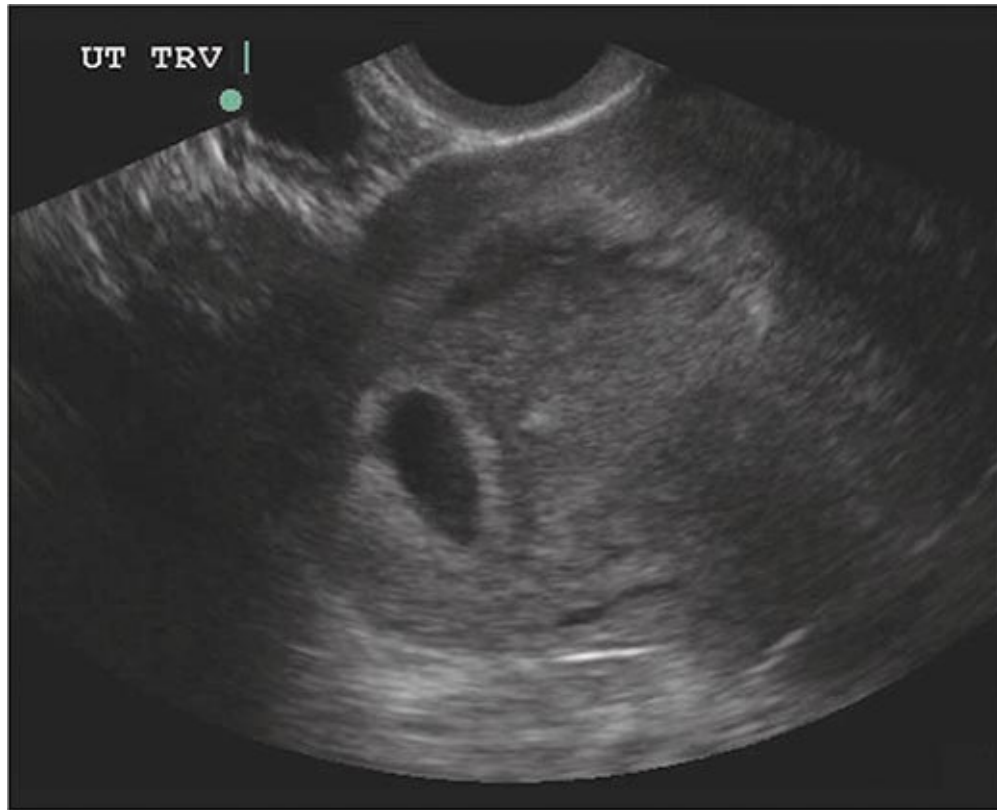


FIGURE 10.40 ■ Intrauterine Gestational Sac. Discrete ring of an intrauterine gestational sac seen on transvaginal ultrasound. No yolk sac is visualized. A double decidual sac sign is seen, lending evidence of a true gestational sac versus a pseudogestational sac formed from a decidual cast in ectopic pregnancy. A thorough look in the adnexa is important in diagnosing ectopic pregnancy when a gestational sac is the only finding. (Photo contributor: Lauren Oliveira, DO.)

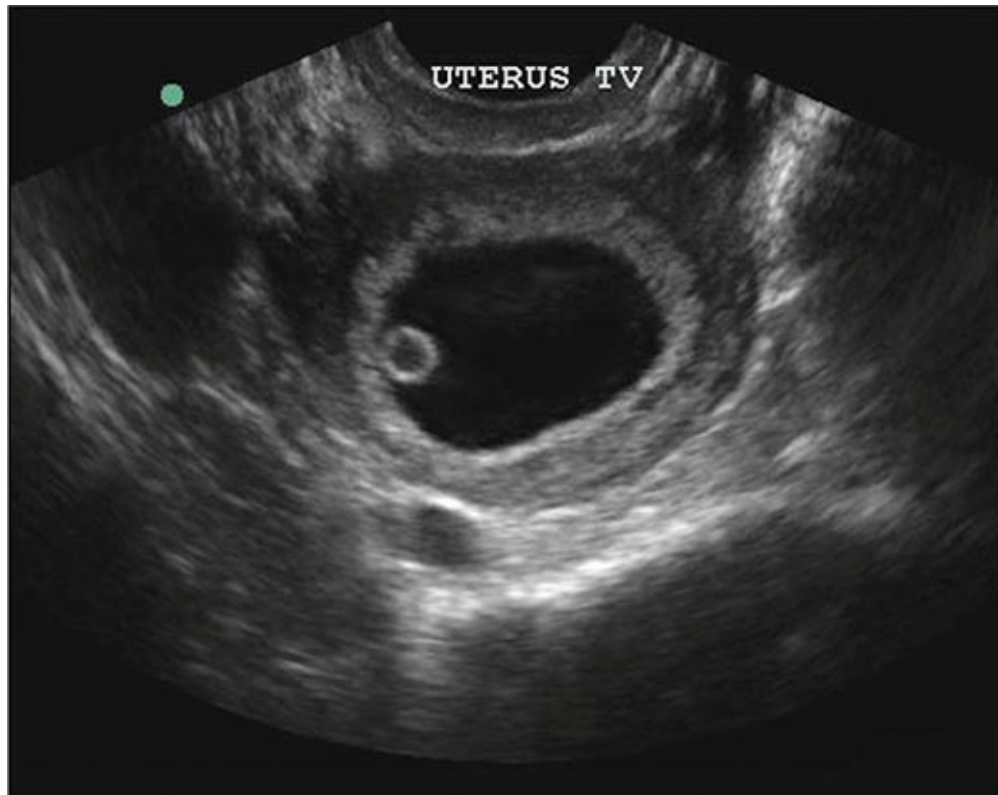


FIGURE 10.41 ■ Intrauterine Yolk Sac. Discrete ring of an intrauterine yolk sac within the gestational sac seen on transvaginal ultra-sound. Definitive diagnosis of IUP can be made once a yolk sac is seen. A double decidual sac sign is also seen. (Photo contributor: Lauren Oliveira, DO.)

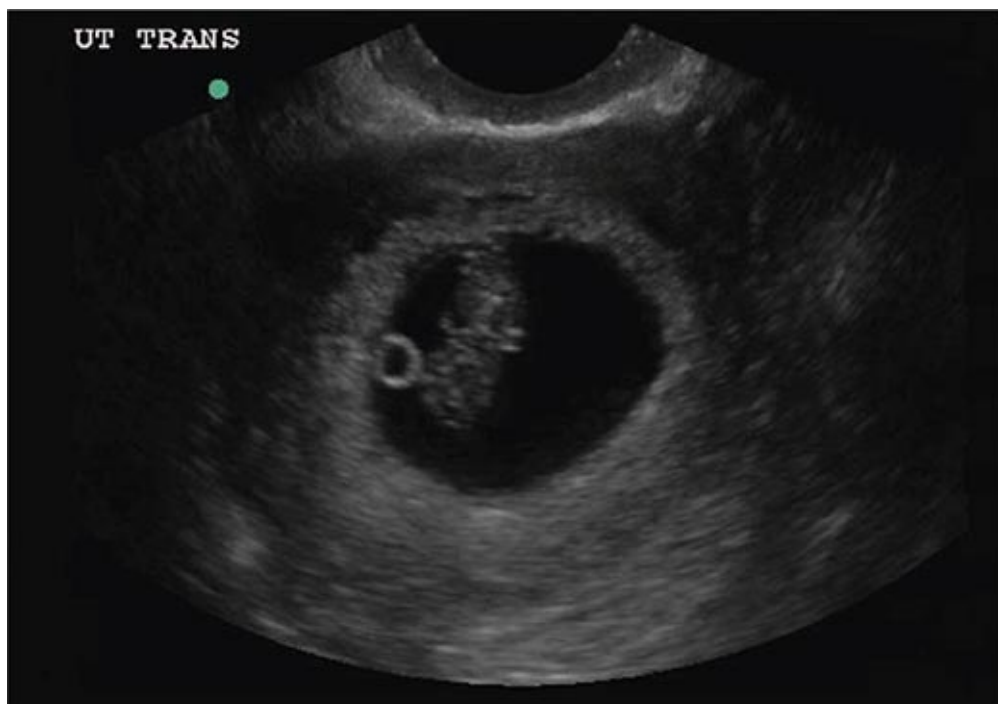


FIGURE 10.42 ■ Intrauterine Fetal Pole. Ultrasound image of an intrauterine pregnancy with a fetal pole

seen adjacent to a yolk sac. (Photo contributor: Lauren Oliveira, DO.)



FIGURE 10.43 ■ Ectopic Pregnancy. A large ectopic pregnancy is seen adjacent to the uterus. (Photo contributor: Lauren Oliveira, DO.)

3. The double decidual sac sign does not definitively confirm an IUP. A yolk sac must be present.
4. Visualization of the adnexa on US may reveal tubal ectopic pregnancy, but failure to show this does *not* necessarily rule out the diagnosis.
5. Heterotopic pregnancy is more common in pregnancies conceived with in vitro fertilization (1-3:100 vs 1:30,000).

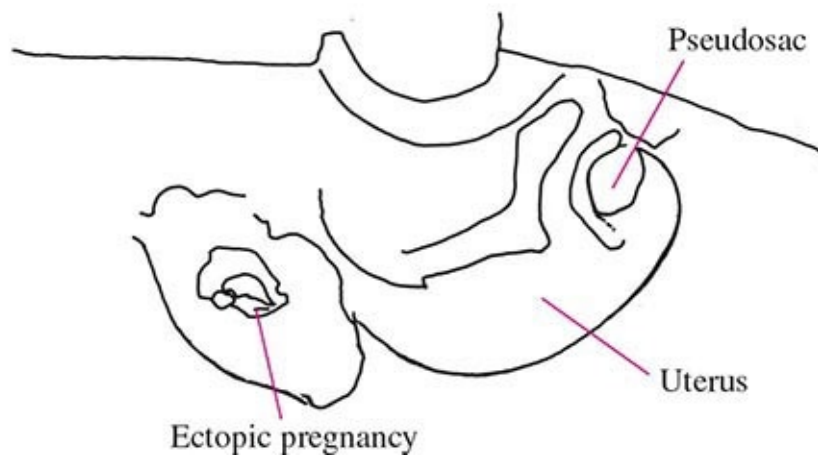
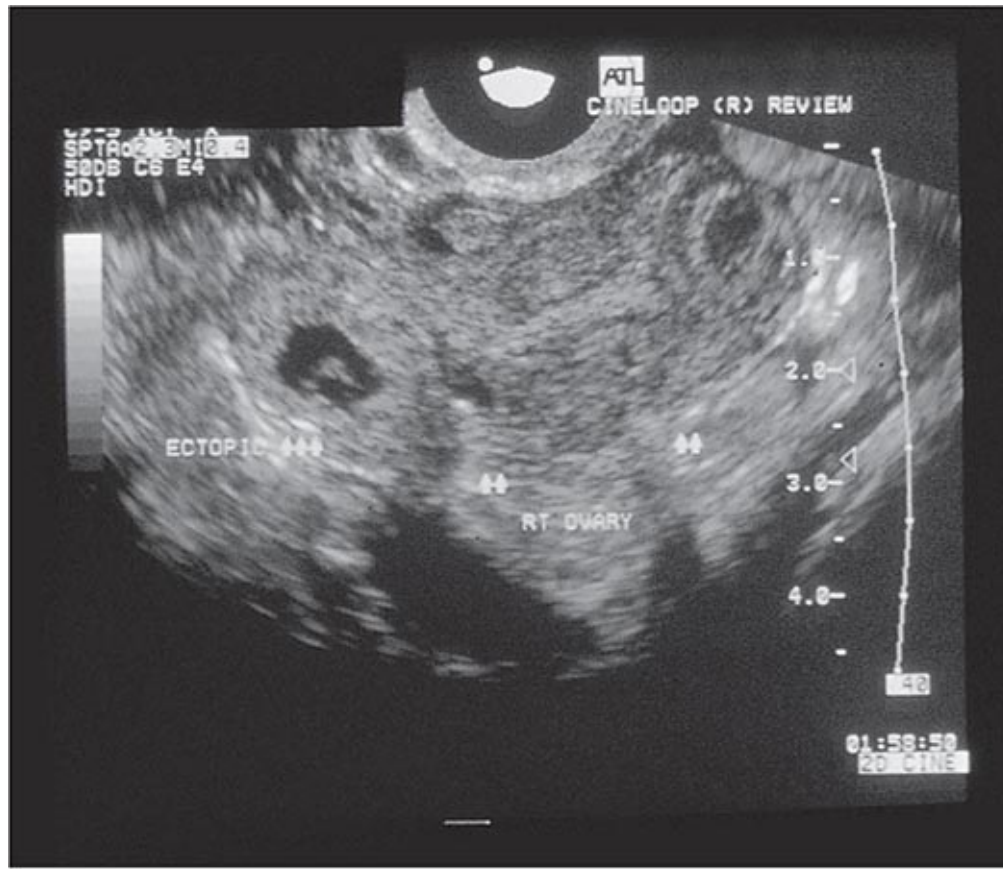


FIGURE 10.44 ■ Ectopic Pregnancy. Transvaginal ultrasound image of a right ectopic pregnancy with a decidual reaction in the uterus resembling a gestational sac, or “pseudosac.” Visualization of a pseudogestational, or “single,” sac sign could be consistent with an early gestational sac or an ectopic pregnancy with a uterine decidual cast. (Photo contributor: Janice Underwood.)

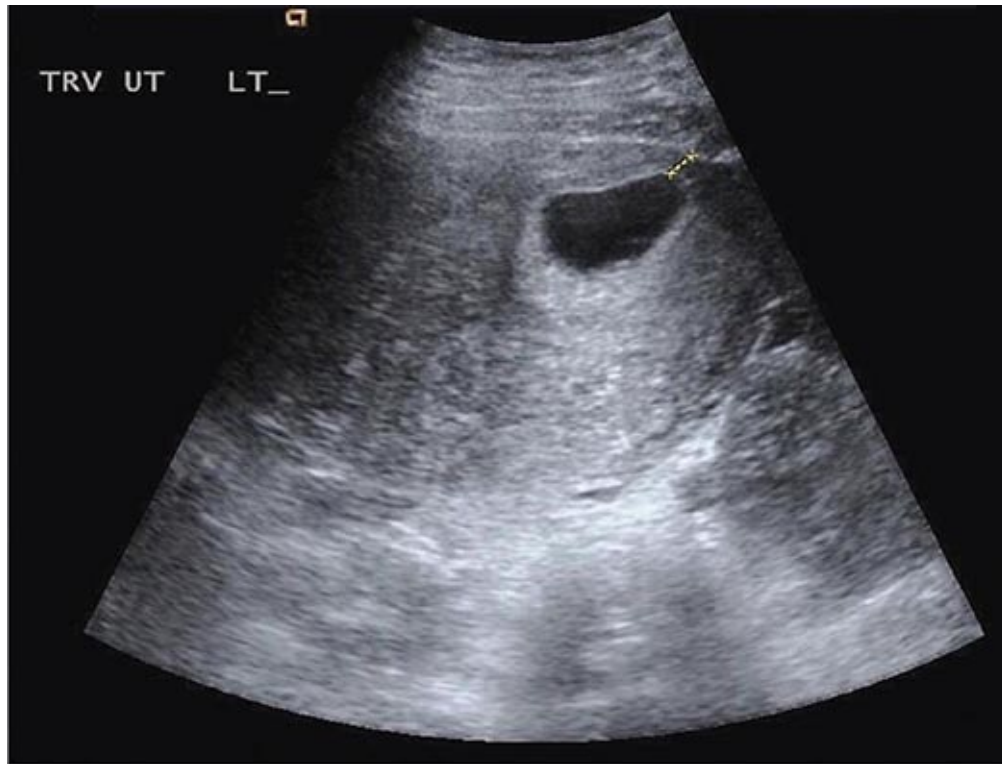


FIGURE 10.45 ■ Interstitial Ectopic. An interstitial ectopic exists when the gestational sac is less than 5 mm away from the uterine wall. This formal ultrasound confirmed a bicornate uterus and viable pregnancy with a distance of 6.1 mm, enough endometrium to support normal pregnancy. (Photo contributor: Lauren Oliveira, DO.)



FIGURE 10.46 ■ Interstitial Ectopic. This MRI confirmed a formal ultrasound of an interstitial ectopic. (Photo contributor: Lauren Oliveira, DO.)

MOLAR PREGNANCY (HYDATIDIFORM MOLE)

Clinical Summary

Obtain gynecologic consultation for dilatation and curettage in all cases. Close monitoring of serum hCG levels is required to rule out the presence of malignant gestational trophoblastic disease.

Management and Disposition

Molar pregnancy is part of a spectrum of gestational trophoblastic tumors that

include benign hydatidiform moles, locally invasive moles, and choriocarcinoma. The classic clinical presentation is painless first-trimester or early second-trimester vaginal bleeding with a uterine size larger than the estimated gestational age based on the last menstrual period. Signs of preeclampsia (hypertension, headache, proteinuria, and edema) in the 1st trimester or early 2nd trimester are highly suggestive of this diagnosis as well. Hyperthyroidism is found in roughly 5% of cases. Acute respiratory distress may occur due to embolization of trophoblastic tissue into the pulmonary vasculature, thyrotoxicosis, or simple fluid overload.

Pearls

1. All patients with pregnancies of less than 20 weeks' gestation with clinical findings of preeclampsia should be considered to have gestational trophoblastic disease until it is ruled out.
2. A “snowstorm” pattern on ultrasonography (demonstrating multiple intrauterine echoes with no fetus) coupled with a high hCG level is typical of molar pregnancy. Molar pregnancies may also resemble a “cluster of grapes” on US.
3. “Moles” commonly produce serum hCG levels greater than 100,000 mIU/mL.



FIGURE 10.47 ■ Molar Pregnancy. “Snowstorm” pattern demonstrating multiple intrauterine echoes with no fetus seen on transvaginal ultrasonography in a patient with a molar pregnancy. Serum β -hCG was greater than 180,000 mIU/mL. (Photo contributor: Robin Marshall, MD.)



FIGURE 10.48 ■ Molar Pregnancy. Transabdominal study showing molar pregnancy. These are exceedingly difficult to identify and diagnose by ultrasound. They can be easily mistaken for nonspecific intrauterine findings (eg, missed abortion, fibroid). (Photo contributor: Lauren Oliveira, DO.)

FAILED INTRAUTERINE PREGNANCY

Clinical Summary

Fetal viability may be in question for some pregnant patients presenting to the ED. Conservative transvaginal US diagnostic criteria for a failed IUP include a crown-rump length of 7 mm with no cardiac activity; a mean GS diameter of ≥ 25 mm and no visible embryo; no heartbeat ≥ 2 weeks after a visualized GS without a yolk sac; no heartbeat ≥ 11 days after a prior documented GS with a yolk sac; and no heartbeat ≥ 2 weeks after a prior documented GS without a yolk sac.

Management and Disposition

Obtain gynecologic consultation and follow-up to confirm ED findings for patients whose fetal viability is in question. Consider repeat US to confirm the diagnosis in stable, asymptomatic patients. For patients with findings suspicious for but not meeting criteria for a failed IUP, refer for outpatient gynecologic evaluation and arrange repeat US in 7 to 10 days.

Pearl

1. Follow strict diagnostic criteria for failed IUP to minimize the possibility of a false-positive finding (ie, incorrectly diagnosing nonviability).



FIGURE 10.49 ■ Indeterminate Viability. This fetal pole with a crown-rump length of 5.8 mm and no cardiac activity is consistent with indeterminate viability versus a failed IUP, which is diagnosed if crown-rump length is ≥ 7 mm and no cardiac activity. (Photo contributor: Kevin J. Knoop, MD, MS.)

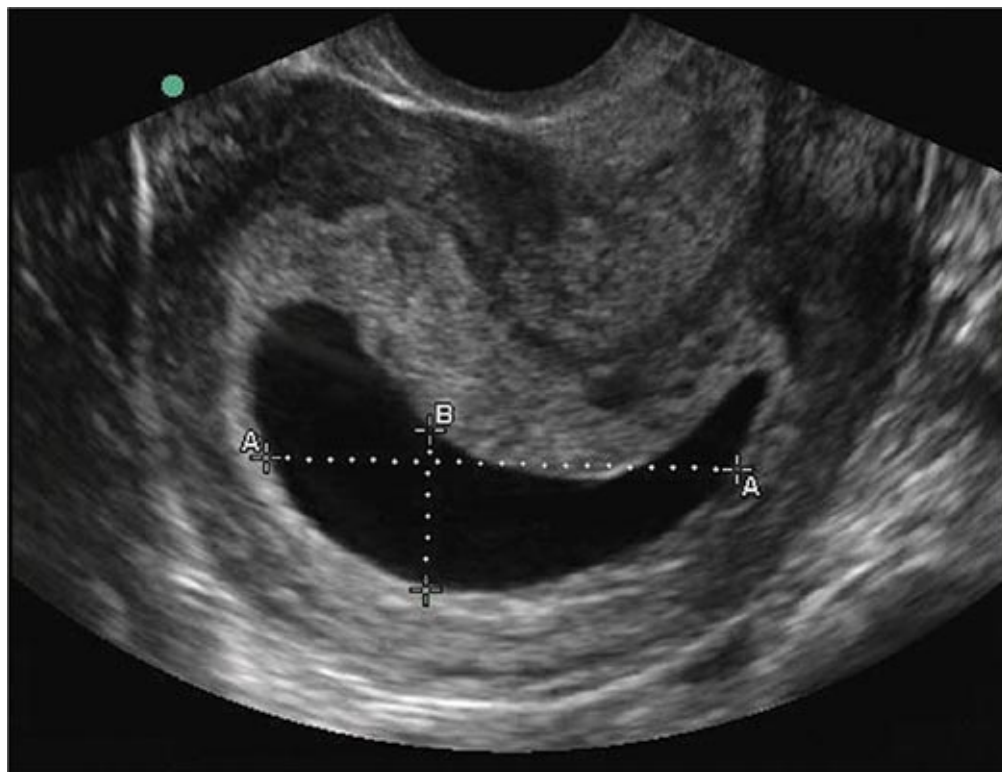


FIGURE 10.50 ■ Failed Intrauterine Pregnancy. This enlarged gestational sac with no visible cardiac activity is consistent with a failed IUP if the mean gestational sac diameter is ≥ 25 mm. (Photo contributor: Lauren Oliveira, DO.)

THIRD-TRIMESTER BLUNT ABDOMINAL TRAUMA

Clinical Summary

Trauma is a major cause of maternal and fetal mortality. In addition to injuries to abdominal organs seen in nonpregnant patients, preterm labor, fetal-maternal hemorrhage, uterine rupture, and, most importantly, abruptio placentae may result from blunt trauma during pregnancy. Abruptio placentae, the premature separation of the placenta from the site of uterine implantation, is found in up to 50% of major blunt trauma patients and up to 5% of those with apparent minor injuries. Signs of uterine hyperactivity and fetal distress are commonly seen when significant placental detachment occurs. Most patients have vaginal bleeding, but up to 20% will present with little or no external bleeding when the margins of detachment are above the cervical os.

Electronic fetal monitoring for a minimum of 4 hours is indicated in all cases of significant trauma in patients beyond 20 weeks' gestation. Normal fetal heart rates average between 120 and 160 bpm. Rapid, frequent fluctuations in the baseline are characteristic of normal "reactivity." The loss of this reactivity can occur during a normal fetal sleep cycle, following narcotic administration, or in the setting of fetal hypoxia or distress. Significant transient decelerations in fetal heart rate are classified as *early*, *late*, or *variable* depending on how they correlate with uterine contractions (via tocometry). *Late* and *variable* decelerations are concerning for fetal distress due to maternal hypotension and/or hypovolemia in trauma.

Management and Disposition

In addition to a standard trauma evaluation, immediate obstetrician consultation for all pregnant trauma patients beyond 20 weeks' gestation is imperative. Obtain blood for type and cross-matching, CBC, prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen, and fibrin degradation products or D-

dimer. Continuous tocometric fetal heart rate monitoring is required for a minimum of 4 hours to rule out preterm labor or fetal distress. US is essential in visualizing placental abruption and differentiates this from vaginal bleeding due to placenta previa or other causes. Indications for emergency cesarean section include placental abruption, signs of ongoing fetal distress, or uncontrolled maternal hemorrhage.

Pearls

1. Ecchymosis from blunt force may not develop on a gravid abdomen. A careful history of the mechanism of trauma and complaints is essential.
2. Anti-Rh immunoglobulin should be administered for all Rh-negative mothers with significant third-trimester blunt abdominal trauma.
3. Laboratory evidence of a consumptive coagulopathy may be seen with significant abruption.
4. Placental abruption, or abruptio placentae, can also occur spontaneously and should be considered in any pregnant patient at greater than 20 weeks' gestation with severe abdominal pain (with or without vaginal bleeding), uterine irritability, and/or fetal distress.

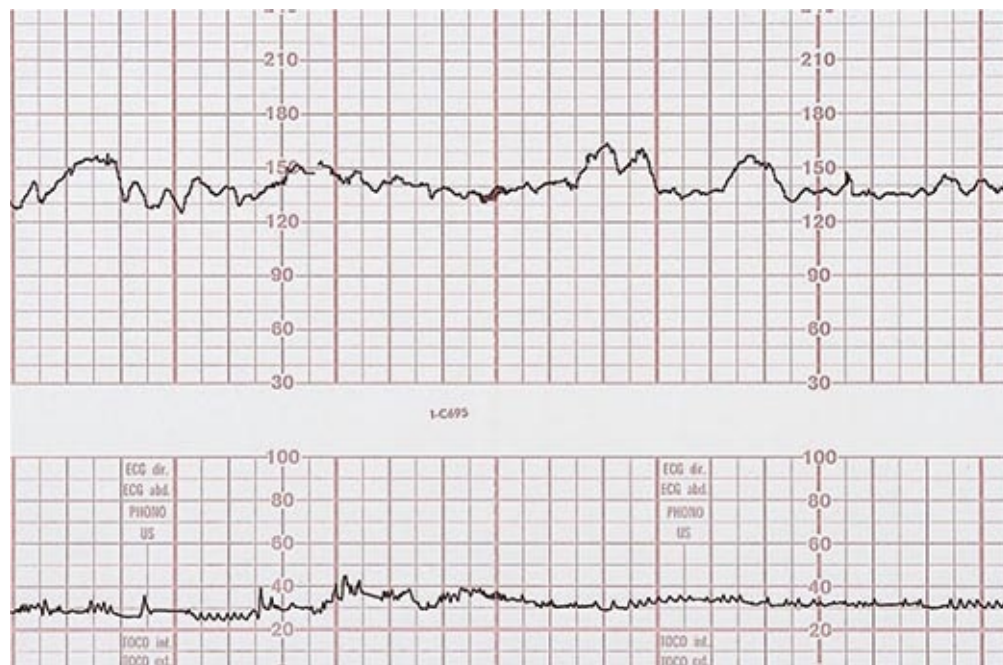


FIGURE 10.51 ■ Normal Beat-to-Beat Variability (BBV). A normal reactive fetal monitor strip showing a baseline heart rate between 120 and 160 bpm with fluctuations in the short- and long-term heart rate. (Photo contributor: Timothy Jahn, MD.)



FIGURE 10.52 ■ Loss of BBV. Loss of beat-to-beat variability (BBV) in the fetal heart rate, which may forewarn of fetal distress. This same pattern may also be seen during a normal fetal sleep cycle or following maternal narcotic administration. (Photo contributor: Gerard Van Houdt, MD.)

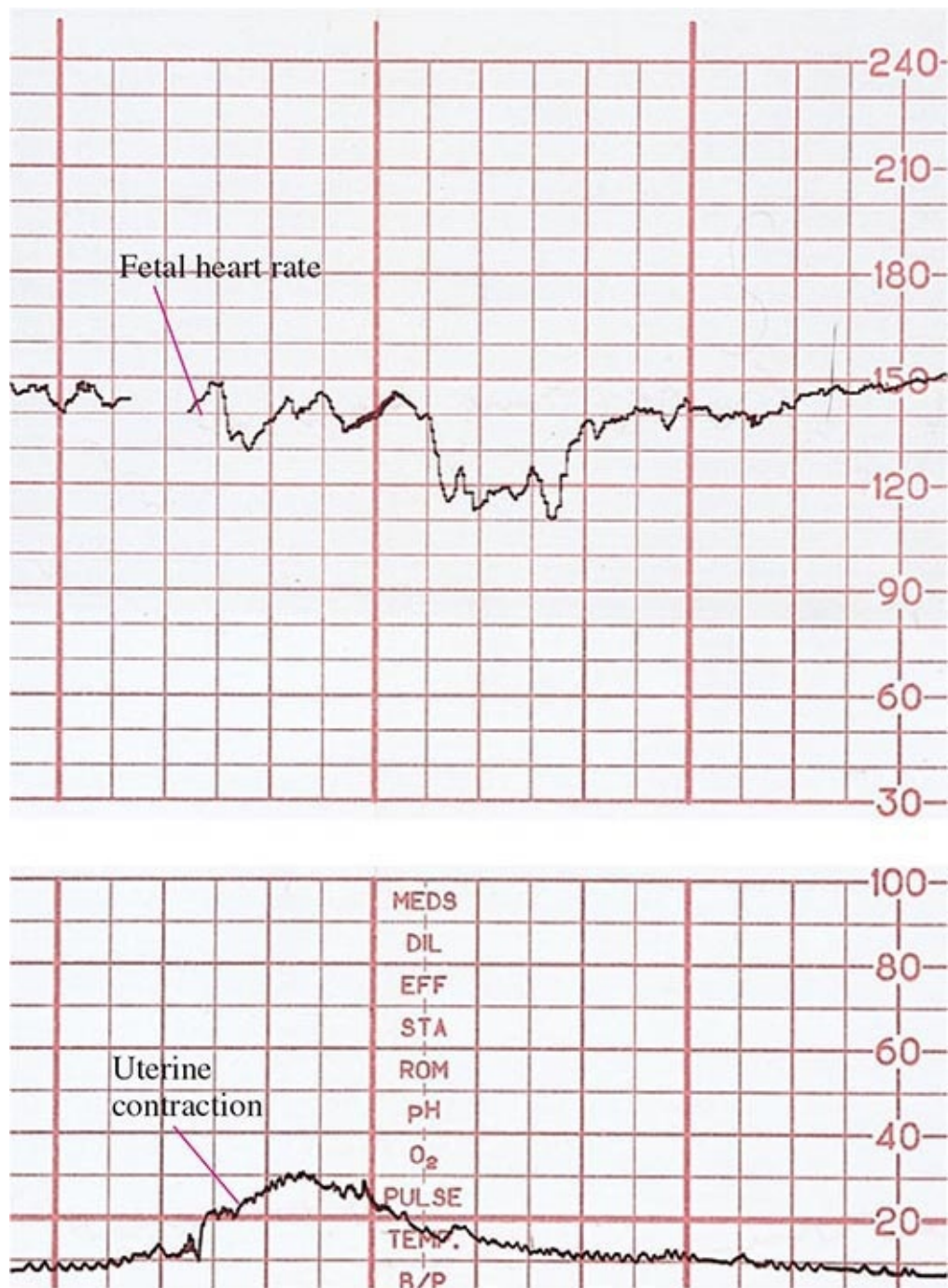


FIGURE 10.53 ■ Late Deceleration. The nadir of a late deceleration always follows the peak of the uterine contraction with the heart rate approaching the baseline after the completion of the uterine contraction; this is suggestive of hypoxia. (Photo contributor: James Palombaro, MD.)

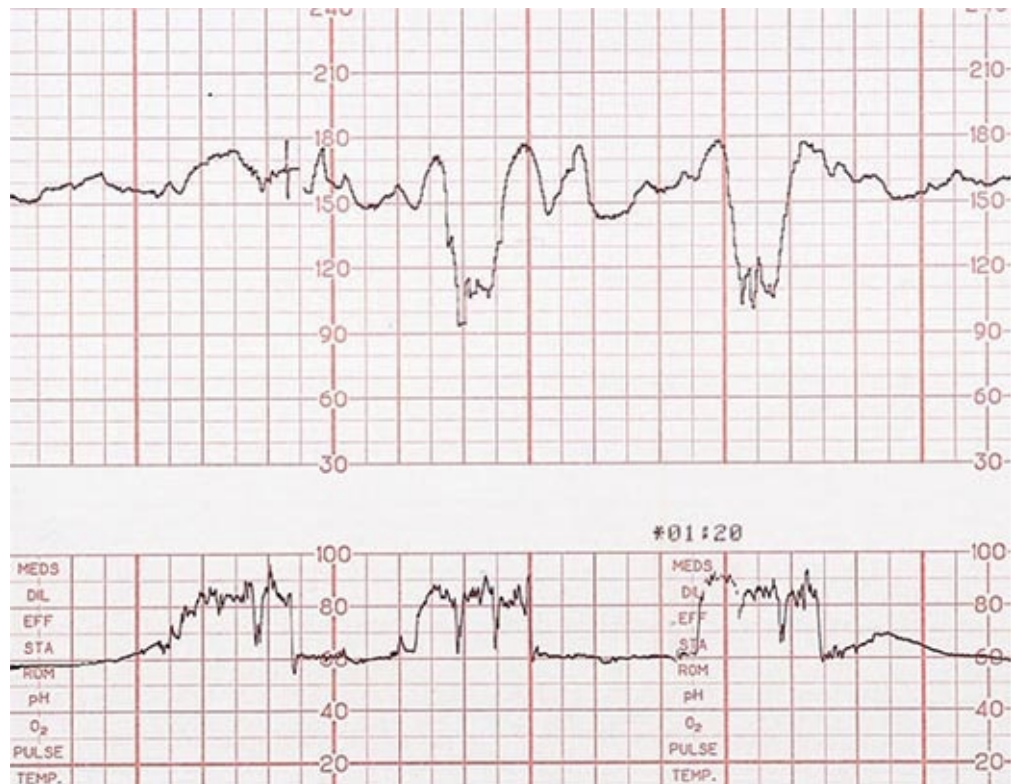


FIGURE 10.54 ■ Variable Deceleration. Variable decelerations are due to cord compression. They are characterized by a rapid onset and recovery and may occur slightly before, during, or after the onset of the contraction. They often include “peaks” on either side of the central deceleration. (Photo contributor: John O’Boyle, MD.)



FIGURE 10.55 ■ Gravid Abdomen. A third-trimester gravid abdomen with ecchymotic markings imparted by a significant blunt force. Fetal assessment should occur simultaneously with maternal resuscitation. (Photo contributor: John Fildes, MD.)

TABLE 10.2 ■ CHARACTERISTICS OF FETAL HEART RATE DECELERATIONS

Deceleration Type	Timing of Deceleration	Characteristics	Significance
Early	Starts after and ends before contraction	Coincides and “nadirs” with contraction peaks	Normal finding
Variable	Varies	Deep (even to < 100 bpm), broad, often have “shoulders”	May indicate cord compression or may be normal finding
Late	Starts and ends after contraction	Prolonged, nadir comes after contraction peaks	Fetal hypoxia

Emergency Vaginal Delivery

EMERGENCY DELIVERY: NORMAL VERTEX

DELIVERY SEQUENCE

Clinical Summary

A gravid woman may present in any stage of labor. Those with regular forceful contractions and the urge to push are in active labor (2nd stage) and may be near delivery. This stage begins when the cervix is fully dilated, which allows for the gradual descent of the fetal head toward the vaginal outlet. As the head approaches the perineum, the labia begin to separate with each contraction and recede once the contraction subsides. *Crowning* refers to separation of the labia by the head that does not recede at the end of the contraction. The appearance of crowning heralds imminent vaginal delivery. At this point, transfer from the ED is not advised and preparations for delivery should begin immediately.

Management and Disposition

Immediately obtain maternal intravenous access, begin continuous fetal monitoring, and prepare equipment for impending delivery and neonatal resuscitation (eg, suction, oxygen, warming light). Notify both obstetric and pediatric consultants of imminent ED delivery. Obtain important relevant history including gestational age, number of previous pregnancies, a diagnosis of twin/multiple gestations, prenatal care, and any problems with the pregnancy to date.

Delivery of the Head

Most commonly, the fetus is facing toward the mother's back. Extension of the fetal head occurs as it exits the vagina. During delivery of the head, support the maternal perineum with your hand and a clean, dry cloth to slow rapid fetal descent. This can reduce birth trauma to the mother. If needed, assist delivery of the fetal head by applying gentle pressure upward on the chin through the perineum (modified Ritgen maneuver) while simultaneously elevating the scalp to support head extension.

Once the head has been delivered, the occiput promptly rotates toward a left or right lateral position. At this stage, sweep the infant's neck checking for a nuchal cord (umbilical cord wrapped around the fetal neck). A nuchal cord can disrupt uterine blood flow during contractions, possibly leading to fetal distress represented on tocometry by variable decelerations ([Fig. 10.54](#)). If a nuchal cord

is identified, slip it over the infant's head. If the cord is wrapped too tightly to be reduced and impedes delivery, it can be clamped and ligated on the perineum, followed by the immediate delivery of the shoulders and body.

If thick meconium is present, use a mechanical suction catheter for deep suctioning of the posterior pharynx and glottic region prior to delivery of the infant's shoulders. Aspiration of thick meconium can lead to pneumonitis and hypoxia in the neonate. Inform consultants of the presence of any meconium detected.

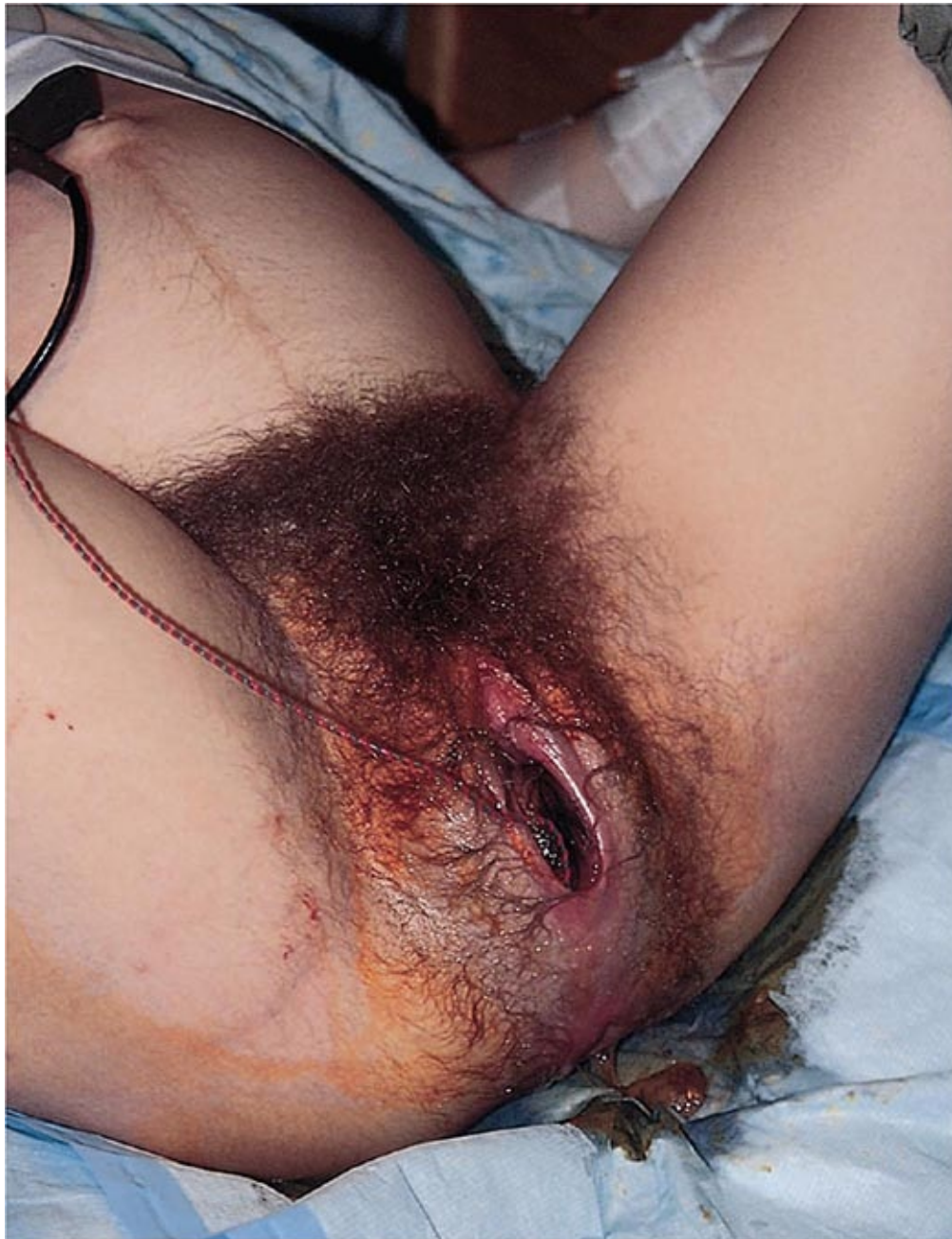


FIGURE 10.56 ■ Crowning. Descent of the fetal head with separation of the labia is known as crowning and heralds imminent vertex delivery. (Photo contributor: William Leininger, MD.)

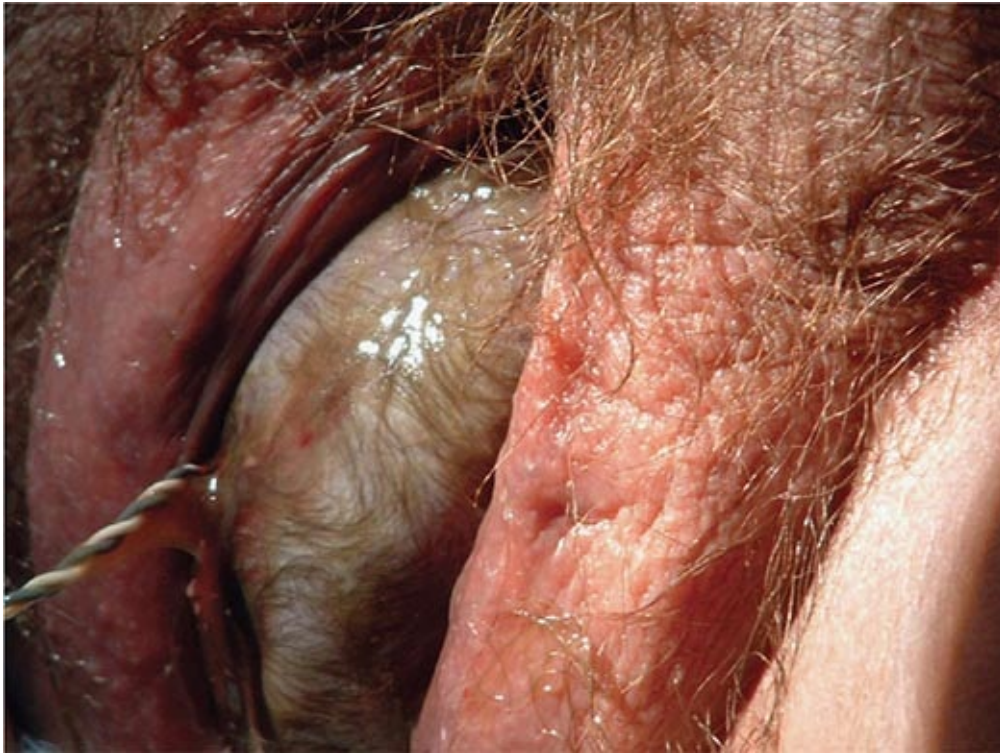


FIGURE 10.57 ■ Meconium. Meconium (greenish brown fetal stool), seen covering the scalp and perineum, is associated with fetal hypoxia and is a clinical indicator of fetal distress. Fetal bradycardia or late decelerations may be present and are also evidence of fetal distress. (Photo contributor: William Leininger, MD.)



FIGURE 10.58 ■ Modified Ritgen Maneuver. Modified Ritgen maneuver: upward pressure is applied on the fetal chin through the perineum. (Photo contributor: William Leininger, MD.)

Delivery of the Shoulders

Delivery of the shoulders generally occurs spontaneously with little manipulation. Occasionally, gentle downward traction applied by grasping the sides of the head with two hands is needed to ease the delivery of the anterior shoulder. The head can then be directed upward to permit the delivery of the posterior shoulder. Following delivery of both shoulders, the body and legs are easily delivered. Attention is then directed toward the immediate care of the newborn. The cord is doubly clamped, ligated, and inspected for three vessels: two umbilical arteries and one umbilical vein. The pediatrician should be notified of a two-vessel umbilical cord. The newborn is immediately placed under a warming lamp for drying, gentle stimulation and observation for signs of distress (heart rate < 100 bpm, limp muscle tone, poor color, or weak cry).



FIGURE 10.59 ■ Nuchal Cord. A loose nuchal cord is seen around the neck. (Photo contributor: William Leininger, MD.)



FIGURE 10.60 ■ Anterior Shoulder Delivery. Delivery of the anterior shoulder is facilitated with gentle downward traction. (Photo contributor: William Leininger, MD.)

Delivery of the Placenta

Following delivery, place gentle traction on the cord while massaging the uterine fundus with your opposite hand. Avoid strong traction on the cord during or after delivery as this can tear the placenta and result in fetal or maternal hemorrhage. The placenta is generally delivered within 20 minutes and should be grossly inspected for evidence of a missing segment or satellite placenta. Notify the obstetrician of these findings. Retained placental fragments may warrant manual exploration of the uterus and can cause postpartum hemorrhage and/or endometritis. Unusual placental vasculature, such as a vena-mentous placenta, should also be communicated to obstetrician and pediatric consultants.

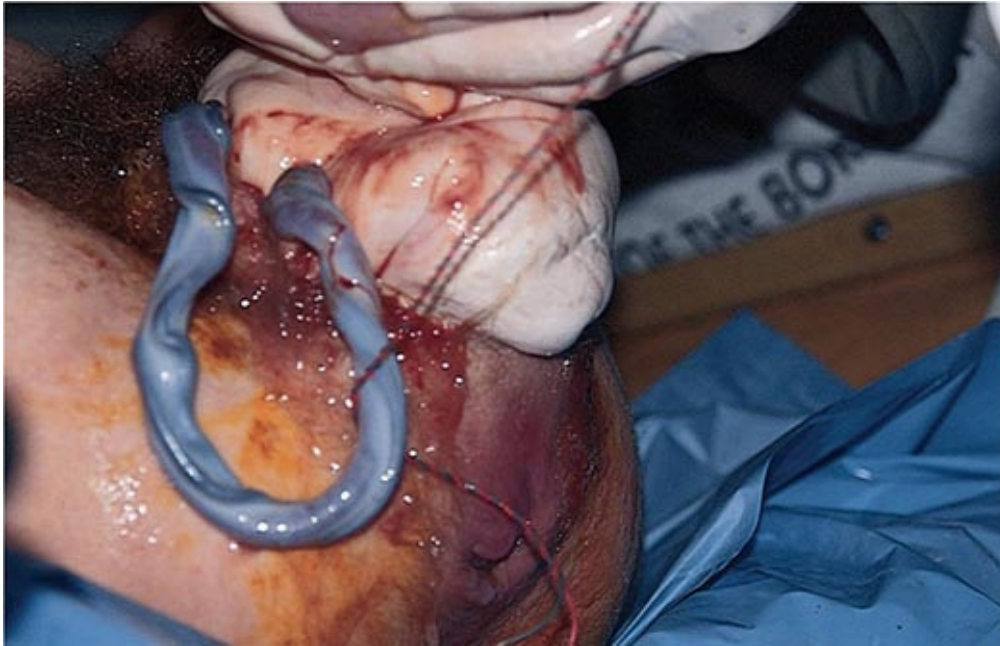


FIGURE 10.61 ■ Posterior Shoulder Delivery. Delivery of the posterior shoulder with gentle upward traction. (Photo contributor: William Leininger, MD.)



FIGURE 10.62 ■ Clamping the Cord. The cord is clamped immediately after delivery. (Photo contributor: William Leininger, MD.)

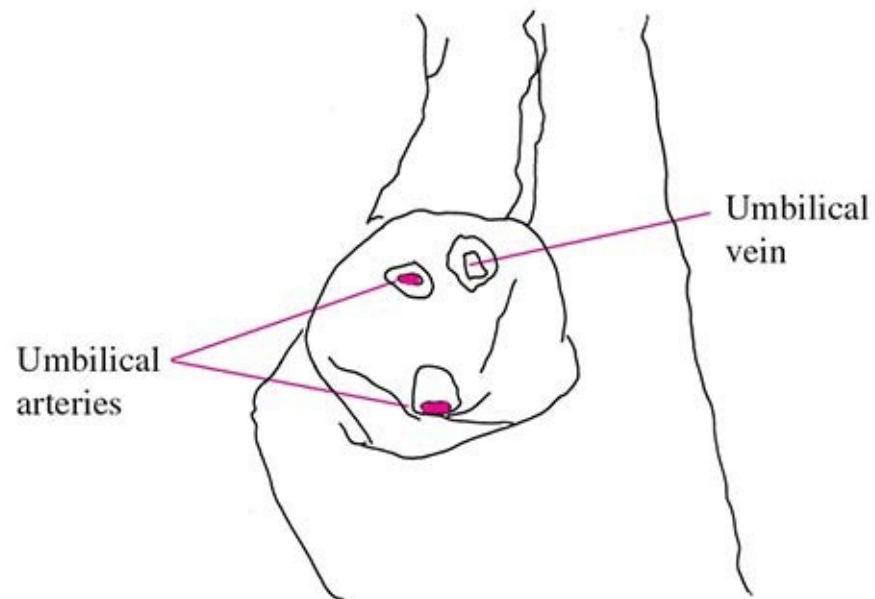


FIGURE 10.63 ■ Normal Umbilical Cord. Cross-sectional view of the two arteries and single vein of a normal three-vessel umbilical cord. (Photo contributor: Jennifer Jagoe, MD.)



FIGURE 10.64 ■ Placenta Delivery. Gentle traction is applied to the cord while the opposite hand massages the uterus. (Photo contributor: William Leininger, MD.)

Pearls

1. Maternal oxygen administration may help to alleviate fetal distress as seen on heart rate monitoring (see Third-Trimester Blunt Abdominal Trauma).



FIGURE 10.65 ■ Placenta Delivery. Delivery of the placenta. (Photo contributor: William Leininger, MD.)

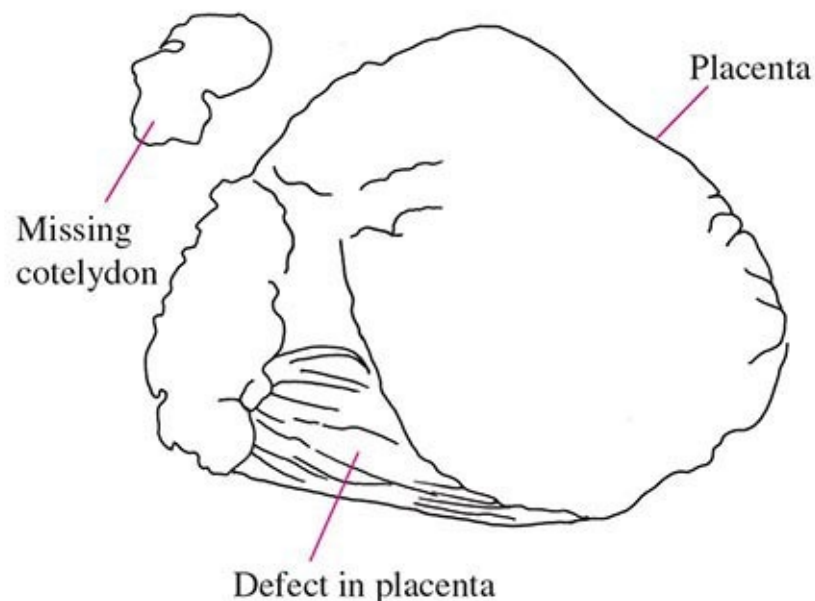
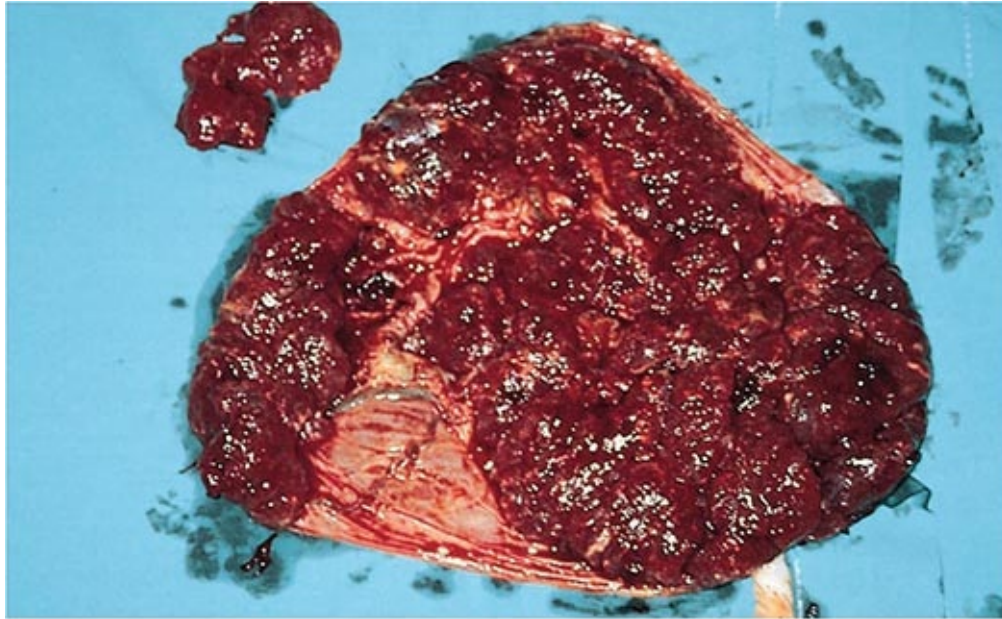


FIGURE 10.66 ■ Placenta. Placenta with a missing segment or cotyledon. The missing placental tissue can be seen in the upper left-hand corner of the photograph. (Photo contributor: John O'Boyle, MD.)

2. Nuchal cord occurs in about 20% of all deliveries. Occasionally two coils are identified.
3. A two-vessel cord (rare) is associated with an increased incidence of congenital defects.
4. Primigravida patients may still require many contractions and pushing to deliver even when crowning on presentation. Regardless, once crowning is present, be prepared for imminent delivery.

5. Guidelines from the Neonatal Resuscitation Program no longer recommend routine suctioning of the infant's mouth and nose at birth as this practice may cause bradycardia in the newborn. Wipe with a soft cloth instead.

BREECH DELIVERY

Clinical Summary

The incidence of singleton breech presentation is low (~3%) but increases (> 20%) in preterm infants weighing less than 2 kg. Frank breech is most common in full-term deliveries and presents with both hips flexed and both knees extended. In a complete breech, both hips and knees are flexed. In both of these, the infant's buttocks are the usual presenting body part at the perineum. A footling breech has one or both legs extended below the buttocks and a foot is the presenting part. Footling presentation is seen in up to half of all preterm deliveries. Breech deliveries carry a much higher neonatal mortality rate than normal deliveries. Complications of breech delivery include umbilical cord prolapse, nuchal arm obstruction, and difficulty in delivery of the head.

Management and Disposition

The specific maneuvers for breech extraction are beyond the scope of this text. If breech delivery appears imminent, prepare for ED delivery as in the previous section and obtain immediate obstetric and pediatric consultation. During delivery, support the infant's presenting parts and apply gentle traction as they spontaneously pass through the vaginal outlet. Keep in mind that the head diameter is greater than either the hip or shoulder diameter and may become entrapped by the cervix if it is not adequately dilated.

Pearl

1. Whenever possible and delivery is not imminent, immediately transfer stable patients with a breech presentation to an appropriate labor and delivery setting.



FIGURE 10.67 ■ Breech Delivery. Footling breech vaginal delivery of the following head. (Photo contributor: John O’Boyle, MD.)

UMBILICAL CORD PROLAPSE IN EMERGENCY DELIVERY

Clinical Summary

In an overt cord prolapse, a loop of umbilical cord is visualized either at the introitus or on sterile speculum examination following membrane rupture. Alternatively, a small loop of cord may be palpated at the cervical os. In a “funic” cord prolapse, a loop of umbilical cord is palpated through intact fetal membranes. Occult prolapse occurs when the umbilical cord descends between the presenting part and the lower uterine segment, but is not visible or palpable on examination. Intermittent compression of the umbilical cord with each uterine contraction may be indicated by variable fetal heart rate and decelerations. Fetal hypoxia may ensue if cord compression is sustained beyond the duration of the contraction, which often happens with overt prolapse.

Management and Disposition

Prolapse of the umbilical cord presents an immediate threat to fetal oxygenation and constitutes a true obstetrical emergency. If an overt prolapse is detected in the ED, do not attempt vaginal delivery. Immediately place the patient in a knee-chest position, consult an obstetrician, and transport the patient directly to the operating room for cesarean delivery. During transport, apply and maintain continuous upward pressure on the fetal presenting part to relieve pressure on the lower uterine segment and cord. Neonatal resuscitative equipment should be available in anticipation of a hypoxic infant.

Pearls

1. Pelvic examination to exclude umbilical cord prolapse should be performed immediately following rupture of membranes, appearance of new variable decelerations, or detection of fetal bradycardia.
2. Rupture of intact membranes with a funic cord may cause or worsen cord prolapse and should never be performed in cases of suspected cord prolapse.



FIGURE 10.68 ■ Umbilical Cord Prolapse. Prolapsed umbilical cord visible at the vaginal introitus in a patient with twin gestations. (Photo contributor: Kevin J. Knoop, MD, MS.)

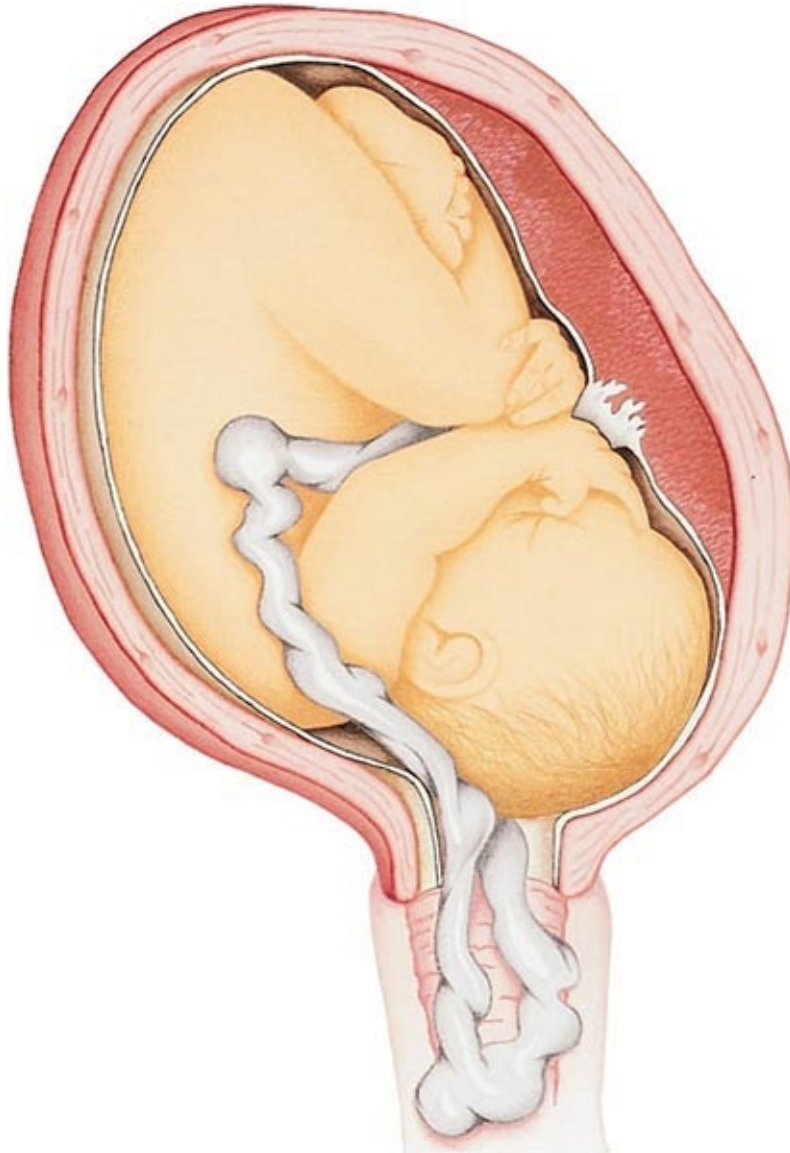


FIGURE 10.69 ■ Umbilical Cord Prolapse. Schematic drawing of an overt prolapse of the umbilical cord through a partially dilated cervical os. (Photo contributor: Judy Christensen.)

SHOULDER DYSTOCIA IN EMERGENCY DELIVERY

Clinical Summary

Shoulder dystocia is defined as failure to deliver the fetal shoulders following delivery of the head. Dystocia is caused by impaction of the fetal shoulders

against the pelvic outlet. Often, it is diagnosed by retraction of the fetal head against the perineum after delivery (“turtle sign”). Risk factors include gestational diabetes, prior shoulder dystocia or delivery of large infants, and postterm delivery.

Management and Disposition

Shoulder dystocia is an acute obstetric emergency due to the immediate threat of fetal asphyxia from compression of umbilical cord and chest. Immediate obstetric and pediatric consultation is imperative. Prepare equipment for delivery, possible episiotomy, and neonatal resuscitation. Upon identification of a shoulder dystocia, attempt McRobert maneuver (the least invasive procedure). This maneuver involves flexing the mother’s knees toward and past her chest in an extreme dorsal lithotomy position while simultaneously applying firm suprapubic pressure in an attempt to disengage the infant’s shoulder from the pelvic rim. Do *not* apply fundic pressure as this may worsen the fetal lodging against the pubic bone. If McRobert maneuver is unsuccessful, a Woods screw maneuver can be attempted by hooking two fingers behind the infant’s posterior scapula and rotating the entire body. As the anterior shoulder rotates downward, it can generally be delivered past the symphysis pubis. If the Woods maneuver fails to deliver the anterior shoulder, delivery of the posterior arm may be attempted by inserting two fingers into the sacral fossa and delivering the entire posterior arm by flexing it at the elbow. The remaining shoulder should then deliver spontaneously or following rotation into the oblique position to facilitate its delivery. In extreme cases, emergency cesarean may be necessary.

Pearls

1. Shoulder dystocia is an acute obstetric emergency that requires quick action.
2. After delivery, look for fractures of the infant’s clavicle or humerus and evidence of a brachial plexus injury. Damage to the 5th and 6th cervical nerve roots with resultant Erb palsy is the most common injury.



FIGURE 10.70 ■ Shoulder Dystocia. Firm approximation of the fetal head against the vaginal outlet consistent with shoulder dystocia. (Photo contributor: William Leininger, MD.)

POSTPARTUM PERINEAL LACERATIONS

Clinical Summary

Lacerations to the perineum most commonly occur following a rapid, uncontrolled delivery of the fetal head. Postpartum perineal lacerations range from minor to severe. First-degree lacerations are limited to the mucosa, skin, superficial subcutaneous, and submucosal tissues. Second-degree lacerations penetrate deeper into the superficial fascia and transverse perineal musculature. Third-degree lacerations extend through deeper tissues and disrupt the anal sphincter, while fourth-degree lacerations extend through rectal mucosa. These injuries can be associated with significant maternal blood loss and multiple long-term complications.

Management and Disposition

In ED deliveries, the repair of perineal lacerations can be deferred to an obstetrician, the details of repair being beyond the scope of this book. If maternal blood loss is significant, apply pressure to control bleeding while awaiting definitive repair.

Pearls

1. Warm perineal compresses and manual perineal support during delivery reduce the risk of significant vaginal trauma from birth.
2. Perineal laceration repair fundamentally involves the sequential anatomic reapproximation of the rectal mucosa, anal sphincter, transverse perineal musculature, vaginal mucosa, and skin using absorbable suture material.



FIGURE 10.71 ■ First-Degree Laceration. First-degree laceration limited to the mucosa, skin, and superficial subcutaneous and submucosal tissues. There is no involvement of the underlying fascia and muscle. (Photo contributor: Jerry Van Houdt, MD.)

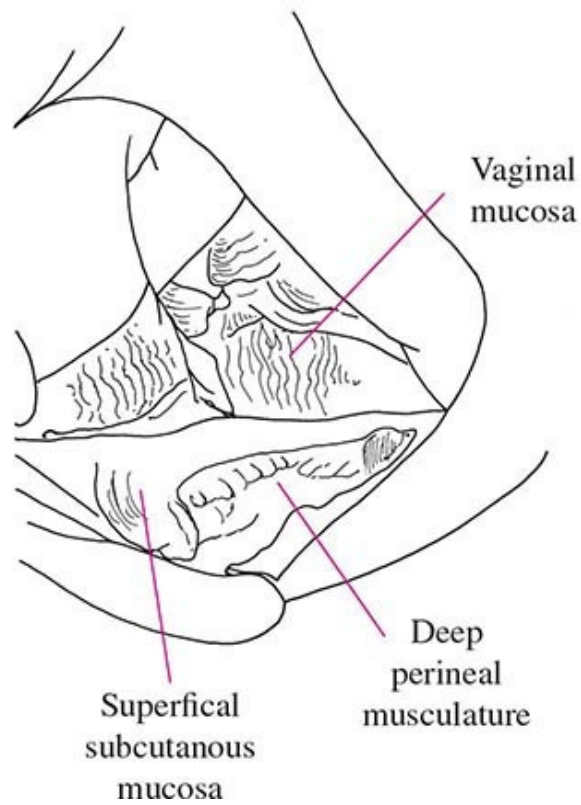


FIGURE 10.72 ■ Second-Degree Laceration. There is disruption of the hymenal ring and the deep perineal musculature, extending into the vaginal mucosa and transversalis fascia, but no involvement of the anal sphincter or mucosa. (Photo contributor: Pamela Ambroz, MD.)



FIGURE 10.73 ■ Fourth-Degree Laceration. Fourth-degree perineal laceration revealing wide separation of the perineal fascia and anal sphincter. The examiner's small finger is in the rectal lumen, showing extension of the tear proximally. (Photo contributor: Timothy Jahn, MD.)

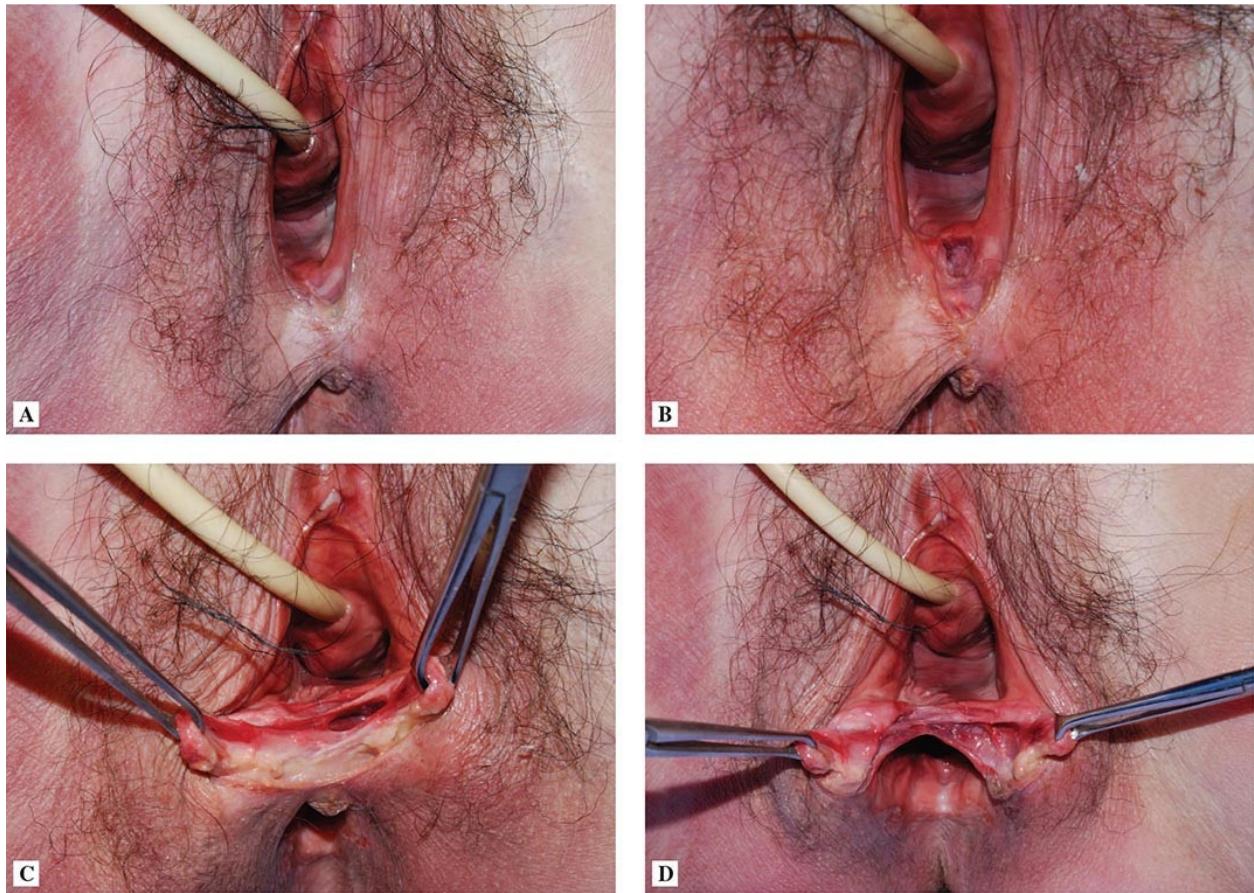


FIGURE 10.74 ■ Postpartum Perineal Lacerations. These cadaveric photographs illustrate the defining anatomy for first- through fourth-degree perineal lacerations. (A) First, (B) second, (C) third, and (D) fourth. (Photo contributor: Barbara Hoffman, MD.)

INTRAUTERINE FETAL DEMISE

Clinical Summary

If the mother is in active labor, prepare for a normal delivery. Obtain obstetric consultation immediately as for any emergency delivery. The presence of fetal heart tracings during delivery or a normal-appearing infant without significant skin changes indicates peripartum demise and neonatal resuscitation should be attempted. If signs of maceration are present at birth, no resuscitative efforts are indicated.

Management and Disposition

Intrauterine fetal demise (IUFD) refers to fetal death prior to delivery and may occur at any time prior to delivery. Determining the timing of the IUFD is important both for counseling the parents and for anticipating the likelihood of successful neonatal resuscitation after delivery. The appearance of the fetus at delivery can be used to estimate whether the fetal demise occurred antepartum or peripartum.

The process of tissue degeneration (maceration) is due to the effects of autolytic enzymes on the fetus in a sterile environment. Lysis occurs at the epidermal-dermal junction with subtle changes in the gross appearance of the fetus (skin desquamation, positive Nikolsky sign) seen as early as 6 hours. Further changes involve desquamation and bullae formation of the face, back, or abdomen by 12 hours, at least 5% of the body surface at 18 hours, and generalized skin desquamation at 24 hours. Sloughing of skin from a large area indicates a prolonged interval between death and delivery. Mummification occurs after approximately 2 weeks.

Pearls

1. Desquamation and/or sloughing of skin from large or numerous areas in an *unresponsive* fetus indicate antepartum IUFD.
2. With the exception of loss of heartbeat detected on live fetal monitoring, it may not be possible to pinpoint the time of fetal death prior to delivery. With loss of heartbeat in labor, assume the demise was peripartum and prepare for neonatal resuscitation.



FIGURE 10.75 ■ Intrauterine Fetal Demise. Desquamation of the entire skin surface, with sloughing of the abdominal and scrotal skin, is seen in this IUFD. (Photo contributor: Lawrence B. Stack, MD.)

POSTOPERATIVE COMPLICATIONS OF CESAREAN SECTION

Clinical Summary

Nearly one in four births are by cesarean section in the United States. As with any major surgery, there are several potential complications related to this procedure. For the mother, immediate complications include hemorrhage, uterine atony, and damage to the uterus and other abdominal organs. Immediately postoperatively, problems with the wound (infection and dehiscence), endometritis, and sepsis are possible. Complications with future pregnancies are long-term concerns. Patients may present to the ED with complaints of problems with the wound (bleeding or purulent discharge), erythema, tenderness, or dehiscence or with signs of pelvic infection or sepsis (fever, hypotension, pelvic

pain, purulent vaginal discharge, or increased bleeding).

Management and Disposition

Management of postoperative complications of cesarean section should involve gynecologic consultation in most cases and is dependent on the specific complication. Controlled minor bleeding or local wound infection can be addressed in the ED with close gynecology follow-up. For significant bleeding, place two large-bore IVs, control bleeding with pressure, and prepare blood products for transfusion as needed for blood loss. Exquisite uterine tenderness accompanied by fever and heavy vaginal bleeding and/or purulent discharge is concerning for endometritis. Suspected endometritis or puerperal sepsis should prompt obtaining blood cultures, fluid resuscitation, and empiric antibiotic administration. Wound dehiscence ranges from superficial with only dermal and epidermal involvement to complete with extravasation of abdominal contents. Immediate gynecologic consultation is indicated for any dehiscence beyond superficial tissues. If bowel or other organs are visible, they should be inspected for signs of strangulation/ischemia (pale or dusky-colored bowel) and covered with sterile gauze wet with saline. Do not attempt to replace the organs. This is a surgical emergency.

Pearls

1. Postcesarean rates of endometritis are quite high, and risk increases with length of labor prior to delivery. Up to 85% of women with prolonged labor followed by emergent cesarean section will develop endometritis without prophylactic antibiotics.
2. Postcesarean wound dehiscence is more common with vertical incisions than with low transverse.



FIGURE 10.76 ■ Cesarean Section Wound Dehiscence. Extrusion of intra-abdominal contents due to dehiscence of cesarean section wound on postoperative day 3. (Photo contributor: Kevin J. Knoop, MD, MS.)

¹The authors acknowledge the special contributions of Robert G. Buckley, MD, and Sean Lisse, MD for contributions to prior editions.

Chapter 11

EXTREMITY TRAUMA

Meghan Breed

Robert Warne Fitch



Degloving injury. (Photo contributor: Lawrence B. Stack, MD.)

ACROMIOCLAVICULAR JOINT SEPARATION

Clinical Summary

Injury to the acromioclavicular (AC) joint usually results from an impact on the superior aspect of the acromion. The classification system includes six types: type I: stretching of the AC ligament; type II: tearing of the AC ligaments and stretching of the coracoclavicular ligaments; and types III to VI: complete disruption of the AC and coracoclavicular ligaments.

Patients complain of pain at the AC joint and will actively splint the injured shoulder. Ecchymosis may be present; however, an obvious deformity is not always seen. There is significant tenderness upon AC joint palpation.

Standard radiographs should include anteroposterior (AP) and axillary lateral views of the shoulder. Type I injuries will appear normal. Type II injuries may show 0% to 50% displacement at the AC joint but no increase in the coracoclavicular interval. Types III to VI will demonstrate displacement at the AC joint and the clavicle will appear to be displaced superiorly (the acromion actually is rotated inferiorly) 50% to greater than 100% its width when compared with the normal side.

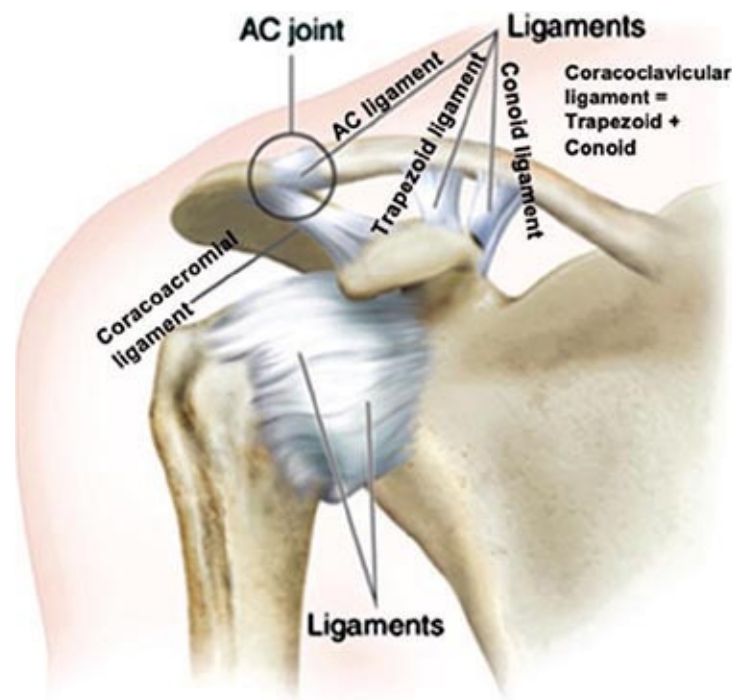
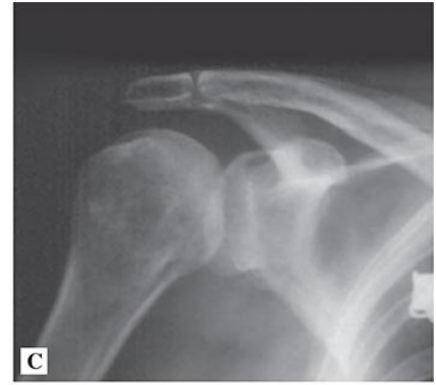


FIGURE 11.1 ■ Shoulder Ligaments. The ligaments that stabilize the glenohumeral and acromioclavicular joints. (Reproduced with permission from The Shoulder. In: Parks E, ed. *Practical Office Orthopedics*. New York, NY: McGraw Hill; Copyright 2018.)

Direction of
trauma force

A

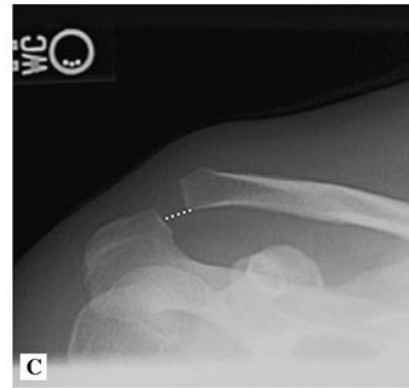
Type I



Direction of
trauma force

A

Type II



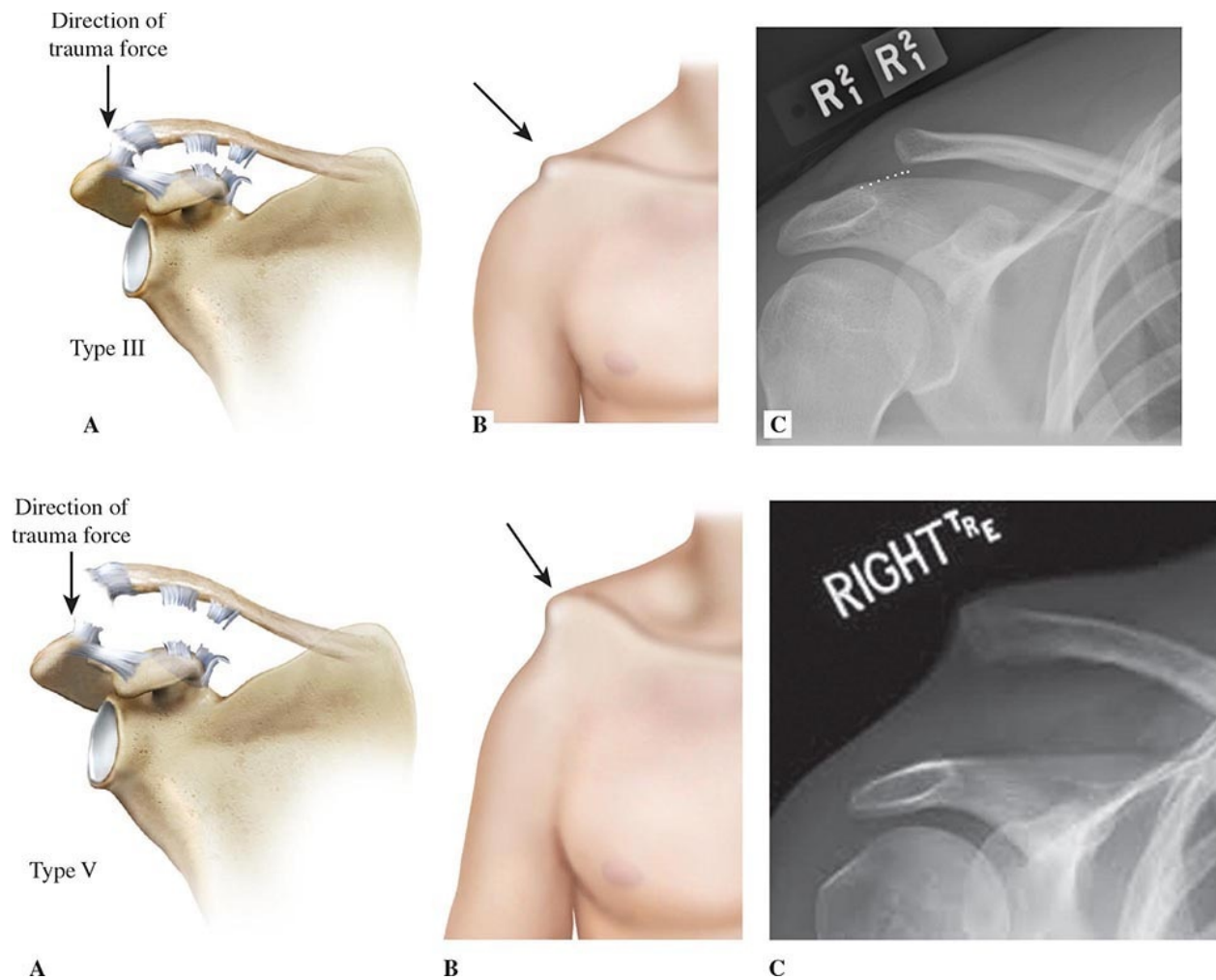


FIGURE 11.2 ■ AC Joint Separation Types. The anatomic basis (left), clinical appearance (middle, with arrow pointing to the AC joint deformity), and x-ray findings seen with a type I, II, III, and V AC joint separation (A-C). (Reproduced with permission from The Shoulder. In: Parks E, ed. *Practical Office Orthopedics*. New York, NY: McGraw Hill; Copyright 2018.)

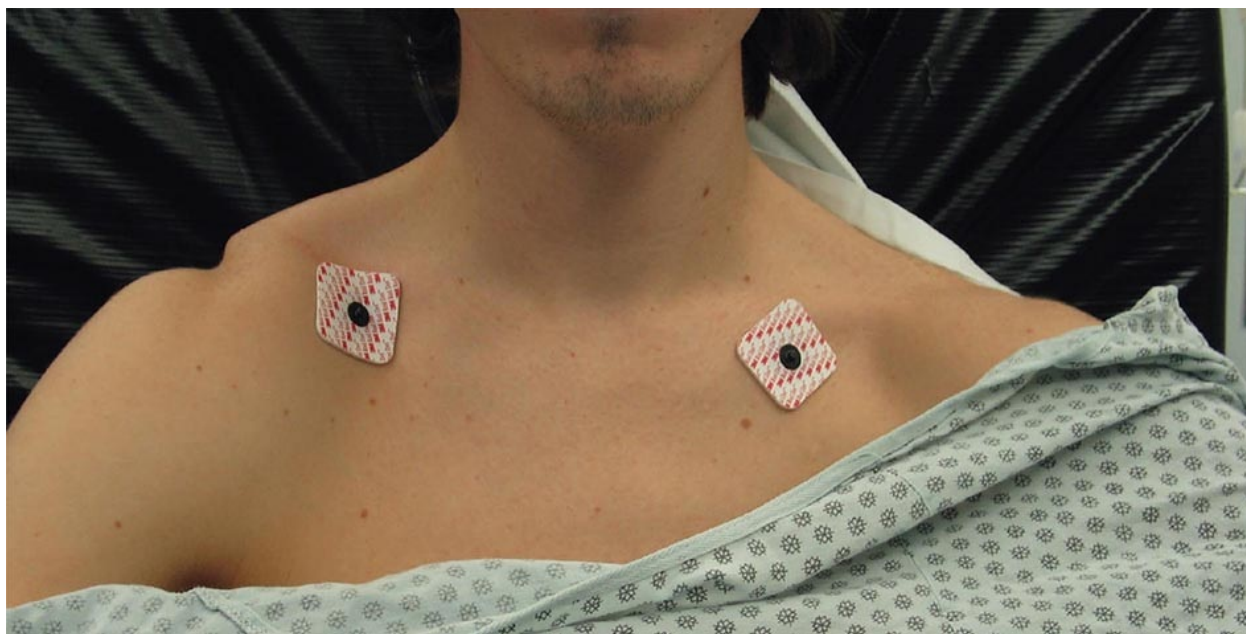


FIGURE 11.3 ■ AC Joint Separation. Large deformity at the right distal clavicle suggesting complete ligament disruption. (Photo contributor: R. Jason Thurman, MD.)

Management and Disposition

Types I and II injuries are treated with rest, ice, analgesics, and a simple sling until acute pain with movement is relieved. Type III injuries may be treated either nonoperatively or operatively; however, these patients can be discharged from the emergency department (ED) in a sling without an emergent orthopedic consult. Types IV, V, and VI are treated operatively. Referral to a musculoskeletal specialist is essential for all AC joint injuries since many patients who initially appear to have minor injuries will have more obvious deformity after the swelling and pain have subsided.

Pearls

1. The early AC joint stress radiograph can be negative due to splinting of the shoulder girdle muscles and does not add anything to acute management.
2. Differentiating between types I and II versus types IV to VI is the goal of the ED physician, since the latter will require an emergency orthopedic consult.

Clinical Summary

Anterior dislocations account for more than 90% of dislocations. They are frequently caused by falling with the arm externally rotated and abducted, causing patients to present with the affected extremity held in adduction and internal rotation due to pain. The acromion becomes prominent with loss of the rounded contour of the deltoid. Neurovascular exam of the upper extremity should be performed to rule out associated injury, most commonly of the axillary nerve (sensation over the deltoid) and musculocutaneous nerve (anterolateral forearm). Vascular injuries are rare. Standard radiographs to evaluate for fracture should include AP and either axillary lateral or scapular Y views.

Posterior shoulder dislocations are commonly missed because of subtle radiographic findings. The arm is held internally rotated and slightly abducted. Patients are unable to externally rotate their shoulder. On exam, a posterior prominence exists. Posterior dislocations can occur with a posterior-directed force as seen during grand mal seizures or electric shock.

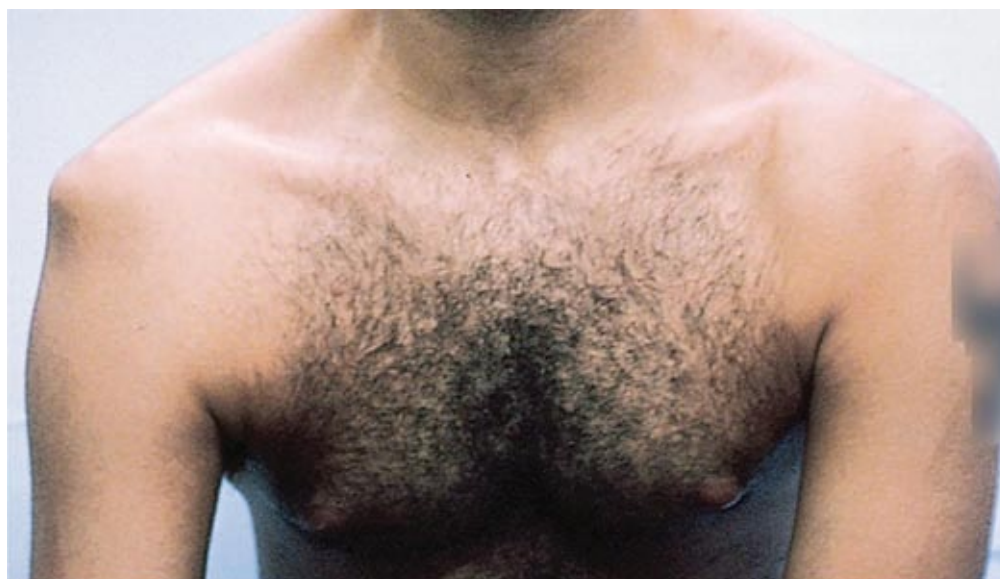


FIGURE 11.4 ■ Anterior Shoulder Dislocation. This right anterior shoulder dislocation occurred when the patient fell while playing basketball. There is an obvious contour deformity as well as prominence of the acromion. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Closed reduction is the treatment and should be completed as soon as possible to avoid humeral head avascular necrosis. Due to shoulder girdle spasm, conscious sedation is often required. There are many methods to reduce anterior shoulder

dislocations, including Stimson, Rockwood traction and countertraction, and Milch. The basic premise is to apply axial traction, externally rotate, and abduct. Neurovascular and radiographic examination should occur before and after reduction. The patient should be placed in a sling after reduction, and follow-up with a musculoskeletal specialist is recommended.

Pearls

1. Occult, nondisplaced greater tuberosity fracture can be identified on postreduction radiographs. These usually occur during the traumatic event, not as a result of the reduction, and are usually only identified when there is no radiographic bony overlap.
2. Luxatio erecta is inferior glenohumeral dislocation and is rare. The humeral head is forced below the inferior aspect of the glenoid fossa due to arm hyperabduction. These patients present with the arm locked fully abducted and externally rotated. Axillary nerve injury is reported to occur in 60% of cases. Vascular injury occurs most frequently with this type of dislocation. Reduction is accomplished with overhead traction.
3. Posterior shoulder dislocations can be bilateral and are often missed due to preserved symmetry on standard chest x-rays.
4. Hill-Sachs lesions (an impaction fracture of the postero-lateral humeral head) can occur in up to 50% of anterior shoulder dislocations. Reverse Hill-Sachs lesions, also called McLaughlin lesions (impaction fracture of anteromedial aspect of humeral head), can occur in posterior shoulder dislocations.

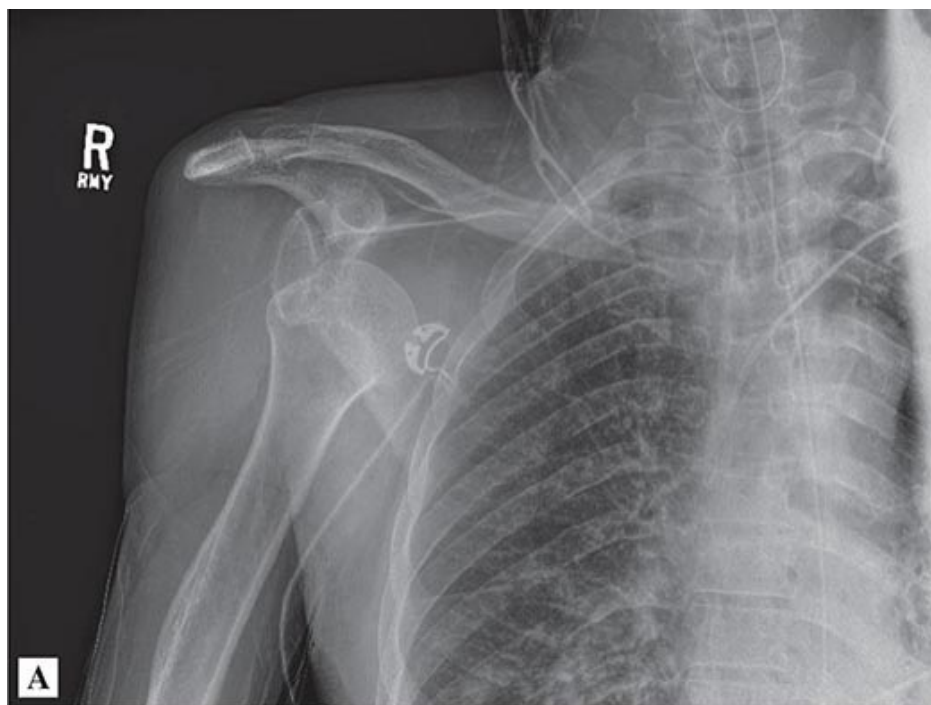


FIGURE 11.5 ■ Anterior Shoulder Dislocation. Radiographic evaluation demonstrates in both the AP (A) and scapular Y view (B) that the humeral head is not in the glenoid fossa but is located anterior and inferior to it. (Photo contributor: Meghan Breed, MD.)

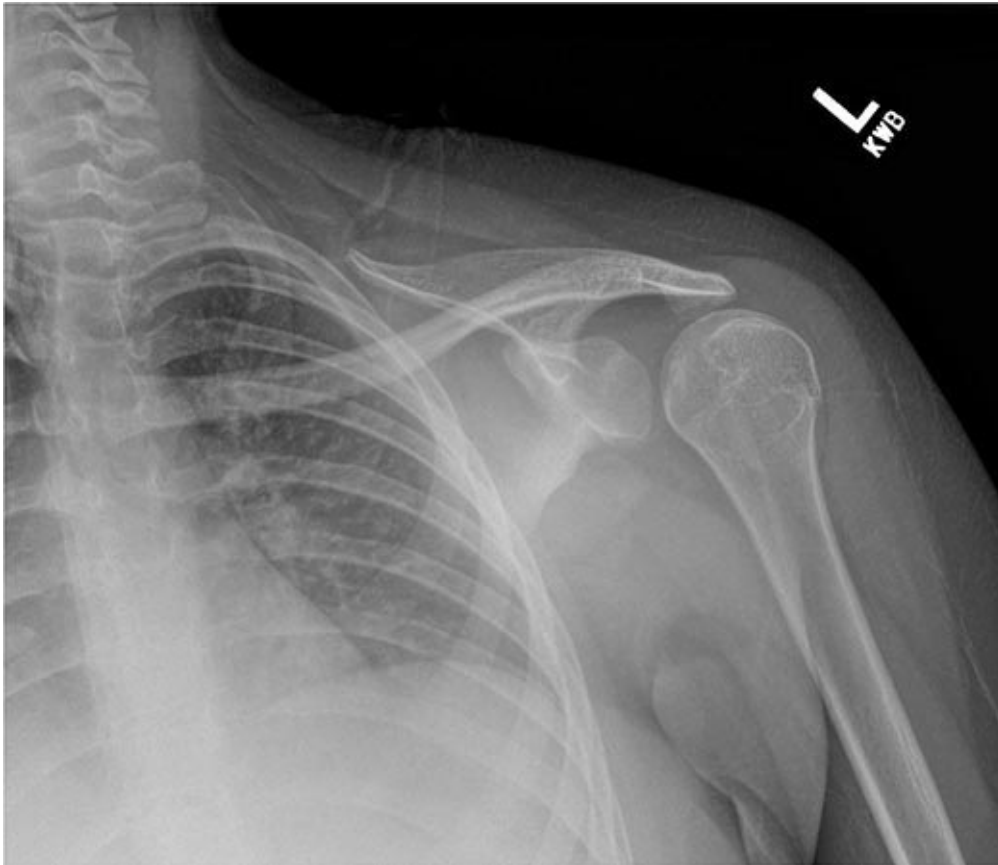


FIGURE 11.6 ■ Posterior Shoulder Dislocation. AP radiograph of this rare type of shoulder dislocation. Because of internal rotation of the greater tuberosity, the humeral head appears like a dip of ice cream on a cone, thus called the “ice cream cone sign.” (Photo contributor: Meghan Breed, MD.)

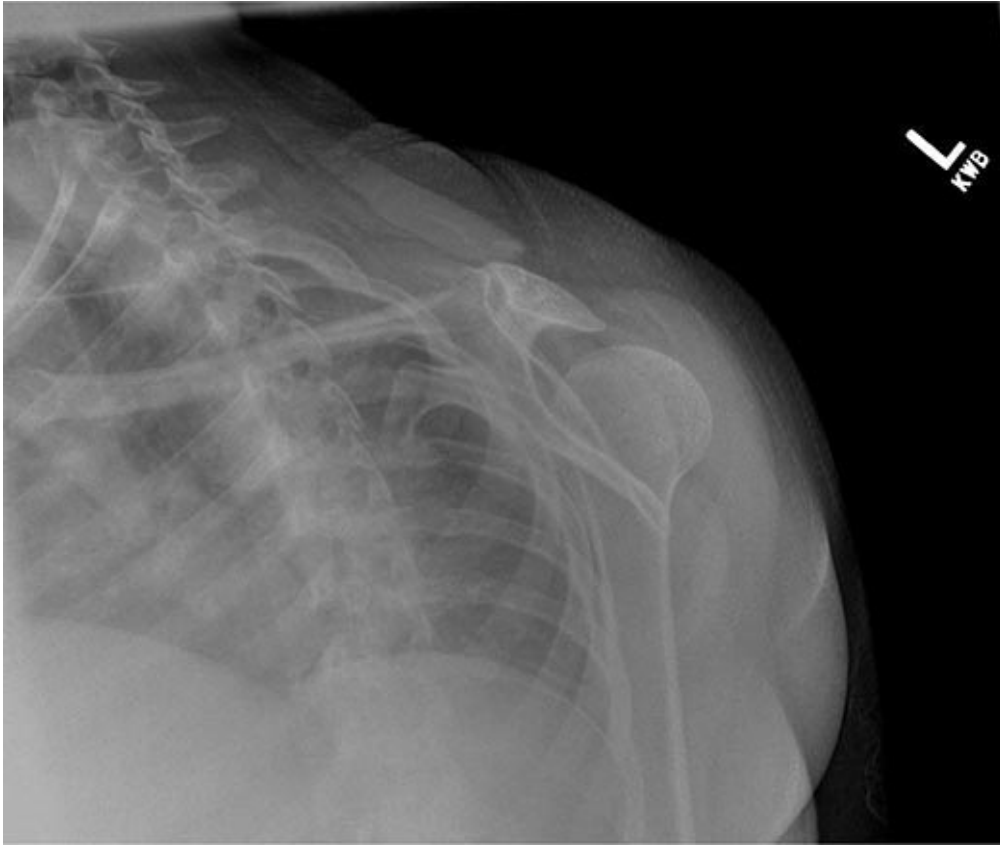


FIGURE 11.7 ■ Posterior Shoulder Dislocation. A scapular Y view of the same patient in Fig. 11.6 confirms the diagnosis. (Photo contributor: Meghan Breed, MD.)



FIGURE 11.8 ■ Luxatio Erecta. Hyperabduction may cause the relatively rare inferior dislocation known as luxatio erecta. The arm is held in elevation and the humeral head may be palpated along the lateral chest

wall. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 11.9 ■ Luxatio Erecta. An inferior dislocation of the humeral head with the arm above consistent with luxatio erecta. (Photo contributor: Benjamin Milligan, MD.)

ELBOW FRACTURES

Clinical Summary

Direct trauma or fall on an outstretched hand may result in elbow fractures. The patient may be unable to extend the elbow and have pain on supination/pronation. AP, lateral, and oblique radiographic views can visualize most fractures. The radial head should be aligned with the capitellum on all views. The anterior fat pad may be seen on normal radiographs, but displacement anteriorly and superiorly (sail sign) suggests effusion or hemarthrosis. The posterior fat pad is not normally visualized, but if seen is indicative of effusion or hemarthrosis.

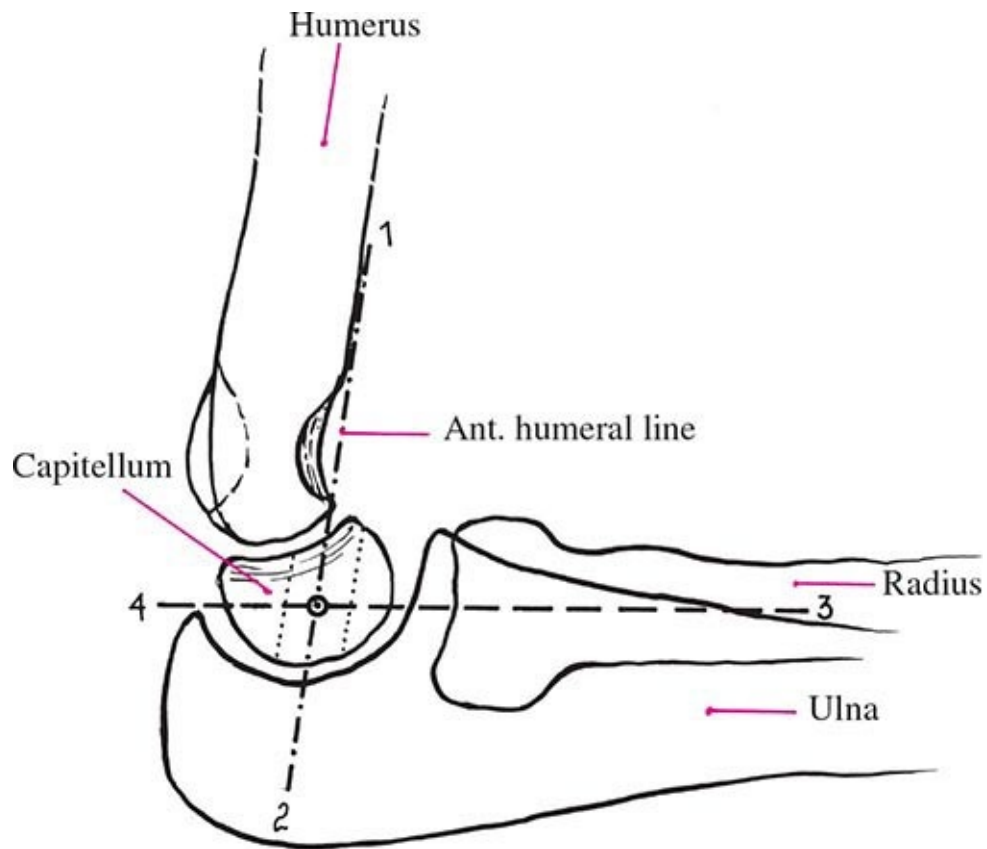


FIGURE 11.10 ■ Radiographic Elbow Relationships. In lateral views, the anterior humeral line (1-2) should bisect the middle third of the capitellum. The radiocapitellar line (drawn through the center of the radius, 3-4) should also pass through the center of the capitellum. Disruption of these relationships may indicate fracture.



FIGURE 11.11 ■ Type I Supracondylar Fracture. This radiograph shows both a pronounced anterior fat pad (sail sign) and posterior fat pad indicative of a supracondylar fracture. (Photo contributor: Alan B. Storrow, MD.)

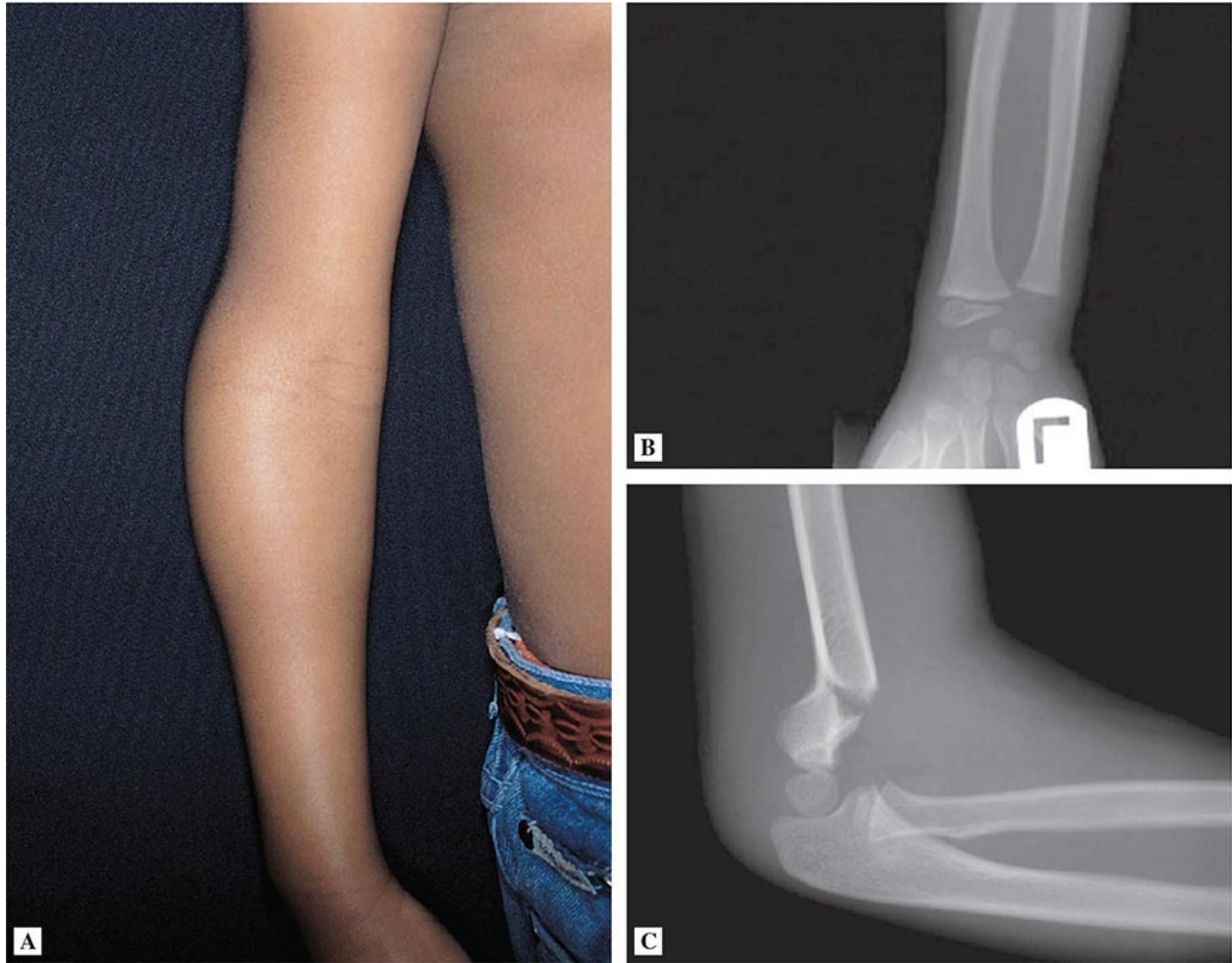


FIGURE 11.12 ■ Type II Supracondylar Fracture. (A) Clinical picture with marked soft tissue swelling in a child with a history of falling on an outstretched arm. (B and C) AP and lateral views of the elbow, demonstrating a distal humerus fracture with posterior displacement of the medial and lateral condyles. (Reproduced with permission from Shah BR, Lucchesi M. *Atlas of Pediatric Emergency Medicine*. New York, NY: McGraw Hill; 2006.)

Supracondylar fractures most often occur in pediatric patients. Neurovascular insult occurs in 7%, with the anterior interosseous nerve most commonly injured. This can be checked by having the patient make an “ok” sign. This neuropraxia usually resolves in 6 months. Arterial injury to the brachial artery occurs in 5% to 20%, with less than 1% of these complicated by compartment syndrome. Arteriography is indicated if the radial pulse is decreased after reduction; however, it should not delay operative evaluation.

Capitellum fractures represent less than 1% of adult elbow fractures and typically occur following high-energy trauma. These are difficult to diagnose radiographically and may require a computed tomography (CT) scan.

Management and Disposition

Treatment is influenced by stability of the fracture pattern as well as associated neurovascular injuries. If neurovascular compromise exists, the physician may need to apply forearm traction to reestablish distal pulses. If the pulse is not restored with traction, emergent operative intervention for brachial artery exploration or fasciotomy is indicated. In children, non-displaced fractures (type I) can be splinted in 90 degrees of flexion. Angulated or displaced fractures (type II and III) often require operative intervention.

Capitellum fractures are treated with immobilization in a posterior long-arm splint with the elbow in 90 degrees flexion and the forearm in supination. Complications of displaced capitellum fractures include arthritis, avascular necrosis, and decreased range of motion.



FIGURE 11.13 ■ Type III Supracondylar Fracture. Radiograph demonstrates complete disruption of the posterior cortex without any cortical contact. (Photo contributor: Lawrence B. Stack, MD.)

Radial head fractures can be treated with a sling for comfort and patients should be told to discontinue the sling as early as possible. Fractures that are

greater than 2 mm displaced, those with more than 20% of articular depression, or open fractures may need operative treatment and should follow-up with an orthopedic surgeon within 1 week.

Pearls

1. An intimal arterial injury may not be initially apparent, and frequent radial artery checks are warranted in the pediatric population. Some practitioners opt to admit supracondylar fractures so these neurovascular checks can be done.
2. Capitellum and radial head fractures often occur together.
3. The presence of a joint effusion or a posterior fat pad with a history of trauma is presumptive evidence of a fracture, most often associated with a radial head fracture in adults and supracondylar fractures in children.

ELBOW DISLOCATION

Clinical Summary

Elbow dislocations are the second most common major joint dislocation and usually occur posteriorly, although they can be anterior, medial, or lateral. All require immediate reduction to relieve pain and prevent neurovascular compromise. Brachial artery function and ulnar, median, and radial nerve integrity must be evaluated. Elbow dislocations are often associated with a radial head fracture. Patients with posterior dislocations present with their elbow held in flexion and a swollen, tender, and deformed elbow with a prominent olecranon. Anterior dislocations, although rare, present with the elbow extended with the forearm supinated and elongated. Radiographs should include an AP and a lateral view. The presence of fractures should be noted, as this may complicate reduction.

Management and Disposition



FIGURE 11.14 ■ Posterior Elbow Dislocation. This patient dislocated his elbow while playing basketball. Note the flexed position and the prominence of the olecranon. (Photo contributor: Frank Birinyi, MD.)

Most patients require conscious sedation prior to reduction. Posterior dislocations are accomplished by applying posterior pressure to the humerus while an assistant applies longitudinal forearm traction. Alternatively, the patient is placed in the prone position so the humerus hangs perpendicular to the stretcher. A 5- to 10-lb weight is applied to the wrist or axial traction is applied to the wrist while the elbow at the humerus is stabilized. After a few minutes, the olecranon slips back into place. During reduction, the nerve may become entrapped, so neurovascular integrity must be checked before and after reduction. If this occurs, orthopedics should be consulted immediately. After successful reduction, the elbow should be immobilized in 90 degrees of flexion in a posterior splint and sling. Associated fractures may make closed reduction

difficult and leave the joint unstable. In these cases, orthopedic consultation is recommended prior to reduction.

Pearls

1. The ulnar nerve is the most common nerve injured.
2. The olecranon should form a straight line with the two epicondyles when the elbow is extended. At 90 degrees of flexion, the olecranon and the two epicondyles should form a triangle. This relationship is disrupted in the dislocated elbow.
3. If there is concern for a possible fracture, consider obtaining a CT scan after reduction is performed.



FIGURE 11.15 ■ Posterior Elbow Dislocation. Lateral radiograph demonstrating posterior elbow dislocation. (Photo contributor: Selim Suner, MD, MS.)

BICEPS TENDON RUPTURE

Clinical Summary

The biceps has two origins, crosses the shoulder and elbow joints, and inserts on the proximal radius. The long head is much more susceptible to injury, and

rupture may occur anywhere along its route. Clinically, patients with proximal rupture present with pain along the anteromedial aspect of the shoulder. On inspection, ecchymosis is often acutely noted. Muscle retraction within the arm may create a “Popeye” deformity. Due to the two proximal attachments, the short head of the biceps can allow for maintenance of forearm supination strength. Rupture may also occur at the tendon insertion into the radial tuberosity at the elbow. This diagnosis is made based on a history of a painful, tearing or popping sensation in the antecubital region. The ability to palpate the tendon in the antecubital fossa may indicate partial tearing.

Management and Disposition

Proximal and distal biceps tendon ruptures can be discharged with a sling, pain control, physical therapy, and referral for operative repair consideration. Distal biceps tendon ruptures often require surgical management due to the significant loss of forearm supination strength.

Pearls

1. Functional deficits from a long head rupture are usually temporary and are influenced by coexistent tears.

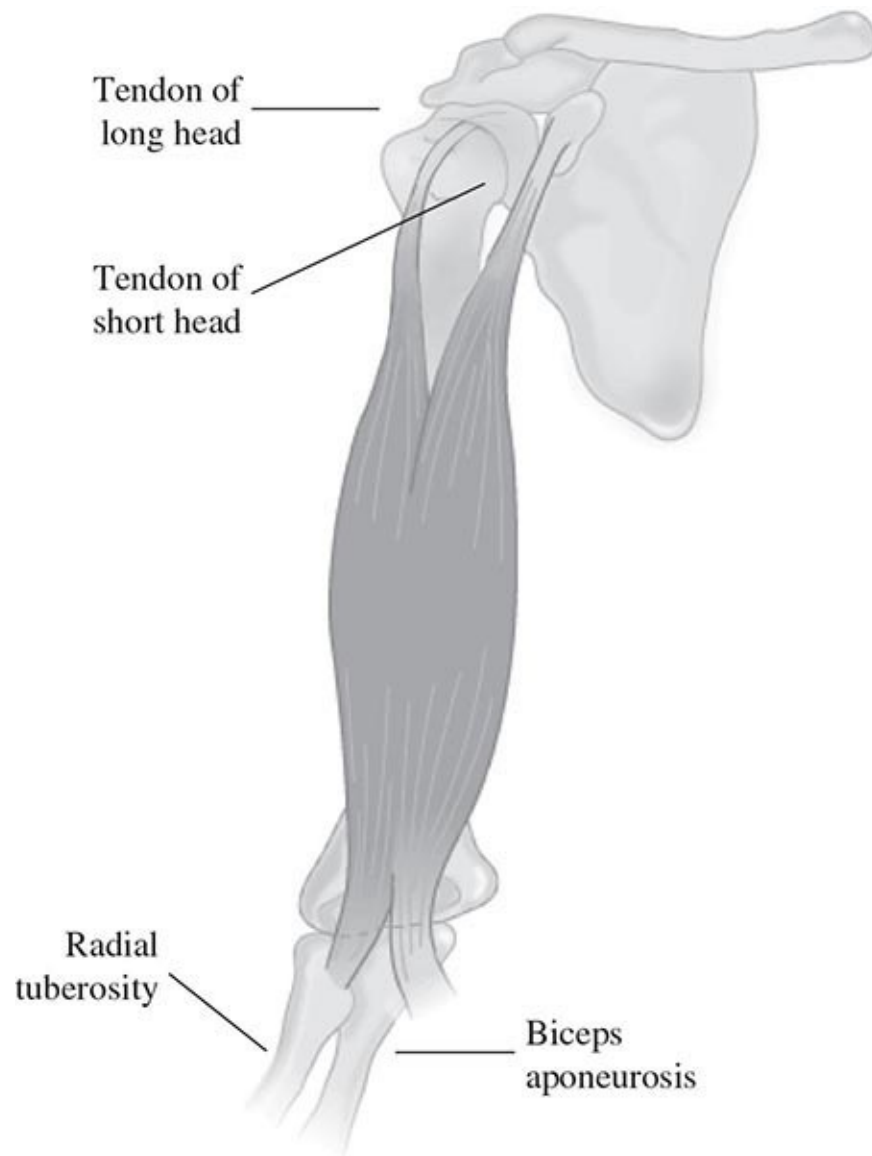


FIGURE 11.16 ■ Biceps Brachii Anatomy. (Reproduced with permission from Chow YC. Elbow and forearm injuries. In: Tintinalli JE, Stapczynski J, Ma O, Yealy DM, Meckler GD, Cline DM, eds. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. 8th ed. New York, NY: McGraw Hill; 2016.)

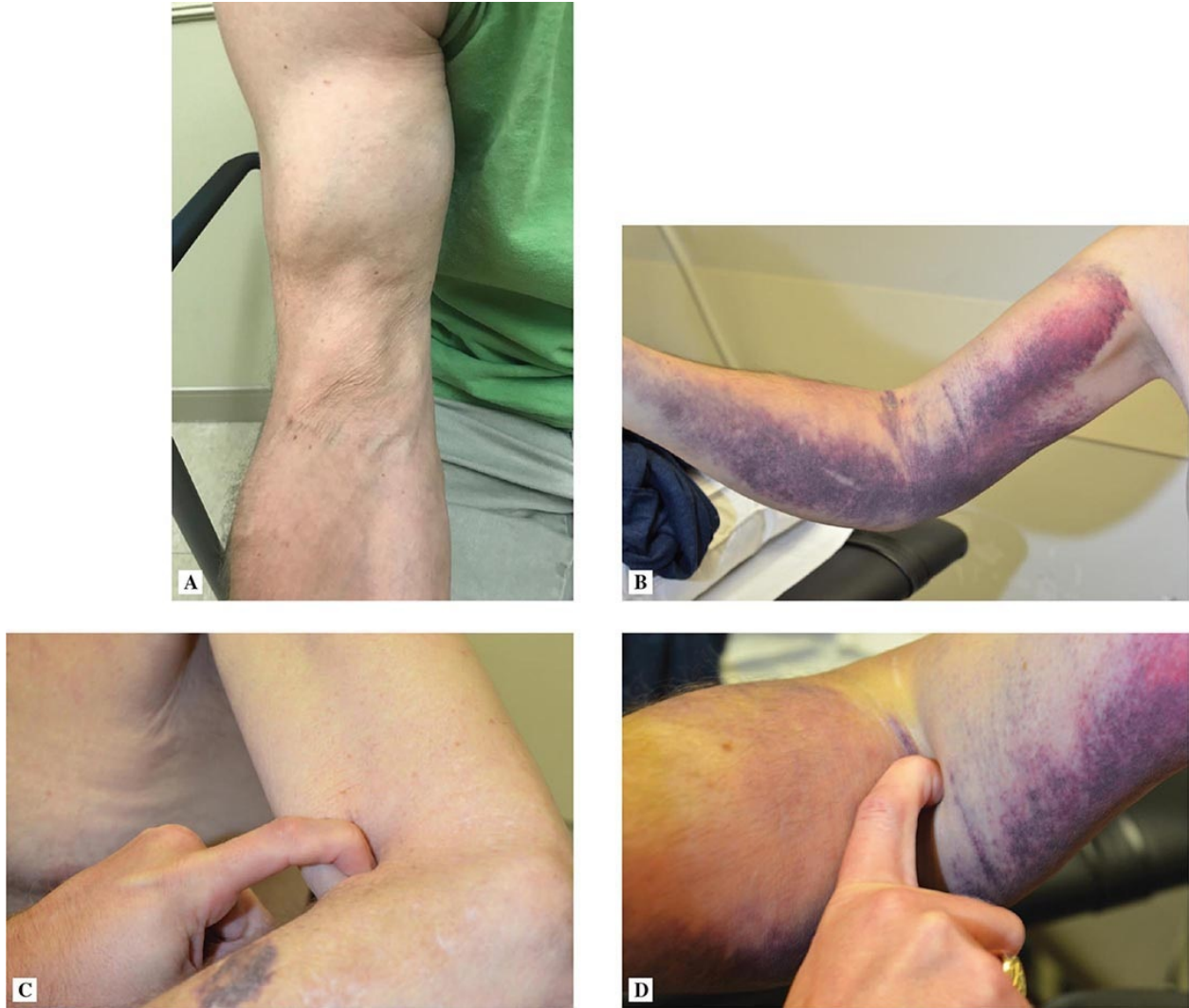


FIGURE 11.17 ■ Distal Biceps Tendon Rupture. The tendon is noted to contract within the arm after rupture (A). Significant ecchymosis is seen in a different patient following distal biceps tendon rupture (B). The same patient in B had a normal hook test in the unaffected extremity (C) and an abnormal hook test in the affected extremity (D). See video. (Photo contributor: Alan B. Storrow, MD.)

2. Biceps rupture occurs most commonly in the dominant extremity of men between 40 and 60 years of age when an unexpected extension force is applied to the flexed arm.
3. The hook test is used to aid in diagnosis of distal biceps tendon rupture. The test is performed by having the patient flex elbow to 90 degrees and supinate forearm. Examiner uses their index finger and attempts to hook the lateral edge of the distal biceps tendon in the antecubital fossa. If tendon is unable to be “hooked,” there should be a high suspicion for tendon rupture (see video).

FOREARM FRACTURES

Clinical Summary

Fractures of the wrist and elbow usually involve a fall onto the outstretched arm, while isolated fractures of the ulnar shaft are more commonly from a direct blow. AP and lateral views of the wrist, forearm, and elbow are required when a forearm fracture is suspected. Functional hand deficits can identify the possibility of an occult injury to forearm neurovascular structures, which could require immediate surgical intervention.

Monteggia fracture-dislocation is a fracture of the ulna (usually proximal third) with an associated proximal radial head dislocation; it is occasionally associated with radial nerve injury. Galeazzi fracture-dislocation is a fracture of the distal one-third of the radius with dislocation of the distal radioulnar joint. It occurs three times more often than a Monteggia fracture and can be associated with ulnar nerve injury. Isolated fractures of the ulna's middle third may result from direct trauma, while the forearm is used to block the blow (nightstick fractures).

Management and Disposition

Both Monteggia and Galeazzi fracture-dislocations require emergent orthopedic consultation and are treated with immobilization in a long-arm splint (with elbow flexed at 90 degrees). The forearm is placed in a neutral position for a Monteggia fracture and supinated for a Galeazzi fracture. Treatment is usually surgical for both injuries, although children may be treated by reduction and casting.

Pearls

1. Any ulnar fracture with greater than 10 degrees of angulation or with a bony fragment displaced more than 50% usually requires surgical correction.
2. Do not be satisfied with the diagnosis of an isolated proximal third ulnar shaft fracture. A line drawn through the radial shaft and head must align with the capitellum in all views to exclude dislocation.
3. An ulnar styloid base fracture can be a clue to a Galeazzi fracture.
4. Since the radius and the ulna create a ring, injury to one of the bones of the

forearm is often associated with fracture or dislocation of the other.

Consequently, upon recognition, one must continue to seek out the other half of the injury by examining both the elbow and wrist joints.

5. Fractures of the forearm may result in compartment syndrome.
6. GRUM is a mnemonic used to remember what fragment is fractured in the Galeazzi and Monteggia fracture patterns: Galeazzi Radius, Ulna Monteggia.



FIGURE 11.18 ■ Nightstick Fracture. Isolated fracture of the middle third of the ulna. (Photo contributor: Lawrence B. Stack, MD.)

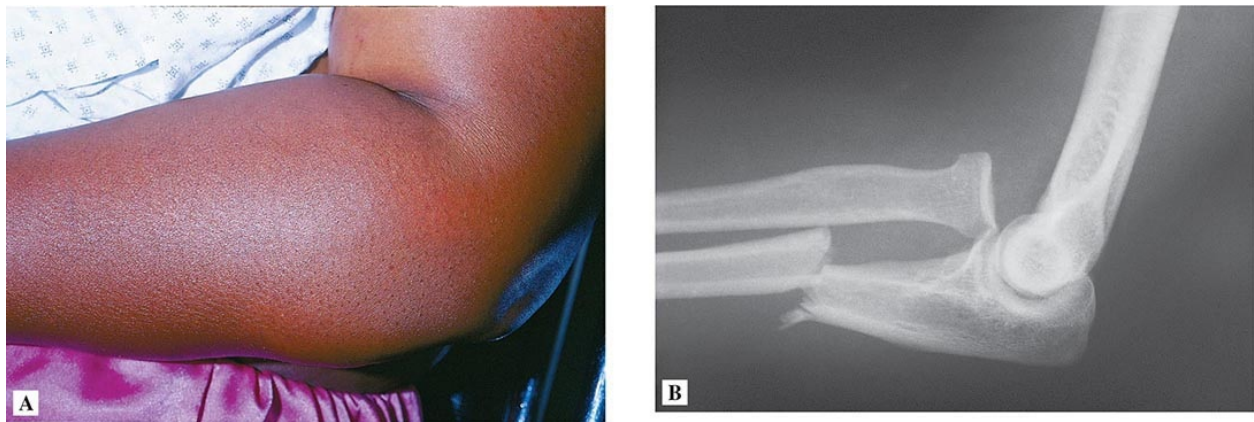


FIGURE 11.19 ■ Monteggia Fracture. (A) Patients present with swelling and pain in the forearm and often a palpable radial head in the ante-cubital fossa. (B) A Monteggia fracture is defined by a fracture of the proximal one-third of the ulna combined with dislocation of the radial head. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.20 ■ Galeazzi Fracture. Note the fracture of the distal one-third of the radius and dislocation of the distal radioulnar joint. (Photo contributor: Lawrence B. Stack, MD.)

FRACTURES OF THE DISTAL RADIUS

Clinical Summary

Falls on an outstretched arm are common, and the radius is often the first to fracture. If there is a supinating component to the fall, the distal ulna may also fracture. In the elderly, distal radius fractures are usually extra-articular metaphyseal fractures, whereas in younger patients, they are usually intraarticular with displacement of the joint surface. There are four types of radial fractures, associated with commonly known eponyms: Colles, Smith, Barton, and Hutchinson (chauffeur).

Colles fractures are the most common. There is dorsal displacement of the distal fragment and apex palmar angulation of the distal fracture fragments; on exam, a “dinner fork” deformity is often described. The *Smith* fracture is of the distal metaphysis with volar displacement and apex dorsal angulation. This usually results from a blow to the dorsum of the wrist or a hyperflexion injury. Exam reveals a reverse dinner fork deformity. *Barton* fractures occur along the dorsal or palmar rim of the distal radius and may be associated with dislocation of the radiocarpal joint. *Hutchinson* fractures are often due to direct impact to the radial styloid with subsequent avulsion.

Several fracture patterns are specific to pediatrics and include the Torus fracture and the Greenstick fracture. *Torus* fractures (also known as buckle fractures) occur due to compression of the bone, which results in a buckling of the periosteum without a complete fracture line. Radiographic findings can be difficult to identify and may only include a subtle asymmetry when compared to the unaffected side. *Greenstick* fractures involve disruption of the cortex on only one side of the periosteum.



FIGURE 11.21 ■ Colles Fracture. The classic dinner-fork deformity is demonstrated. The distal forearm is displaced dorsally. (Photo contributor: Kristin L. Stevens, MD.)

Management and Disposition

Evaluation requires AP, lateral, and oblique views. Orthopedic consultation is required for comminuted, displaced, unstable, or open fractures, as well as those with greater than 20 degrees of angulation or more than 1 cm of shortening. The patient can be immobilized in a sugar-tong splint. Detailed discharge instructions should be given regarding symptoms of median nerve impingement, including paresthesias and hand weakness, which should prompt return evaluation.



FIGURE 11.22 ■ Colles Fracture. Dorsal displacement of the distal fragment. (Photo contributor: Kristin L. Stevens, MD.)

Torus fractures are treated by splinting in position of function with close follow-up; reduction is not required because there is no angulation or displacement. Greenstick fractures require reduction and subsequent casting. Radiographs should be repeated in approximately 1 week to ensure alignment has been maintained.

Pearls

1. All fractures of the distal radius must be evaluated for median nerve function before and after reduction.
2. Colles fractures warrant a high index of suspicion for intraarticular injury,

especially when a radial styloid fracture is noted.

3. With a Hutchinson fracture, associated ligamentous injuries should be sought, especially scapholunate dissociation and perilunate or lunate dislocation.



FIGURE 11.23 ■ Smith Fracture. A Smith fracture is sometimes described as a reverse Colles. (Photo contributor: Frank Birinyi, MD.)



FIGURE 11.24 ■ Smith Fracture. The radiograph reveals volar displacement of the distal radial fragment together with the bones of the wrist and hand. (Photo contributor: Frank Birinyi, MD.)



FIGURE 11.25 ■ Torus Fracture (Buckle Fracture). Compression to the distal portion of the forearm results in buckling of the periosteum without an apparent fracture line. (Photo contributor: Lawrence B. Stack, MD.)

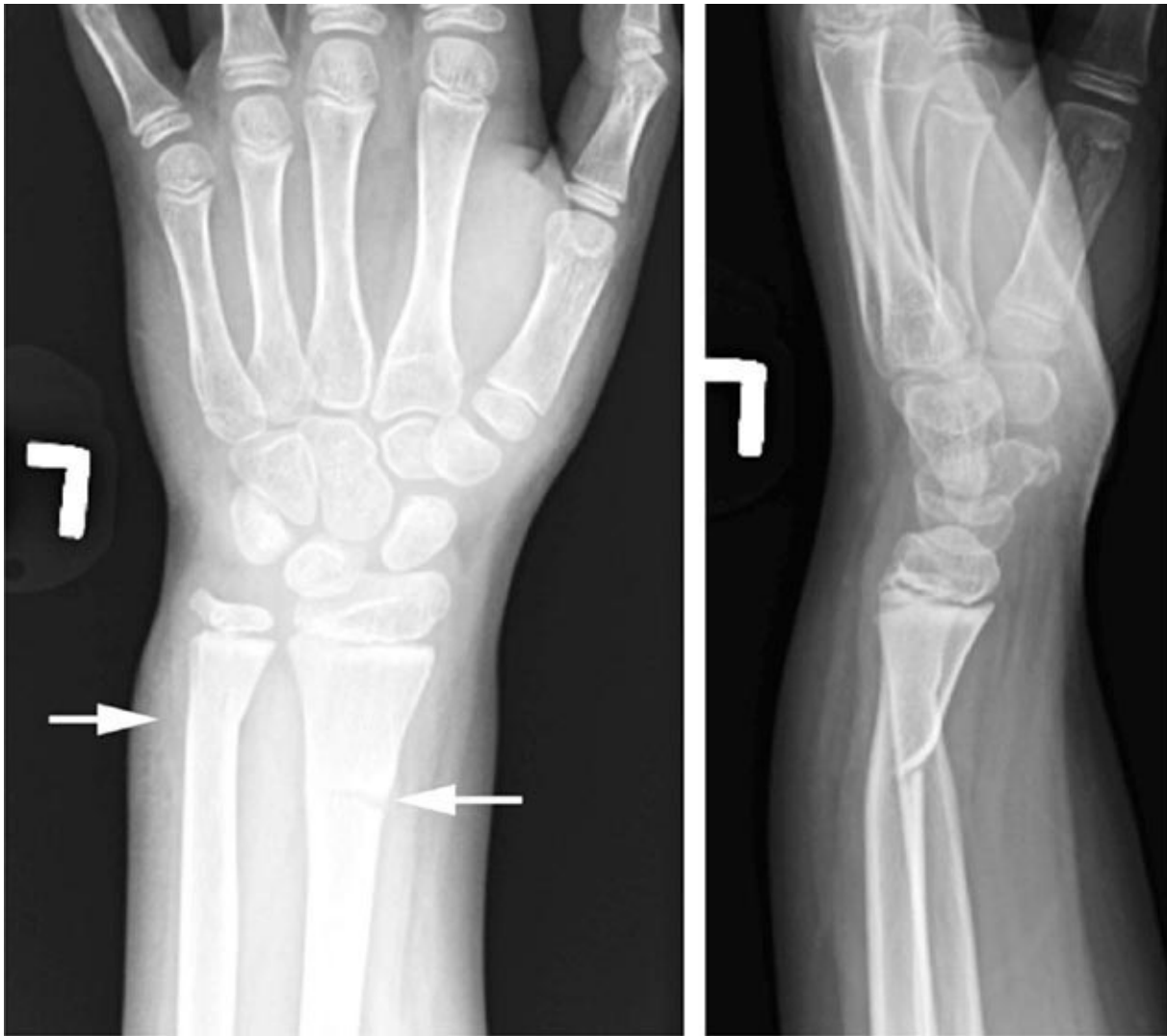


FIGURE 11.26 ■ Greenstick Fracture. Anteroposterior and lateral radiographs demonstrating greenstick fractures of the distal radius and ulna in a child. (Reproduced with permission from Sherman SC. *Simon's Emergency Orthopedics*. 7th ed. © 2015, McGraw Hill, New York. Fig. 13-15.)

CARPAL AND CARPOMETACARPAL DISLOCATIONS

Clinical Summary

Carpal and carpometacarpal dislocations are serious wrist injuries usually occurring from hyperextension. Patients complain of decreased range of motion, pain, swelling, and ecchymosis. Lunate dislocation can occur in a palmer or

dorsal position with the lunate displaced relative to the other carpals (“spilled teacup sign”). The normal lunoradial relationship is disrupted, and the median nerve is commonly involved. If the lunoradial articulation is intact and the other carpal bones are dislocated relative to the lunate, it is termed a perilunate dislocation.

Another potentially serious injury is scapholunate dissociation, often mistakenly diagnosed as a sprained wrist. Although exam may be unremarkable except for pain, an AP radiograph reveals a widening of the scapholunate joint space (Terry Thomas sign). A space of ≥ 3 to 4 mm should prompt suspicion of scapholunate ligament disruption. The lateral radiograph may reveal an increase of the scapholunate angle to greater than 60 to 65 degrees (normal 45-50 degrees). All these injuries may present with concomitant fractures of the carpal bones or distal forearm.

Carpometacarpal dislocations of the index and long meta-carpals are fortunately rare since functional loss is often marked. Thumb, ring, and small finger carpometacarpal dislocations are more common and are frequently missed injuries.

Management and Disposition

Initial management includes adequate radiographic evaluation followed by ice, elevation, and splinting. Referral to a hand specialist is essential for adequate reduction and long-term care.

Pearls

1. A true lateral wrist radiograph best demonstrates a lunate dislocation by exhibiting the usual cup-shaped lunate bone as lying on its side and displaced either dorsally or palmarly.
2. On lateral wrist radiographs, the metacarpal, capitate, lunate, and radius should all align within a line drawn through their long axes. If this is not found, some element of dislocation, subluxation, or ligamentous instability likely exists.
3. Carpometacarpal dislocations are frequently difficult to reduce and require open reduction and fixation in approximately 50% of cases.
4. Trauma to the lunate can lead to Kienbock’s disease (avascular necrosis of the lunate), which causes chronic stiffness and pain. Other medical conditions such as lupus and sickle cell disease can predispose patients to developing this

condition.



FIGURE 11.27 ■ Lunate Dislocation. This photograph demonstrates swelling associated with a volar lunate dislocation. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.28 ■ Perilunate Dislocation. The force from a fall on an outstretched hand disrupted the lunate-capitate articulation; the capitate and other carpal bones were driven posteriorly with respect to the lunate. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.29 ■ Lunate Dislocation. (A) PA wrist view shows disruption of the carpal arcs. (B) Lateral reveals volar displacement of the lunate. The radius, capitate, and third metacarpal align. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. *The Atlas of Emergency Radiology*. New York, NY: McGraw Hill; 2013.)



FIGURE 11.30 ■ Carpometacarpal Dislocation. Note the prominent deformity of the proximal metacarpals, II to IV, on the dorsal hand and the normal prominence of the ulnar styloid. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.31 ■ Perilunate Dislocation. (A) Posteroanterior wrist view shows disruption of the carpal arcs. (B) Lateral reveals the lunate aligns with the distal radius, while the capitate and metacarpals are dislocated dorsal to the lunate. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. *The Atlas of Emergency Radiology*. New York, NY: McGraw Hill; 2013.)



FIGURE 11.32 ■ Scapholunate Dissociation. Note the widened scapholunate joint space. This injury is often misdiagnosed as simple wrist sprain. (Photo contributor: Alan B. Storrow, MD.)

SCAPHOID FRACTURE

Clinical Summary

The scaphoid is the most common carpal bone fractured. Injuries result from either a fall on an outstretched dorsiflexed hand or an axial load along the thumb's metacarpal. Misdiagnosis of a scaphoid fracture can result in delayed healing or avascular necrosis due to lack of a direct blood supply to the bone's proximal portion. Tenderness on anatomic snuffbox palpation is common. Exam of the wrist in ulnar deviation exposes more of the scaphoid to palpation within the snuffbox. Eliciting pain in this area when the patient resists supination or pronation of the hand or pain with axial pressure directed along the thumb's metacarpal is also suggestive of injury. Negative radiographs do not rule out an occult scaphoid fracture.

Management and Disposition

Initial management includes adequate radiographic evaluation followed by ice, elevation, and a thumb spica splint. Referral to a hand specialist is essential.

Pearls

1. Patients in whom there is a clinical suspicion of an occult scaphoid fracture should receive a thumb spica splint and a repeat examination in 7 to 10 days.
2. Avascular necrosis can lead to disabling arthritis.
3. Magnetic resonance imaging (MRI) is the gold standard for diagnosis.



FIGURE 11.33 ■ Scaphoid Fracture. Fracture of the waist, or middle third, of the scaphoid. These injuries can be associated with delayed healing and avascular necrosis. (Photo contributor: Alan B. Storrow, MD.)

CLENCHED FIST INJURY (FIGHT BITE)

Clinical Summary

The clenched fist injury classically occurs when the metacarpophalangeal (MCP) joint contacts human teeth, resulting in a laceration. Many patients will not divulge the true circumstances surrounding the injury; all wounds at the MCP joint, especially of the dominant hand, must be considered a fight bite until proven otherwise. Serious complications can result, including infection, loss of function, and amputation. Most wounds are polymicrobial. Patients who present early may have little evidence of intra-articular injury on examination, whereas those who present more than 18 hours after injury are more likely to have evidence of infection, including pain, swelling, erythema, and purulent drainage.

Management and Disposition

All wounds should be irrigated, debrided, explored, and immobilized. Patients should receive antibiotics directed at both oral and skin flora. Augmentin is the treatment of choice unless a patient has a penicillin allergy. Tetanus prophylaxis is given if needed. Radiographs should be obtained to evaluate for fractures and foreign bodies. These wounds should never be closed and should be allowed to heal by secondary intention. Reliable patients who present early, without evidence of infection or significant comorbidity, and no involvement of bone, joint, or tendon, may be treated on an outpatient basis with 24-hour follow-up. Any patient who does not meet these requirements must be hospitalized for intravenous antibiotics and wound care.

Pearls

1. Complications include cellulitis, lymphangitis, septic arthritis, abscess formation, osteomyelitis, and flexor tenosynovitis.
2. All wounds need to be examined in full flexion and extension to evaluate for tendon injuries.



FIGURE 11.34 ■ Clenched Fist Injury. The lacerations in this photograph were sustained from teeth during a fight. Note the subtle black ink stamp across the proximal metacarpals possibly revealing a clue about the wound's etiology. (Photo contributor: Lawrence B. Stack, MD.)

BOXER'S FRACTURE

Clinical Summary

A metacarpal neck fracture of the 5th digit may occur after a direct blow to the MCP joints of the clenched fist. The apex of the fractured metacarpal bone is dorsal. On physical examination, the “knuckle” is flattened and can be palpated on the volar surface. Exam should address neurovascular integrity and rotational deformity. Rotational deformity is evaluated by having the patient flex the digits at the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints; the four digits should point toward the scaphoid. If the involved digit overlaps another digit or does not point toward the scaphoid, a rotational deformity is

present and needs to be corrected.

Management and Disposition

Up to 40 degrees of angulation on radiography is acceptable for the 4th and 5th metacarpals, whereas angulation of 10 to 20 degrees is acceptable for the 2nd and 3rd metacarpals. Closed reduction for angulation exceeding these limits should be attempted in the ED under appropriate nerve block. Treatment includes ice, elevation, and immobilization in a short-arm gutter splint in the intrinsic plus position (MCP joints are positioned in flexion and the PIP and DIP joints are positioned in extension). For reduction, the MCP joint is held in 90 degrees of flexion and pressure exerted on the metacarpal head, directed dorsally. Simultaneously, the apex of the fracture is directed palmarly. Postreduction radiographs are needed to ensure adequate reduction. Early follow-up (within 7 days) with a hand specialist is essential as the reduction can be lost with simple splinting. Higher degrees of angulation and fractures with any rotational deformity require follow-up for possible open reduction and fixation.

Pearls

1. Subtle malrotation can be recognized by looking at the alignment of the nail beds with the digits flexed.
2. Complications include collateral ligamentous damage, extensor injury damage, and malposition or clawing of the fingers secondary to incomplete reduction.



FIGURE 11.35 ■ Boxer's Fracture. Radiograph reveals a nondisplaced fracture through the neck of the metacarpal in the posteroanterior (PA) view. (Photo contributor: Meghan Breed, MD.)



FIGURE 11.36 ■ Boxer's Fracture. This fracture occurred when the patient punched a wall. There is loss of the “knuckle” when the dorsum of the hand is examined, especially noticeable when the patient makes a fist. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.37 ■ Boxer's Fracture: Rotational Deformity. Malpositioning of the right fifth digit. Normally, all digits point toward a single spot on the scaphoid. (Photo contributor: Alexander T. Trott, MD.)

PERIPHERAL NERVE INJURY

Clinical Summary

Chronic ulnar nerve injury results in the classic claw-hand (intrinsic minus) deformity due to atrophy and contracture of the lumbrical and interosseus hand muscles. The deformity is formed by MCP joint hyperextension and flexion at the PIP and DIP joints of the 4th and 5th digits. There is wasting of the interosseous and hypothenar muscles, as well as the hypothenar eminence. The acutely injured patient is unable to abduct or adduct the digits. Chronic median nerve damage also results in the claw-hand deformity, but to the 2nd and 3rd digits with associated atrophy of the thenar. Acute and chronic damage to the proximal portion of the median nerve results in weakness of wrist flexion, forearm pronation, thumb apposition, and flexion of the 1st three digits.

Wrist drop is the most common symptom seen with radial nerve damage, occurring in situations of acute compression. It is frequently referred to as “Saturday night palsy” (as when a person falls asleep on an arm, or with the arm over a chair, resulting in temporary radial nerve neuropraxia).

Management and Disposition

Treatment is aimed at recognizing the underlying cause, including laceration of the nerve, compression from swelling, or hematoma formation. Patients should be placed in a splint and discharged with referral.

Pearls

1. Long-term nerve injury results in muscle wasting. Prior to any nerve damage, the thenar and hypothenar eminences have a full appearance. Initially, there is flattening of each eminence, followed by a concave or hollow appearance.
2. Sensory and two-point discrimination sense is lost in the distribution of any acutely or chronically injured nerve.



FIGURE 11.38 ■ Claw Hand. Claw-hand appearance resulting from median and ulnar nerve injury. Note metacarpophalangeal joint hyperextension. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 11.39 ■ Claw Hand. Atrophy of the thenar and hypothenar eminences also occurs as a result of damage to the median and ulnar nerves, respectively. Note the concavity to the hypothenar eminence. (Photo contributor: Cathleen M. Vossler, MD.)

BENNETT AND ROLANDO FRACTURES

Clinical Summary

A *Bennett* fracture is an intra-articular fracture at the ulnar aspect of the base of the 1st metacarpal, with radial displacement of the thumb metacarpal and subluxation or dislocation of the carpometacarpal joint. Patients complain of pain, swelling, and decreased range of motion. A *Rolando* fracture is an intra-articular comminuted fracture at the base of the 1st metacarpal, with radial and ulnar fragments resulting in a Y- or T-shaped intra-articular fragment.

Management and Disposition

Treatment consists of ice, elevation, immobilization in a thumb spica splint, and early referral to a hand specialist. These fractures generally require operative reduction and fixation.

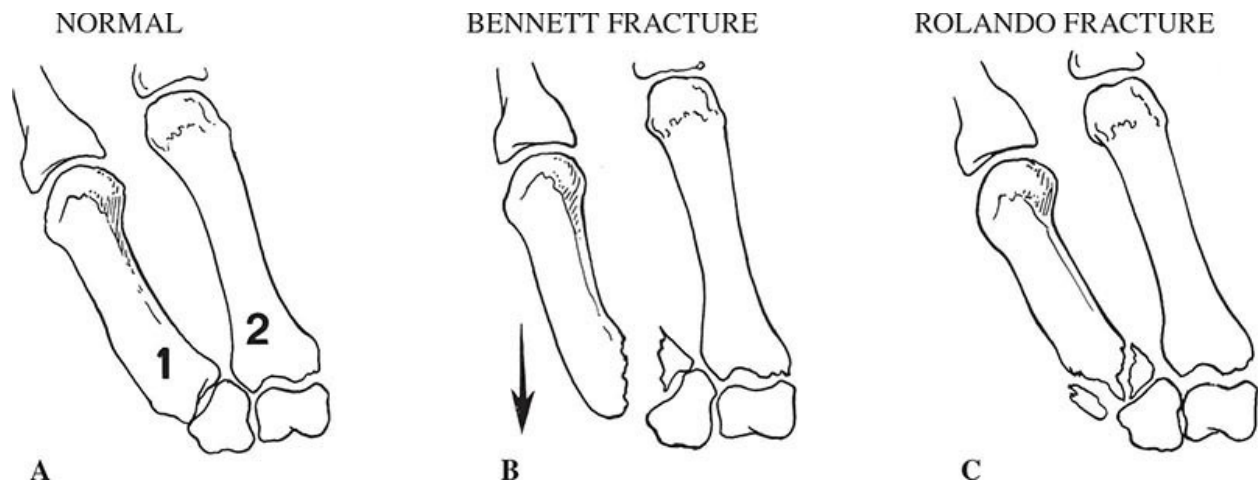


FIGURE 11.40 ■ Intra-articular Fractures of the First Metacarpal Base. Normal anatomy (A). A radial, intra-articular fracture at the base of the first metacarpal is a Bennett fracture (B). A comminuted intra-articular fracture at the base of the first metacarpal is a Rolando fracture (C).



FIGURE 11.41 ■ Bennett Fracture. Bennett fracture involves the base of the first metacarpal. The digit is

swollen and ecchymotic over the affected area. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 11.42 ■ Bennett Fracture. Radiographic examination of a Bennett fracture illustrates an intra-articular fracture at the base of the first metacarpal with the metacarpal displaced radially and proximally. (Photo contributor: Cathleen M. Vossler, MD.)

Pearls

1. Osteoarthritis is a common long-term complication, even after optimal management.
2. Swelling can mask significant angulation.
3. Neurovascular and tendon injuries are not commonly associated with Bennett and Rolando fractures.



FIGURE 11.43 ■ Rolando Fracture. Note the comminuted intra-articular fracture at the base of the first metacarpal. (Photo contributor: Cathleen M. Vossler, MD.)

BOUTONNIÈRE AND SWAN-NECK DEFORMITIES

Clinical Summary

The *boutonnière* deformity results from injury to the central slip insertion of the extensor hood on the dorsal surface of the middle phalanx. After a tear of the central slip, the flexor tendon is unopposed at the PIP and the lateral bands of the extensor tendon contract. With time, these displace volarly, resulting in additional PIP joint flexion and DIP joint extension. The central slip rupture may result from forceful flexion of the PIP joint during full extension, a dorsal PIP joint laceration, or a palmar PIP joint dislocation. The deformity may not be

immediately apparent as it takes time for the lateral bands to slide down to create extension of the DIP joint. Pain and swelling over the dorsal PIP joint, tenderness over the PIP central slip, inability to extend the PIP, and possible DIP joint hyper-extension are common. Radiographically, a small bone fragment may be seen at the proximal portion of the dorsal middle phalanx.

The *swan-neck* deformity occurs because of the contracture of intrinsic hand muscles secondary to systemic diseases such as rheumatoid arthritis and systemic lupus erythematosus. The digit is contorted with hyperextension of the PIP and flexion of the DIP and MCP joints.

Management and Disposition

For a closed boutonnière deformity, immobilization of the PIP joint in extension for 4 weeks is adequate, followed by active range of motion. Open injuries must be carefully explored and repaired. If the deformity is associated with a bony fragment, surgical repair may be necessary. Swan-neck deformities should be splinted and referred as an outpatient. Both require outpatient referral to a hand specialist.

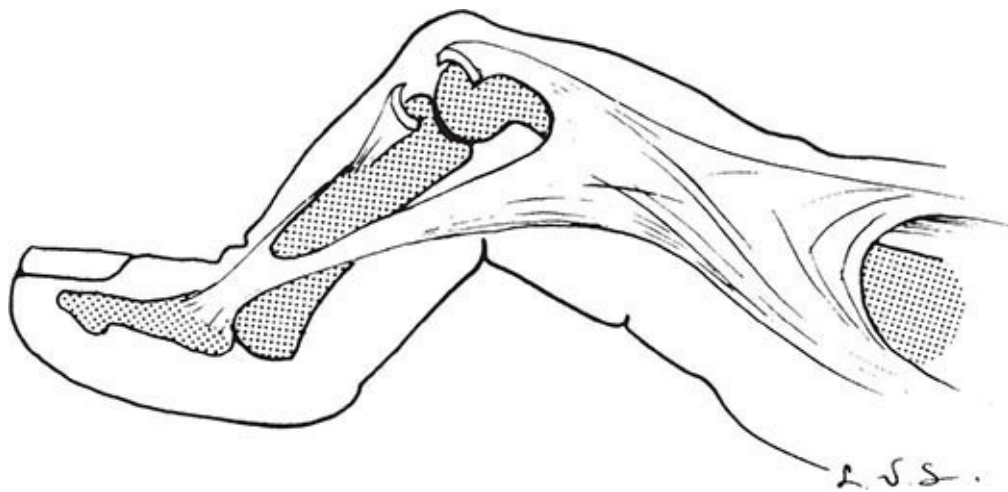


FIGURE 11.44 ■ Boutonnière Deformity. Avulsion or laceration of the central extensor mechanism results in a flexion deformity at the PIP joint and hyperextension of the DIP joint—the boutonnière, or buttonhole, deformity. (Modified with permission from Way LW. *Current Medical Diagnosis & Treatment*. 10th ed. Norwalk, CT: Appleton & Lange; 1994.)



FIGURE 11.45 ■ Boutonnière Deformity. Fourth digit flexion of the PIP joint and extension of the DIP joint. (Photo contributor: E. Lee Edstrom, MD.)

Pearls

1. Boutonnière deformity generally develops weeks after the initial injury as the lateral bands contract.
2. The Elson test is the most reliable way to diagnose a central slip injury before the deformity is present. Have the patient bend the PIP joint to 90 degrees over the edge of the table and extend the middle phalanx against resistance. If a central slip injury is present, there will be weak PIP extension, and the DIP will become rigid. If the central slip tendon remains intact, the DIP remains floppy because all force is applied to extension of the PIP joint and the lateral bands are not activated.
3. Surgical repair may be required for patients when conservative therapy yields inadequate results.



FIGURE 11.46 ■ Swan-Neck Deformity. Hyperextension of the PIP joint and flexion of the DIP joint. (Photo contributor: Cathleen M. Vossler, MD.)

GAMEKEEPER'S THUMB (SKIER'S THUMB)

Clinical Summary

Forced radial deviation (abduction) of the thumb can cause a rupture of the ulnar collateral ligament (UCL) known as “gamekeeper’s” or “skier’s” thumb. The tear usually occurs at the proximal phalanx insertion and can be associated with volar plate and dorsal capsule injury. Pain and swelling are present over the ulnar aspect of the proximal phalanx and thumb metacarpal.

Radiographs may reveal a small avulsion fracture of the proximal phalanx. Abduction stress testing (stabilizing the metacarpal with one hand while applying radial stress on the proximal phalanx) may provide additional clinical information, especially in patients with normal radiographs. Classically, more

than 30 to 40 degrees radial angulation indicates complete rupture. Stress testing should be done on both sides in extension and 30 degrees of flexion while feeling for a firm endpoint.

Management and Disposition

Apply a thumb spica splint and provide analgesia. Patients with pincer function weakness, point tenderness at the volar-ulnar aspect of the thumb MCP joint, a bony fragment of greater than 15% of the articular surface, avulsed fragment displacement of greater than 5 mm, or significant angulation on stress testing should prompt hand surgery referral. Complete tears need repair within 1 week. A sprain without instability is commonly treated with thumb spica casting or splinting for 4 to 6 weeks followed by range of motion exercises.



FIGURE 11.47 ■ Gamekeeper's Thumb. Laxity of 30 to 40 degrees more than the uninjured thumb, measured in neutral and 30 degrees of flexion, is strongly suggestive of a complete ulnar collateral ligament tear. There is no “endpoint” to the radial deviation of the phalanx. (Used with permission from Brunicaardi FC, Anderson DK, Billar TR, et al. *Schwartz's Principles of Surgery*. 8th ed. New York, NY: McGraw Hill; 2005. © 2005 by McGraw Hill.)

Pearls

1. Radial collateral ligament rupture can also occur with forced adduction but is

uncommon.

2. Skier's thumb refers to an acute injury, whereas gamekeeper's has classically been associated with repetitive trauma.
3. Thumb MCP joint laxity is highly dysfunctional, painful, and may lead to late arthritis.
4. Complete UCL tears put patients at risk for developing a Stener lesion, which occurs when the aponeurosis of the adductor pollicis becomes interposed between the ends of the torn ligament and prevents healing. MRI is the gold standard for diagnosis.

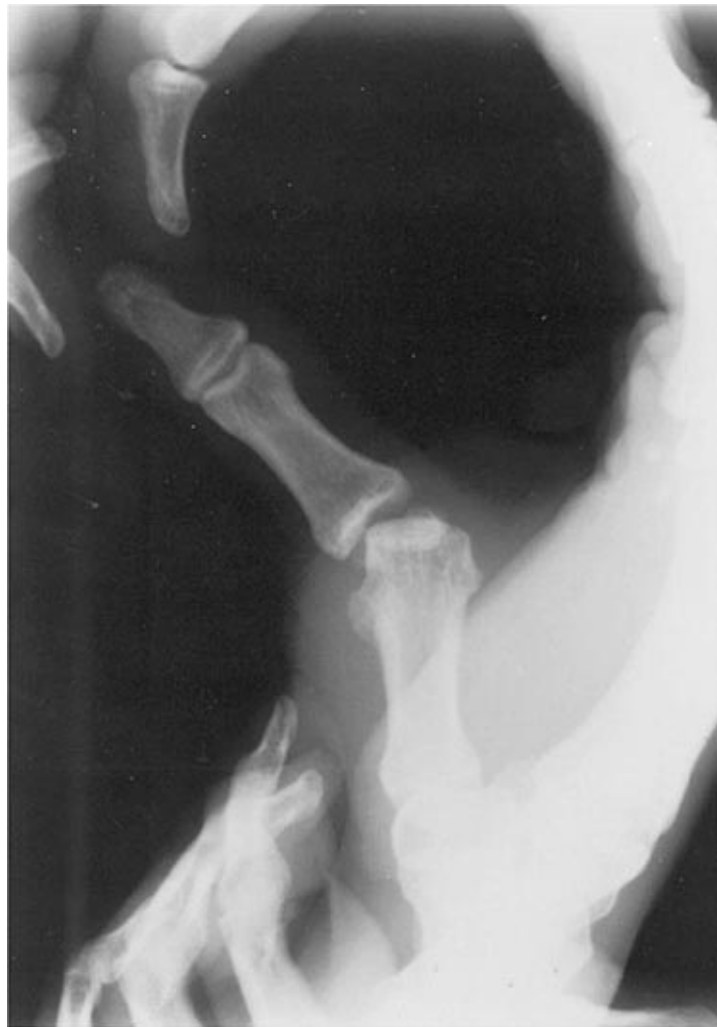


FIGURE 11.48 ■ Gamekeeper's Thumb. Stress x-ray of a thumb with a complete ulnar collateral ligament tear demonstrates marked instability of the ulnar side of the MCP joint and radial deviation of the proximal phalanx. (Used with permission from Brunicaudi FC, Anderson DK, Billar TR, et al. *Schwartz's Principles of Surgery*. 8th ed. New York, NY: McGraw Hill; 2005. © 2005 by McGraw Hill.)

PHALANGEAL DISLOCATIONS

Clinical Summary

Phalangeal dislocations are common, generally dislocate dor-sally, are caused by hyperextension and axial compression, and may have associated volar plate damage. PIP volar dislocations can be irreducible secondary to rupture of the extensor tendon and herniation of the proximal phalanx through the extensor hood, requiring operative repair. MCP joint dorsal dislocations are often due to hyperextension. DIP dislocations are the rarest but can occur when an axial force is applied to the distal phalanx. Gross deformity is noted on examination, with the distal phalanx generally displaced dorsally.



FIGURE 11.49 ■ Phalangeal Dislocation. This patient dislocated the long finger PIP joint during an altercation. The PIP joint is displaced dorsally with an obvious deformity. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.50 ■ Phalangeal Dislocation. Medial angulation of the ring finger suggests PIP dislocation. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 11.51 ■ Volar Plate Injury. Subtle PIP swelling and ecchymosis of the long digit seen with a volar plate injury (A). Radiographs of volar plate injuries often reveal a small fragment on the volar surface of the PIP joint (B). (Photo contributor: Cathleen M. Vossler, MD.)

Management and Disposition

Digital nerve block for reduction and splinting is appropriate anesthesia for the PIP and DIP joints. Ulnar, median, or radial nerve blocks are necessary for the MCP joints. Reduction of dorsal dislocations is accomplished via joint hyperextension with concurrent application of horizontal traction followed by joint flexion. Flexion at the MCP joint will facilitate reduction of interphalangeal joints. Postreduction radiographs are necessary. After closed reduction of a dorsal dislocation, the DIP joint should be splinted in slight flexion and the PIP joint in at least 20 degrees of flexion for 3 to 5 weeks, depending on the degree of ligamentous damage. Hand specialist follow-up is recommended.

Pearls

1. All joints should be tested for instability after reduction, using a digital nerve block to facilitate testing.
2. PIP joint volar dislocation can be unstable, requiring open reduction and internal fixation. Joint dislocations with volar plate entrapment may be impossible to reduce and require surgery.

MALLET FINGER AND JERSEY FINGER

Clinical Summary

A *mallet finger* commonly occurs after the DIP joint is forcibly flexed against an actively extended finger, tearing the extensor mechanism as it inserts on the distal phalanx. This can occur after a sudden axial blow to an extended fingertip. The patient presents with an inability to actively extend the distal phalanx while maintaining a normal passive range of motion, and the DIP joint remains passively flexed. On radiography, there may be a small bony avulsion fragment on the dorsum at the distal phalanx.

A *jersey finger* involves an avulsion of the distal phalanx flexor mechanism. The flexor digitorum profundus (FDP) tears because of forced extension of a fully flexed DIP, as would occur when someone grabs the jersey of an opponent while attempting to tackle them. Most commonly, the ring finger is involved. Clinically, the patient presents with an inability to actively flex the DIP joint

while maintaining a full passive range of motion. Radiographically, a bony avulsion fragment may be present.



FIGURE 11.52 ■ Mallet Finger. This illustration demonstrates that the unopposed flexion of the DIP joint is secondary to the complete tear of the tendon (**A**), or an avulsion of a small chip fragment (**B**).

Management and Disposition

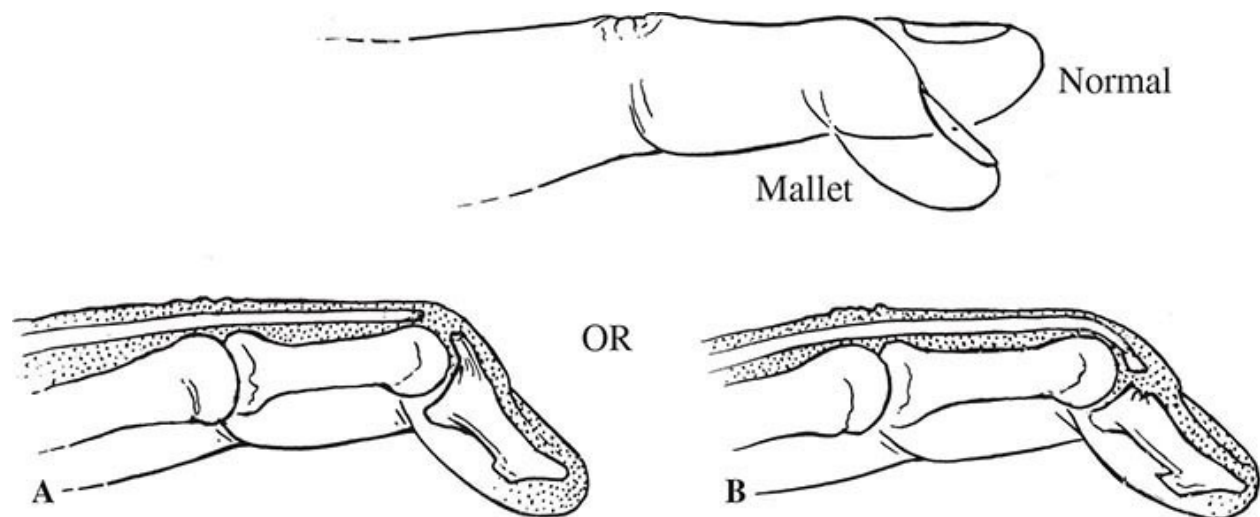


FIGURE 11.53 ■ Mallet Finger. The distal phalanx is held in flexion and the patient is unable to extend it. (Photo contributor: Kevin J. Knoop, MD, MS.)

A closed mallet finger without involvement of the joint can be treated by splinting the DIP joint in extension; the PIP joint should not be splinted. This splint should be worn continuously for at least 6 weeks. Operative treatment is usually not required. A jersey finger often requires surgical repair; early referral

is recommended. Prognosis worsens if treatment is delayed or severe tendon retraction is present.

Pearls

1. Avulsion of a significant portion of the mallet finger articular surface (more than one-third) may require open reduction with internal fixation.
2. The jersey finger involves the FDP tendon at the DIP joint. It may be tested by isolating the affected DIP (ie, holding the MCP and PIP joints in extension while the other fingers are in flexion) and asking the patient to flex the DIP joint.



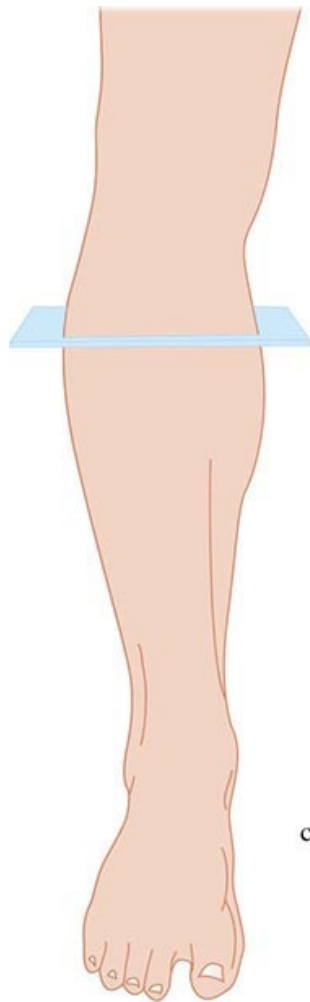
FIGURE 11.54 ■ Mallet Finger. Classic mallet finger—the long finger remains flexed at the DIP joint while the patient is attempting to actively extend his fingers. (Photo contributor: Matthew Kopp, MD.)



FIGURE 11.55 ■ Jersey Finger. The normal cascade of flexion is disrupted in the injured hand, consistent with a Jersey finger. (Used with permission from Brunickardi FC, Andersen DK, Billiar TR, et al. *Schwartz's Principles of Surgery*. 8th ed. New York, NY: McGraw Hill; 2005. © 2005 by McGraw Hill.)

COMPARTMENT SYNDROME

Clinical Summary



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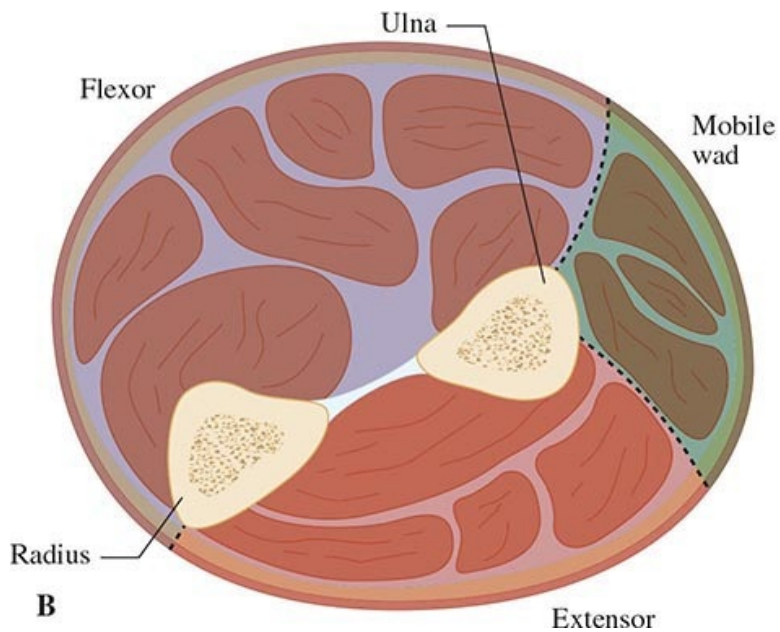
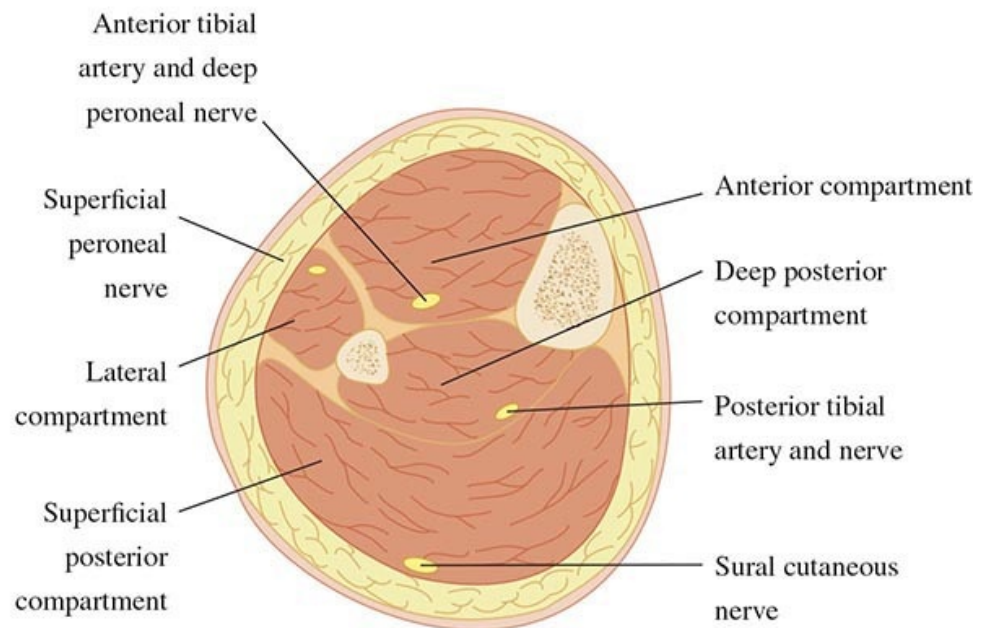


FIGURE 11.56 ■ Compartments. The four compartments of the lower leg (A) and forearm compartments (B). (Reproduced with permission from Tintinalli JE, Stapczynski J, Ma O, Yealy DM, Meckler GD, Cline DM, eds. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. 8th ed. New York, NY: McGraw Hill; 2016.)

Compartment syndrome develops when the pressure in the inelastic fascial space increases to a point where it causes compression and dysfunction of venous outflow. Major vascular and neural compromise lead to the classic five “Ps” of late compartment syndrome: *pallor*, *paresthesias*, *poikilothermia*, *paralysis*, and *pulselessness*. Compartment syndrome may result from exertion, circumferential burns, frostbite, constrictive dressings, arterial bleeding, severe soft tissue injury, and fractures. It can occur anywhere, but most commonly occurs in the anterior compartment of the leg and volar compartment of the forearm.

The earliest symptom is severe pain out of proportion to the physical findings. The involved compartment is extremely firm. The pain is worsened with passive range of motion due to ischemic muscle fiber stretch. Consequently, the patient often holds the injured part in a position relaxing the involved muscle groups. Paresthesia is a late sign of nerve compromise, commonly with vibratory sensation lost first. Motor weakness, pallor, poikilothermia, and pulselessness are very late signs and only occur after irreversible muscle, nerve, and vascular damage. The goal is to identify compartment syndrome before these late signs occur; ischemic injury occurs around 4 hours and becomes irreversible around 8 hours.

The diagnosis is confirmed by measuring compartment pressures; greater than 30 mm Hg is suggestive and should prompt surgical consultation for fasciotomy consideration. A delta pressure (diastolic blood pressure – measured compartment pressure) less than 30 mm Hg should also prompt surgical evaluation. Measurements should be obtained within 5 cm of the site.

A serious complication is Volkmann ischemic contracture, classically following a supracondylar fracture. Postischemic swelling produces increased pressure within the enclosed osteofascial forearm compartment and reduces capillary blood perfusion below the level necessary for tissue viability. If not addressed, muscle and nerve necrosis eventually become replaced by fibrotic tissue and produces a contracture. Refusal to open the hand, pain with passive extension of the fingers, and forearm tenderness are signs of Volkmann ischemia.



FIGURE 11.57 ■ Compartment Syndrome. Late anterior compartment syndrome of the left lower extremity is manifested by anterior tibial pain and tense “woody” swelling. (Photo contributor: Timothy Coakley, MD.)

Management and Disposition

Initial treatment is removal of constrictive dressings and jewelry as well as frequent reevaluation. If there is no improvement, decompression via a fasciotomy should be considered. Aside from muscle and nerve damage, complications include myonecrosis, which can cause myoglobinuria and renal failure.

Pearls

1. The diagnosis should be made early and be based on clinical evaluation and the mechanism of injury. Crush or compression injuries should heighten suspicion.
2. Anterior compartment of the leg involvement is commonly due to proximal tibial fractures, whereas volar compartment of the forearm is secondary to ulna, radius, or supracondylar fractures.

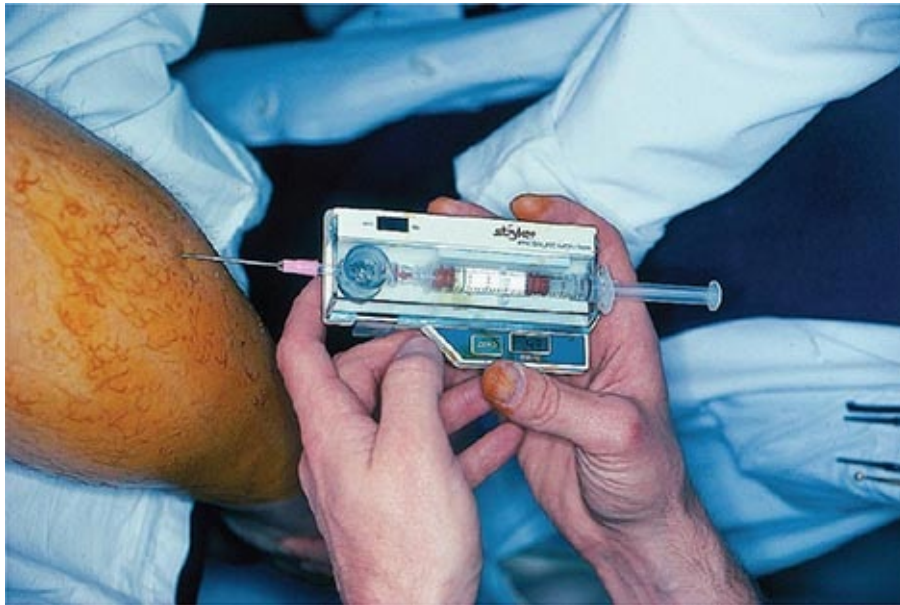


FIGURE 11.58 ■ Compartment Pressures. Intracompartmental pressure monitoring can be accomplished with commercially available devices. Normal tissue pressures should be less than 10 mm Hg; orthopedic consultation is recommended when pressures exceed 30 mm Hg. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 11.59 ■ Compartment Syndrome. Volkmann contracture is a serious late complication of unrelieved compartment syndrome. (Photo contributor: Lawrence B. Stack, MD.)

HIGH-PRESSURE INJECTION INJURY

Clinical Summary

Many commercial devices can deliver liquids and gases at pressures exceeding 5000 psi. Unfortunately, these may accidentally introduce these substances into the body, leading to possible ischemia because of direct chemical irritation, venous outflow obstruction, arterial compression secondary to the volume of material, spasm, or edema. The injected material spreads along fascial planes, so the extent of injury can be quite misleading and is often subtle on initial presentation. Swelling and pain increase over time, but on initial evaluation, the examiner may find no apparent skin break or only a small puncture wound.

Management and Disposition

Immediate operative debridement is the treatment of choice; early consultation with orthopedics is necessary. Radiographic examination to evaluate for fractures and to delineate the spread of the injected material should be considered. Tetanus, analgesia, and broad-spectrum antibiotics should be administered. The affected extremity should be elevated and splinted.

Pearls

1. Do not be misled by the “benign” appearance of the initial injury. Delays in treatment can lead to compartment syndrome.
2. Digital blocks are contraindicated because of the potential for increased tissue pressure and compromise of tissue perfusion.



FIGURE 11.60 ■ High-Pressure Injection Injury. Injury incurred by a grease gun that accidentally discharged into a hand. Note the swelling and erythema. (Photo contributor: Richard Zienowicz, MD.)

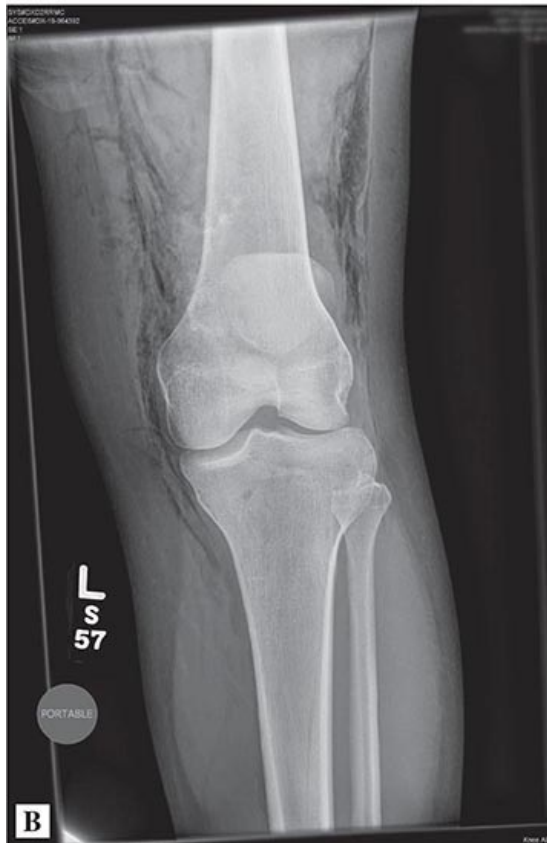


FIGURE 11.61 ■ High-Pressure Injection Injury. Industrial worker with injection injury from sandblaster in the leg (**A**). Note the spread along fascial planes demonstrated radiographically (**B**). (Photo contributors: Timothy S. Forsythe, DO (**A**); Kevin J. Knoop, MD, MS (**B**).)

SUBUNGUAL HEMATOMA

Clinical Summary

A subungual hematoma is a collection of blood underneath the nail, usually occurring secondary to distal digit trauma. Patients often present with throbbing pain secondary to pressure beneath the nail. Associated injuries include nail bed trauma and distal tuft fractures. When fractures are present, these are considered open.

Management and Disposition

A radiograph should be done to evaluate for fracture. Acutely, if the subungual hematoma involves less than 50% of the nail matrix, trephining the nail with a sterile needle or electrocautery is adequate to relieve pain by allowing drainage. A digital block may be required prior to trephination. The involved digit can be soaked in sterile water with peroxide after trephination to encourage drainage.

Management of larger hematomas is controversial. Some authors advocate nail removal for bed injury repair if the hematoma covers more than 50% of the nail or if there is an associated fracture. Some believe nail removal is best reserved for injuries damaging the nail plate and surrounding tissues, regardless of the size of the hematoma or presence of fracture. After removal, the patient's clean nail, a piece of foil, or petroleum gauze should be placed between the eponychial nail fold and the germinal matrix.

The patient with a fracture should be discharged with a splint supporting the distal joint in extension, and all dressings should be kept dry. Antibiotics are not required unless the area is contaminated.



FIGURE 11.62 ■ Subungual Hematoma. Two subungual hematomas secondary to crush injury. (Photo contributor: R. Jason Thurman, MD.)

Pearls

1. Subungual hematomas are a sign of nail bed injury.
2. Subungual hematomas with surrounding nail bed and nail fold injuries require nail removal and evaluation of the bed for injury and careful repair if needed.
3. A hand-held, high-temperature, portable cautery device is a good tool for trephination (see video).



FIGURE 11.63 ■ Trephination. The hot paper clip formed a nice hole in the nail plate, and the blood drained out spontaneously. This relieved the pressure and gave the patient immediate pain relief. (Photo contributor: Richard P. Usatine, MD. Reproduced from Usatine RP, Smith MA, Mayeaux EJ Jr, Chumley H. *The Color Atlas and Synopsis of Family Medicine*. 3rd ed. New York, NY: McGraw Hill; 2019.)



FIGURE 11.64 ■ Nail Bed Laceration. Bleeding from a nail bed laceration causes a subungual hematoma. This image depicts a nail bed laceration seen after nail removal. (Photo contributor: Alan B. Storrow, MD.)

HIP DISLOCATION

Clinical Summary

Hip dislocations are often associated with fractures of the acetabulum, ipsilateral femur, or knee, and can be anterior, posterior, or inferior. Ninety percent are posterior, resulting from significant forces exerted on a flexed hip and knee (eg, a passenger in a motor vehicle collision whose knees hit the dashboard).

Anterior hip dislocations occur when there is forced external rotation of the extended hip, which forces the head out of the acetabulum, either by tearing the anterior capsule or by fracturing the anterior wall of the acetabulum.

Patients complain of severe hip pain and decreased range of motion. Posterior dislocations present with the extremity shortened, internally rotated, adducted, and flexed. With anterior dislocations, the leg is often abducted, externally rotated, and slightly flexed; however, this presentation can vary.

Management and Disposition

Treatment for dislocations is early closed reduction, often with conscious sedation, although ultrasound-guided femoral nerve blocks are an option. A neurovascular and radiographic evaluation should occur before and after reduction attempts. Since the muscles around the hip are so strong, a general anesthetic with complete paralysis may be required. Posterior dislocations are reduced using in-line traction with the hip and knee flexed to 90 degrees, followed by gentle internal to external rotation; several different techniques can be used for reduction: the Allis maneuver, the Bigelow maneuver, and the Captain Morgan technique. Anterior dislocations are reduced using strong inline traction with the hip in neutral flexion extension or slight extension, slight adduction and internal rotation, followed by abduction.

Orthopedic consultation should be obtained as early as possible. These patients require admission, with frequent neurovascular evaluation. Hip replacements tend to dislocate more easily than native hips and usually require less energy for reduction. Radiographs should be done before and after reduction to evaluate for periprosthetic fractures.

Pearls

1. Complications of posterior hip dislocations include sciatic nerve injury and avascular necrosis. Neurovascular complications of anterior hip dislocations are uncommon.
2. In the young patient, immediate reduction of a native hip is imperative and should be accomplished within 6 hours.

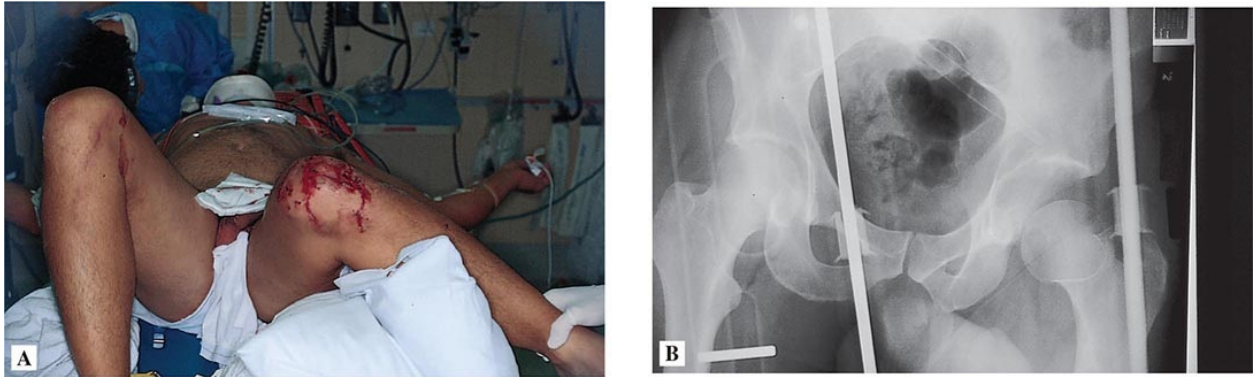


FIGURE 11.65 ■ Hip Dislocation. Typical patient appearance of a left posterior hip dislocation. Note internal rotation of the affected extremity (**A**). Radiograph (**B**). (Photo contributor: Cathleen M. Vossler, MD.)

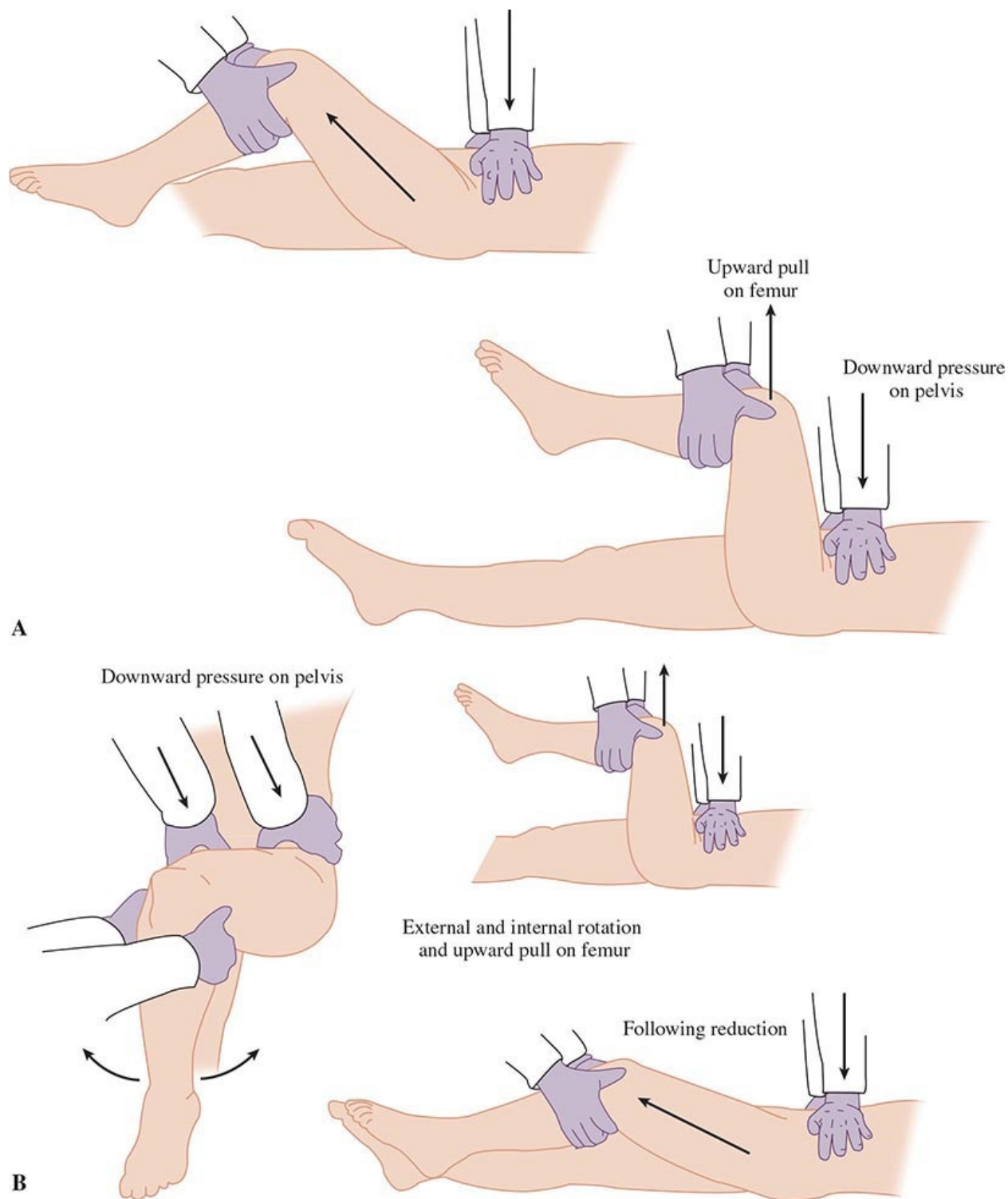


FIGURE 11.66 ■ Allis Maneuver for Reduction of Posterior Hip Dislocation. (Reproduced with permission from Tintinalli JE, Ma O, Yealy DM, Meckler GD, Stapczynski J, Cline DM, Thomas SH. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. 9th ed. New York, NY: McGraw Hill; 2020.)

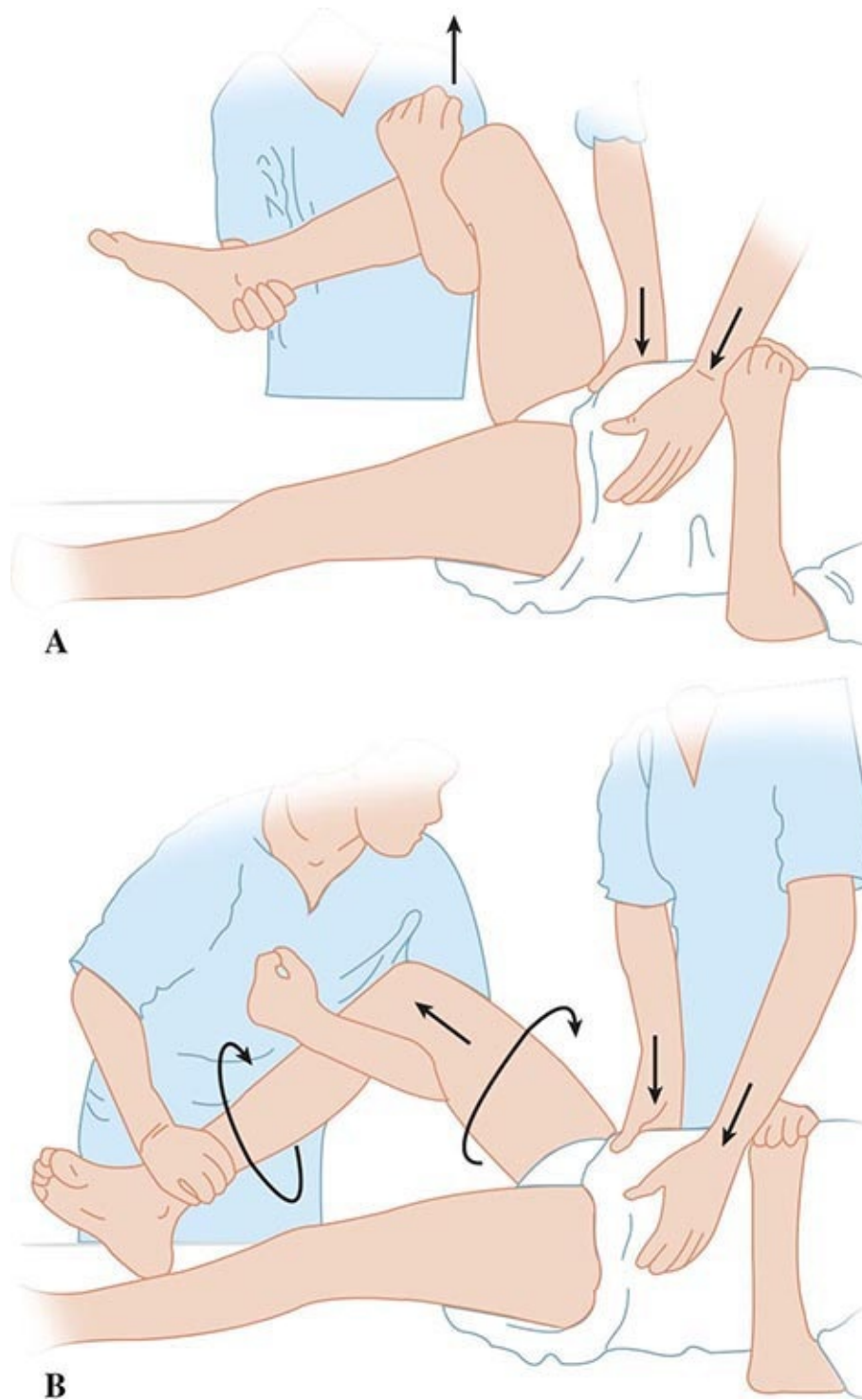


FIGURE 11.67 ■ The Bigelow Maneuver for Reduction of Posterior Hip Dislocation. (A) The physician applies upward traction on the femur while an assistant stabilizes the pelvis. (B) The hip is externally rotated and extended while the femur is distracted. (Reproduced with permission from Reichman EF. *Emergency Medicine Procedures*. 3rd ed. New York, NY: McGraw Hill; 2020.)



FIGURE 11.68 ■ The Captain Morgan Technique for Reduction of Posterior Hip Dislocation. (1) Stabilize the patient's pelvis by placing the patient on a backboard in the supine position and strapping the pelvis to the board, or have an assistant stabilize the pelvis on the stretcher by placing both hands on the patient's iliac crests and using pressure to keep the pelvis stable. (2) To reduce the dislocation, place your foot on the stretcher or board with your knee posterior to the patient's knee. (3) With the patient's knee in flexion, gently pull downward at the patient's ankle while applying an upward force to the patient's hip by lifting your heel by stepping on your toes and contracting your calf. Gently rotate the patient's hip while applying the upward traction behind the patient's knee. (Reproduced with permission from Tintinalli JE, Ma O, Yealy DM, Meckler GD, Stapczynski J, Cline DM, Thomas SH. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. 9th ed. New York, NY: McGraw Hill; 2020.)

HIP FRACTURE

Clinical Summary

Fractures above the level of the lesser trochanter are termed hip fractures. The femoral head has a tenuous blood supply, and fractures can compromise blood flow, resulting in avascular necrosis. For classification, hip fractures are generally divided into *intracapsular* (femoral head and neck fractures) and *extra-capsular* (trochanteric, intertrochanteric, and subtrochanteric). Accurate classification is important because intracapsular fractures are more likely to have vascular disruption.

Complaints include groin or buttock pain, tenderness, and an inability to bear weight on the affected side. There can be shortening of the affected leg, as well

as abduction and external rotation.

Both the Shenton line (the curved line formed by the top of the obturator foramen and the inner side of the neck of the femur) and the neck shaft angle (normal is 120-130 degrees) should be evaluated, but can be normal in nondisplaced fractures.

Management and Disposition

Fractures of the hip require early orthopedic consultation for admission; in most cases, fractures require surgical reduction and fixation. Femoral head fracture-dislocations are an orthopedic emergency and require immediate reduction. A neurovascular exam should be performed before and after any reduction attempts.

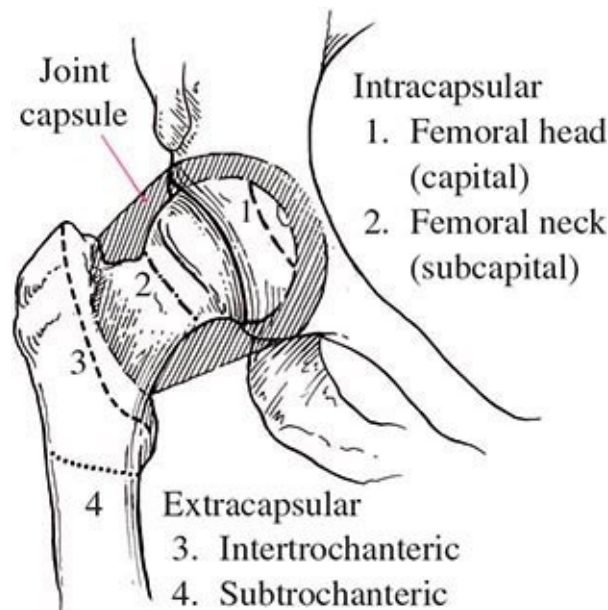


FIGURE 11.69 ■ Hip Fractures. This illustration depicts the different types of proximal femoral fractures.

Pearls

1. Fracture-dislocation of the femoral head requires a significant amount of force; intra-abdominal and retroperitoneal injuries should be considered.
2. Hip fractures may be diagnosed by auscultation of differences in bone conduction between the extremities. This is performed by placing the stethoscope's diaphragm on the anterosuperior iliac spine and giving the patella soft taps.

3. Any elderly patient with an inability to bear weight has a hip fracture until proven otherwise. Hip fractures can be secondary to osteoporosis, so there does not need to be a history of trauma. Since plain films may not be sensitive enough to identify some hip fractures, other imaging such as CT or MRI should be considered.



FIGURE 11.70 ■ Hip Fracture. Patients with hip fractures often present with the affected extremity shortened, externally rotated, and abducted. Note the rotation and shortening in this patient with a right intertrochanteric fracture. (Photo contributor: Cathleen M. Vossler, MD.)

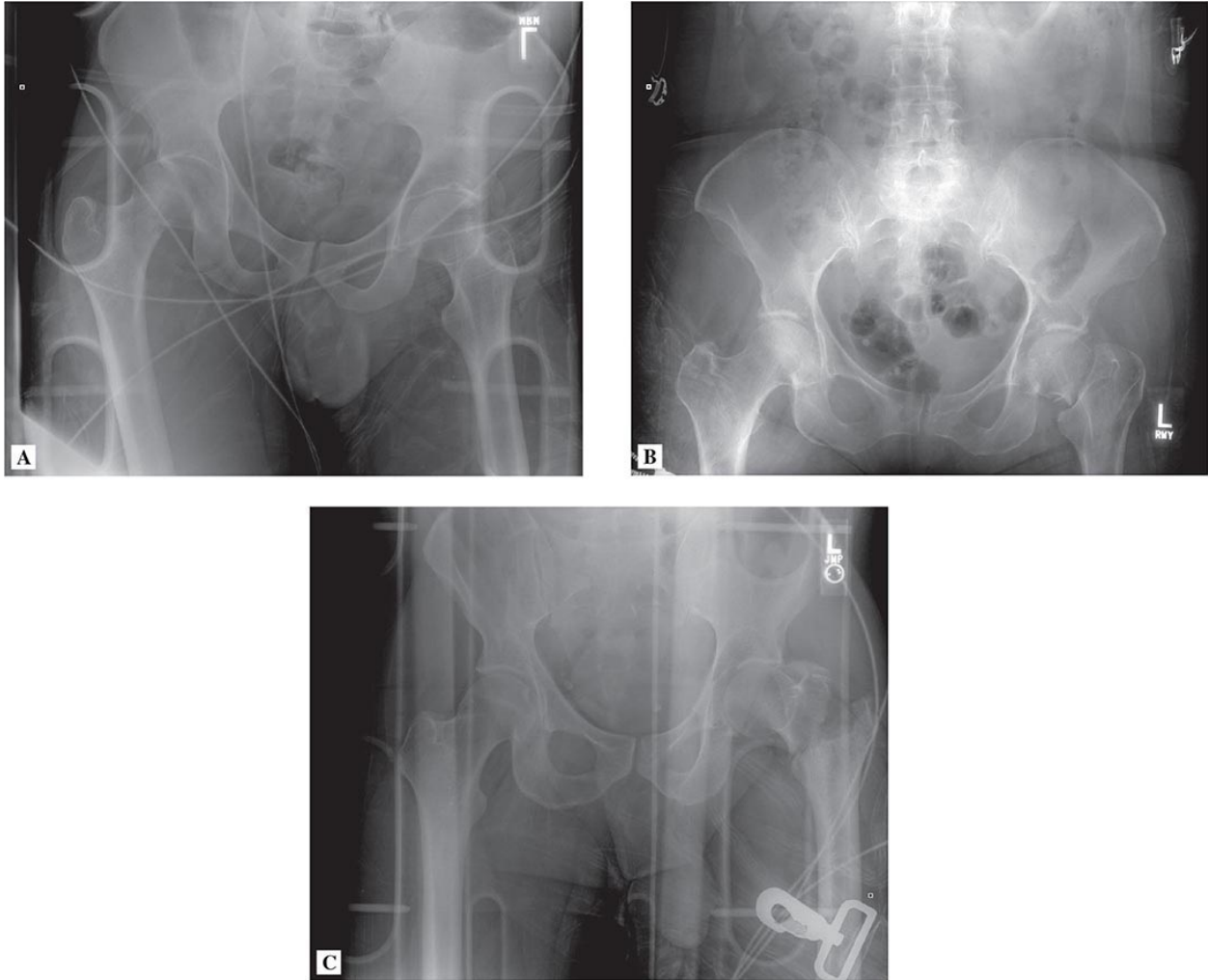


FIGURE 11.71 ■ Hip Fractures. Radiographs reveal the different types of hip fractures. Displaced and impacted right femoral head fracture (**A**). Left femoral neck fracture (**B**). Displaced left intertrochanteric fracture (**C**). (Photo contributor: Meghan Breed, MD.)

KNEE EXTENSOR INJURIES

Clinical Summary

Knee extensor injuries usually occur from three different mechanisms: *quadriceps tendon tear*, *patellar tendon tear*, and *patellar fracture*. Extension may be limited by any disruption of these three. Patients with a history of trauma, other systemic conditions, steroid injections, or fluoroquinolone use are pre-disposed to tendon disruptions.

Quadriceps tendon ruptures are the most common extensor failure and are

more often seen in the elderly. Forced flexion during quadriceps contraction may cause rupture; patients may experience sudden buckling and pain. The patella is inferiorly displaced with proximal patellar tenderness and swelling. A soft tissue defect at the distal aspect of the quadriceps is often apparent.

Patellar tendon rupture occurs in the younger, more active population and results in proximal displacement of the patella with inferior pole tenderness and swelling. Patellar fractures may be transverse (most common), comminuted, or vertical. They may be caused by direct trauma or because of high eccentric tension forces. Tenderness, swelling, and sometimes a palpable defect are typically present. Exam will reveal weakness of knee extension against gravity. Patients with complete tears will not be able to extend their knee.



FIGURE 11.72 ■ Quadriceps Tendon Rupture. Inferior displacement of the patella and a distal quadriceps defect suggest quadriceps tendon rupture. (Photo contributor: Robert Trieff, MD.)



FIGURE 11.73 ■ Patellar Tendon Rupture. Proximal displacement of the patella and inferior pole tenderness in a patient with left patellar tendon rupture. (Photo contributor: Meghan Breed, MD.)



FIGURE 11.74 ■ Patellar Tendon Rupture. A lateral radiograph reveals the proximal patellar displacement seen with complete patellar tendon rupture. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 11.75 ■ Patella Fracture. The inferior pole of the patella is comminuted and displaced. Internal fixation is indicated if the patient cannot actively extend the knee. (Used with permission from Brunicki FC, Andersen DK, Billiar TR, et al. *Schwartz's Principles of Surgery*. 8th ed. New York, NY: McGraw Hill; 2005. © 2005 by McGraw Hill.)

Management and Disposition

Lateral radiographs may help distinguish between the two tendon injuries. Patients demonstrating either partial or complete tendon ruptures can be discharged with their knee in extension and follow-up with orthopedics. Care must be taken to differentiate between a bipartite patella and a fracture.

Nondisplaced and displaced patellar fractures should receive splinting in full extension and orthopedic referral.

Pearls

1. MRI may distinguish partial from complete tears.
2. Patellar fractures may be complicated by future degenerative arthritis.

PATELLAR DISLOCATIONS

Clinical Summary

Patellar dislocations can result from either direct trauma or powerful quadriceps contraction with knee flexion. Commonly, the patella dislocates laterally. Patients who present after a patellar dislocation may state their knee dislocated and spontaneously reduced. A hemarthrosis can be present. Common complaints include pain, swelling, and deformity. The patellar apprehension test (gently displacing the patella laterally or medially while in extension) is often positive in spontaneously reduced patellar dislocations. The patient will become apprehensive as you attempt to reproduce the dislocation. Radiography will confirm the dislocation and help rule out associated fracture. Fractures of the patella or lateral femoral condyle occur in 5% of patients.



FIGURE 11.76 ■ Patellar Dislocation. An obvious lateral deformity of the right patella in a patient with dislocation. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.77 ■ Patellar Dislocation. Lateral deformity of the left patella in a patient with dislocation. (Photo contributor: Suzanne Dooly-Hash, MD.)

Management and Disposition

Lateral dislocations are reduced by flexing the hip, extending the knee, and gently directing pressure medially on the patella. Reduction is easily accomplished and results in immediate relief of pain. Postreduction films should be obtained. After successful reduction, patients require a knee immobilizer in full extension for 4 to 6 weeks. Outpatient referral to a musculoskeletal specialist should be obtained for long-term follow-up.

Pearls

1. A dislocated patella may reduce spontaneously prior to presentation; it is a possibility in any patient who presents with knee pain. This may be elucidated by inquiring about a knee deformity at the time of injury that is no longer present.
2. Complications of patellar dislocation include degenerative arthritis, recurrent dislocations, and chondral or osteochondral loose bodies.

KNEE DISLOCATION

Clinical Summary

Knee dislocations are classified by tibial displacement relative to the femur (anterior, posterior, medial, lateral, or rotatory). They invariably cause multiple ligamentous injuries and are usually the result of motor vehicle collisions, falls, sports, and industrial injuries. Anterior dislocations are more common and usually occur after high-energy hyperextension injuries. Knee dislocations are associated with popliteal artery and common peroneal and tibial nerve injuries. Popliteal artery injury can result from both anterior and posterior dislocations and is more common than nerve injury. Injury can be present despite normal pulses, and if not identified and repaired within 8 hours, amputation may be necessary. Common peroneal nerve injury can cause decreased sensation on the lateral foot, impaired dorsiflexion and eversion, and impaired sensation over the 1st dorsal web space. Knee dislocations can spontaneously relocate, so the physician must maintain a high index of suspicion. Injuries are painful and visually striking. An effusion will often be absent since the capsule has been violated. On exam, the knee will be grossly unstable since dislocations tend to injure most of the surrounding ligaments.



FIGURE 11.78 ■ Anterior Knee Dislocation. Anterior displacement of the tibia in relation to the femur. (Photo contributor: Selim Suner, MD, MS.)

Management and Disposition

Management includes early reduction, immobilization, assessment of distal neurovascular function, and emergent orthopedic referral. Reduction of anterior dislocation is accomplished by flexing the hip 20 degrees and having an assistant apply longitudinal traction on the leg while keeping one hand on the tibia and simultaneously lifting the femur back into position. A posterior splint with the knee in 20 degrees of flexion is used for immobilization and to avoid tension on the popliteal artery. If there is concern for possible arterial injury, consider calculating an ankle-brachial index; a reading of less than 0.9 is abnormal and should raise concern. The patient should be admitted for observation and, likely, angiography.



FIGURE 11.79 ■ Posterior Knee Dislocation. Posterior displacement of the tibia in relation to the femur. (Photo contributor: Paul R. Sierzenski, MD.)



FIGURE 11.80 ■ Posterior Knee Dislocation. Posterior displacement of the tibia in relation to the femur. (Photo contributor: David Efron, MD.)

Pearls

1. Knee dislocations are often associated with a fracture of the tibial plateau.
2. The presence of distal pulses in the foot does not rule out an arterial injury.
3. Vascular repair after 8 hours of injury carries an amputation rate of greater than 80%.

FEMUR FRACTURE

Clinical Summary

While femoral fractures often occur secondary to serious trauma, they can be seen in low-energy injuries associated with the elderly, osteoporosis, or bone cancer. Diagnosis is usually evident on visualization and confirmed radiographically. Significant hematoma formation and blood loss is common. Patients with comminuted femoral shaft fractures are at risk for fat emboli syndrome. For distal fractures, it is important to rule out intra-articular involvement.



FIGURE 11.81 ■ Midshaft Femur Fracture. A closed midshaft femoral fracture. Note the deformity in the middle of the thigh. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 11.82 ■ Femur Fracture. Radiographic examination reveals a comminuted displaced distal femoral fracture. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.83 ■ Pathologic Femur Fracture. A varus-angulated and mildly displaced fracture through an aggressive, moth-eaten, lytic lesion in the proximal left femoral diaphysis. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. *The Atlas of Emergency Radiology*. New York, NY: McGraw Hill; 2013.)

Management and Disposition

Initial management includes stabilization and evaluation for any life-threatening injuries. A large amount of blood loss (average 1000 mL) can occur; patients should have two large-bore lines and be cross-matched for transfusion.

Radiography should include the hip and knee. In-line traction may be required for initial stabilization and to reduce blood loss and pain. This can be held temporarily in the acute setting with a Hare traction or box splint.

An open fracture is an orthopedic emergency; these patients require tetanus prophylaxis, antibiotic coverage, and emergent irrigation and debridement in the operating room. Orthopedic consultation should be obtained with any femur fracture since the majority requires operative fixation and stabilization.

Pearls

1. Pain can be referred. Any injury between the lumbosacral spine and the knee can be referred to the thigh or knee.
2. Vascular compromise can occur and should be suspected with an expanding hematoma, absent or diminished pulses, or progressive neurologic signs.
3. Femur fractures can mask the clinical findings of a hip dislocation; thus, radiographs of the pelvis, hips, and knees should be obtained routinely.

TIBIAL-FIBULAR FRACTURES

Clinical Summary

Fibular fractures may be isolated or associated with tibia injuries. Isolated fibular fractures are caused by direct lateral leg trauma and are anatomically splinted by an intact tibia. Distal fibular fractures may include a disrupted ankle joint, as evidenced by a widened mortise on the AP radiograph. Tibial fractures are classified by their location, amount of displacement, and presence of comminution.

Compartment syndrome can be seen following a tibia fracture, particularly tibial plateau fractures; distal neurovascular status should be documented. Suspect tibial fractures with trauma to the lower extremity, pain, and inability to bear weight.

The Maisonneuve fracture is a combination of an oblique proximal fibular fracture, disruption of the interosseous membrane and tibiofibular ligament distally, and a medial malleolar fracture or tear of the deltoid ligament. It occurs when an external rotational force is applied to the foot, producing a proximal third fibula fracture. Findings include tenderness at the medial and anterolateral ankle joint in combination with proximal fibular tenderness.

Management and Disposition

Treatment is dictated by the degree of pain and the involvement of the ankle joint. Nondisplaced fractures can be treated with an air cast; those with displacement should receive a sugar-tong splint and referred for short-term orthopedic evaluation. Treatment of a Maisonneuve fracture is most commonly operative.

All open fractures require immediate orthopedic evaluation. Closed fractures that cannot be reduced may also need open reduction. Patients with isolated nondisplaced tibial fractures may be splinted, started on ice therapy, and referred for outpatient treatment. Displaced tibial fractures should also be evaluated by orthopedics due to risk for compartment syndrome.

Pearls

1. Early follow-up is required for all tibial fractures due to the risk of compartment syndrome.
2. The peroneal nerve crosses over the head of the fibula and is subject to injury with a Maisonneuve fracture.
3. Some patients with Maisonneuve fracture may complain only of ankle pain; always examine the proximal fibula in patients complaining of ankle pain.



FIGURE 11.84 ■ Tibial-Fibular Fracture. Deformity associated with a midshaft tibial and fibular fracture. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 11.85 ■ Maisonneuve Fracture. (A and B) Abnormal widening of the medial aspect of the ankle mortise and distal tibiofibular syndesmosis, as well as slight lateral subluxation of the talar dome. Because no “exit” fracture is present on the distal fibula, a fracture of the proximal fibula (C) was suspected and confirmed. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. *The Atlas of Emergency Radiology*. New York, NY: McGraw Hill; 2013.)

FRACTURE BLISTERS

Clinical Summary

Fracture blisters are vesicles or bullae secondary to swelling from soft-tissue injury. The most affected areas include the tibia, ankle, and elbow. Patients note formation within 1 to 2 days after trauma and complain of pain, swelling, ecchymosis, and decreased range of motion. Complications include infection, deep venous thrombosis, and compartment syndrome.

Management and Disposition

Blisters are generally left intact, and the underlying fracture is treated.

Pearls

1. Blisters can be seen with other conditions, including barbiturate overdose and burns. In the setting of trauma, however, they frequently indicate an underlying fracture.
2. Blisters are managed in a similar fashion to second-degree burns.



FIGURE 11.86 ■ Fracture Blisters. This patient fell down four steps on the evening prior to presentation. Upon awakening the next morning, he noted ecchymosis, swelling, and blister formation. Radio-graphs revealed fracture of the fibula. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 11.87 ■ Fracture Blisters. Blisters associated with an underlying ankle fracture. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 11.88 ■ Fracture Blisters. Blisters associated with an underlying ankle fracture. (Photo contributor: Selim Suner, MD, MS.)

ACHILLES TENDON RUPTURE

Clinical Summary

Rupture occurs most frequently in middle-aged males involved in athletic activities, but patients with other systemic conditions, steroid injections, or fluoroquinolone use are predisposed. Rupture occurs 2 to 6 cm above the tendon's attachment to the calcaneus. Patients may hear or feel a pop and subsequently develop weakness when pushing off the foot; pain, edema, and ecchymosis may develop. Note that loss of plantar flexion is not necessarily seen as there are other tendons that can compensate. Thompson test can be diagnostic of an Achilles rupture; the patient is placed in a prone position or kneeling on a stool, the knee and ankle are flexed to 90 degrees, and the gastrocnemius muscle should be grasped and squeezed. If the Achilles tendon is even partially intact, then the foot will plantar flex; if ruptured, there will be no foot movement. Radiographic analysis should include a lateral view of the ankle as the Achilles tendon can sometimes be seen. Ultrasound can also be diagnostic.

Management and Disposition

Treatment is either operative or conservative. In either case, the extremity is immobilized in slight plantar flexion, and the patient is made non-weight bearing upon ED discharge. Acute treatment also involves elevation, analgesia, and ice. These patients can be discharged home with orthopedics follow-up.

Pearls

1. Approximately 25% of these injuries are initially misdiagnosed as ankle sprains.
2. Palpation of the tendon alone may not



FIGURE 11.89 ■ Achilles Tendon Rupture. Note the loss of the normal resting plantar flexion due to right Achilles tendon rupture (A). Swelling is also apparent over the injury site. Similar findings are present due to left Achilles tendon rupture (B). (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 11.90 ■ Thompson Test. There is no plantarflexion with squeezing the calf of the affected leg, or less plantarflexion compared with the normal leg. (Adapted with permission from Stone CK, Humphries

ANKLE DISLOCATION

Clinical Summary

The ankle is a hinge joint with the talus sitting in the mortise formed by the distal tibia and distal fibula. Ankle dislocations require forces of great magnitude. Posterior and lateral dislocations are most common, but the ankle can also dislocate medi-ally, superiorly, or anteriorly. A posteriorly dislocated ankle is locked in plantar flexion with the anterior tibia easily palpable. The foot has a shortened appearance with an edematous ankle. Anterior dislocations present with the foot dorsiflexed and elongated. Lateral dislocations present with the entire foot displaced laterally. Ankle dislocations are frequently associated with fractures and may be open.

Management and Disposition

An immediate neurovascular examination should be performed. If vascular compromise is present, the ankle should be emergently reduced, even if it is open. The skin may also be taut and can be at risk for necrosis. If time permits, radiography should include AP and lateral ankle views.

To reduce the ankle, gentle traction is applied to the foot with one hand cupping the heel and the other hand on the dorsal aspect of the foot while an assistant applies countertraction. Neurovascular status should be checked before and after any reduction attempts or immobilization. Reduction usually requires conscious sedation or general anesthesia. Patients should be placed in a posterior splint with immediate referral to an orthopedic surgeon for hospitalization.

Pearls

1. These injuries are commonly associated with malleolar fractures and often require open reduction and internal fixation.
2. Fifty percent of ankle dislocations are open and require surgical debridement.
3. The subtalar joint may also dislocate and appear clinically similar. The lateral

x-ray will show overlap of the talus and calcaneus, while the AP mortise view will show an intact mortise.

4. The worse the ankle appears clinically and radiographically, the easier it is to reduce due to more severe ligamentous disruption.

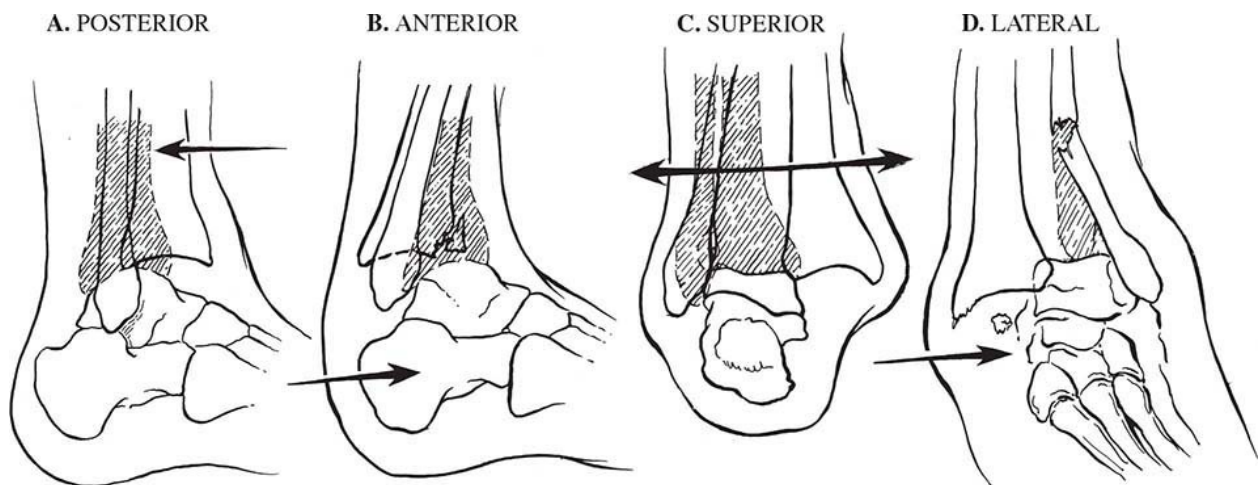


FIGURE 11.91 ■ Ankle Dislocations. This illustration depicts different types of ankle dislocations. Arrows denote direction of the injury force. (Adapted with permission from Simon R. *Emergency Orthopedics: The Extremities*. Norwalk, CT: Appleton & Lange; 1987: p 402.)



FIGURE 11.92 ■ Posterior Ankle Dislocation. Deformity associate with posterior dislocation. (Photo contributor: Mark L. Madenwald, MD.)



FIGURE 11.93 ■ Posterior Ankle Dislocation. The talus is dislocated posteriorly in relation to the tibia.
(Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 11.94 ■ Lateral Ankle Dislocation. The talus is laterally displaced in relation to the tibia. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 11.95 ■ Subtalar Dislocation. Complete dislocation of the hindfoot at the subtalar joints while playing basketball. Neurovascular status was intact, and the ankle, which was promptly reduced after x-ray, showed no associated fracture. (Photo contributor: Kevin J. Knoop, MD, MS.)

ANKLE FRACTURE

Clinical Summary

Most ankle fractures are isolated malleolar fractures, but bimalleolar and trimalleolar fractures occur in up to one-third of cases. While there are numerous classification systems, the simplest is based on the radiographic appearance of these malleoli.

Management and Disposition

Essential treatment is directed at stability and exclusion of associated injuries. Neurovascular status (deep peroneal, superficial peroneal, medial and lateral plantar nerves, posterior tibial artery, and dorsalis pedis artery) should be assessed. The entire length of the fibula, including the proximal portion should be palpated to rule out additional fractures. All ankle fractures, except for fibular avulsions, require immobilization by cast or reduction followed by casting. Except for unimalleolar fractures, most require open reduction and fixation; thus, orthopedic consultation is recommended. In the ED, fractures should be splinted with a posterior mold, kept non-weight bearing, elevated, and iced for 24 hours. Appropriate analgesia and consultation are addressed. The overall goal is to restore anatomic relationships, maintain them during healing, and institute early mobilization. Complications, although rare, include skin necrosis, osteomyelitis, osteoarthritis, and malunion.





FIGURE 11.96 ■ Trimalleolar Ankle Fracture. (A-C) A tiny avulsion fracture of the medial malleolus tip, an oblique fracture of the lateral malleolus, and an oblique fracture of the distal tibia's posterior lip. (D-E)

Reformatted CT images nicely demonstrate the same fractures. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. *The Atlas of Emergency Radiology*. New York, NY: McGraw Hill; 2013.)

Pearls

1. Postreduction arthritis can occur in up to 30% of cases.
2. Avulsion fractures may be treated as stable ankle sprains if they are less than 3 mm in diameter, they are minimally displaced, and there is no evidence of significant ligamentous injury.

ANKLE SPRAIN

Clinical Summary

Classification of these injuries is based on examination; radio-graphs are often not required to guide management. The most common mechanism is an inversion stress that injures, in order, the anterior talofibular, calcaneofibular, and posterior talofibular ligaments. The medial deltoid is the strongest ligament; therefore, isolated injuries are rare, and medial ankle sprains are often associated with lateral malleolar or syndesmotic injuries.

Management and Disposition

Examination should include an anterior drawer test to assess the integrity of the anterior talofibular ligament. The Ottawa Ankle Rules recommend radiographs for: (1) tenderness over the lateral or medial malleolus, or (2) inability to bear weight for four steps both immediately after injury and in the ED. If able to bear weight, injuries are treated with ice packs, elevation, air cast or splint, and early mobilization. If unable to bear weight despite normal radiographs, use a posterior splint, crutches, and close follow-up with orthopedics.

Pearls

1. Ankle injuries are the most common musculoskeletal problem in emergency medicine.

2. Complications include instability, persistent pain, recurrent sprains, and peroneal tendon dislocation.
3. Both malleoli, the proximal fibula, and the 5th metatarsal should be examined for tenderness when evaluating a patient with an ankle sprain.



FIGURE 11.97 ■ Ankle Sprain. Note the dependent ecchymosis and swelling in this patient with a grade 2 left lateral ankle sprain. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 11.98 ■ Medial Ankle Sprain. An eversion injury in a professional athlete caused this rare isolated deltoid ligament sprain. Note localized ecchymosis. Tarsal tunnel syndrome developed over several weeks, taking months to resolve. (Photo contributor: Mimi Knoop.)



FIGURE 11.99 ■ Ankle Sprain. Marked swelling and ecchymosis in a patient with a probable grade 3 ankle sprain. (Photo contributor: Selim Suner, MD, MS.)

CALCANEUS FRACTURE

Clinical Summary

The calcaneus is the most frequently fractured tarsal bone. Patients present with severe heel pain in association with soft tissue swelling and ecchymosis extending to the arch. The normal contour of the heel can be distorted.

Radiographs should include AP and lateral views of the foot and a Harris axial view if possible. Fractures can involve the tuberosities, the sustentaculum, or the body and are classified as intra-articular or extra-articular. Bohler angle should be calculated for all fractures involving the body to rule out a depression, as this will change management. The angle is normally between 20 and 40 degrees; if approaching or less than 20 degrees, a depressed fracture should be suspected.

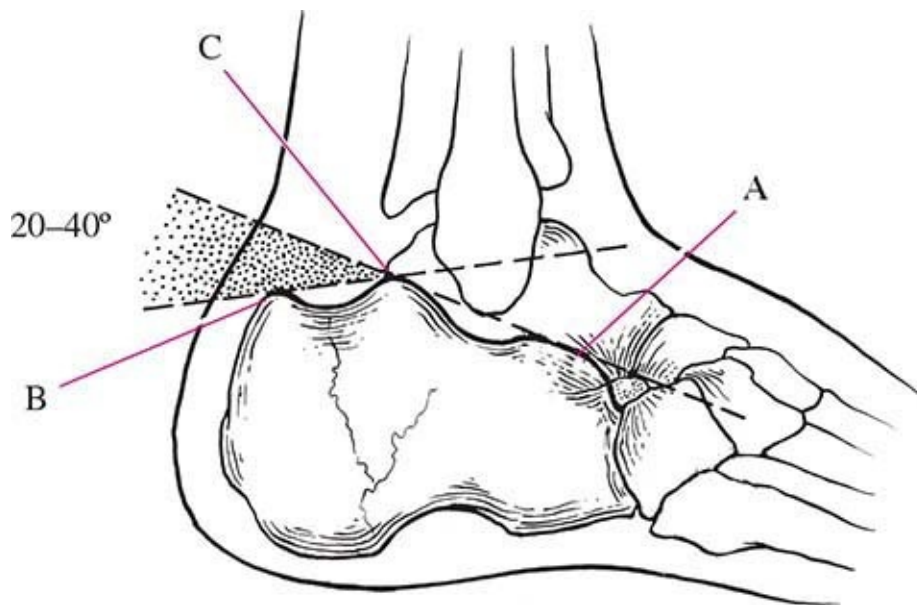


FIGURE 11.100 ■ Calcaneus Fracture: Bohler Angle. The Bohler angle is formed by the intersection of lines drawn tangentially to the anterior (**A**) and posterior (**B**) elements of the superior surface of the calcaneus (**C**). A normal angle is approximately 20 to 40 degrees.



FIGURE 11.101 ■ Calcaneus Fracture. This patient fell from a ladder and struck his heel. A cortical step-off is seen on the inferior aspect of the calcaneus. The Bohler angle has been calculated at approximately 22 degrees. (Photo contributor: Alan B. Storrow, MD.)

Management and Disposition

CT scans should be obtained to further delineate fracture patterns and rule out involvement of the subtalar joint. Intraarticular fractures require orthopedic consultation; open reduction and internal fixation are usually needed. Nondisplaced extra-articular fractures generally heal well with bulky compressive dressings, rest, ice, elevation, and non-weight bearing for 8 weeks. Orthopedic referral is necessary since some may require open reduction. Complications include fracture blisters, nonunion, and chronic pain.

Pearls

1. Calcaneal fracture warrants a diligent search for associated injuries. Twenty percent are associated with spinal fractures, 7% contralateral calcaneal fractures, and 10% compartment syndromes. The subtalar joint is disrupted in 50% of cases. A high index of suspicion for thoracic aortic rupture and renal vascular pedicle disruption must be maintained.

2. Minimally displaced fractures of the anterior calcaneus are easily missed and should be suspected in a patient who does not recover appropriately from a lateral ankle sprain.
3. CT scanning is the optimal imaging technique for characterizing fracture dislocations, while MRI may be used to evaluate ligamentous injury.



FIGURE 11.102 ■ Bilateral Calcaneal Fracture. A fall from a ladder caused bilateral calcaneal fractures. Note swelling and ecchymosis. (Photo contributor: Lawrence B. Stack, MD.)

FRACTURES OF THE FIFTH METATARSAL BASE

Clinical Summary

Patients complain of pain, swelling, decreased range of motion, and tenderness over the lateral aspect of the midfoot. Fractures of the 5th metatarsal base have been generically referred to as Jones fractures. However, acute fractures can be divided into two types depending on their anatomic location; treatment is determined by this delineation.

The classic Jones fracture is a transverse fracture of the 5th metatarsal at the metadiaphyseal-diaphyseal junction, just distal to the 4th and 5th intermetatarsal joint. It occurs when a force is applied to a plantar flexed and inverted foot. This is not to be confused with an avulsion fracture of the 5th metatarsal base, resulting from sudden foot inversion. The avulsion injury is caused by traction on the lateral cord of the plantar fascia.



FIGURE 11.103 ■ Jones Fracture. This patient sustained an injury of the fifth metatarsal and presented with pain and swelling over this site. His radiograph revealed a fracture. (Photo contributor: Cathleen M. Vossler, MD.)

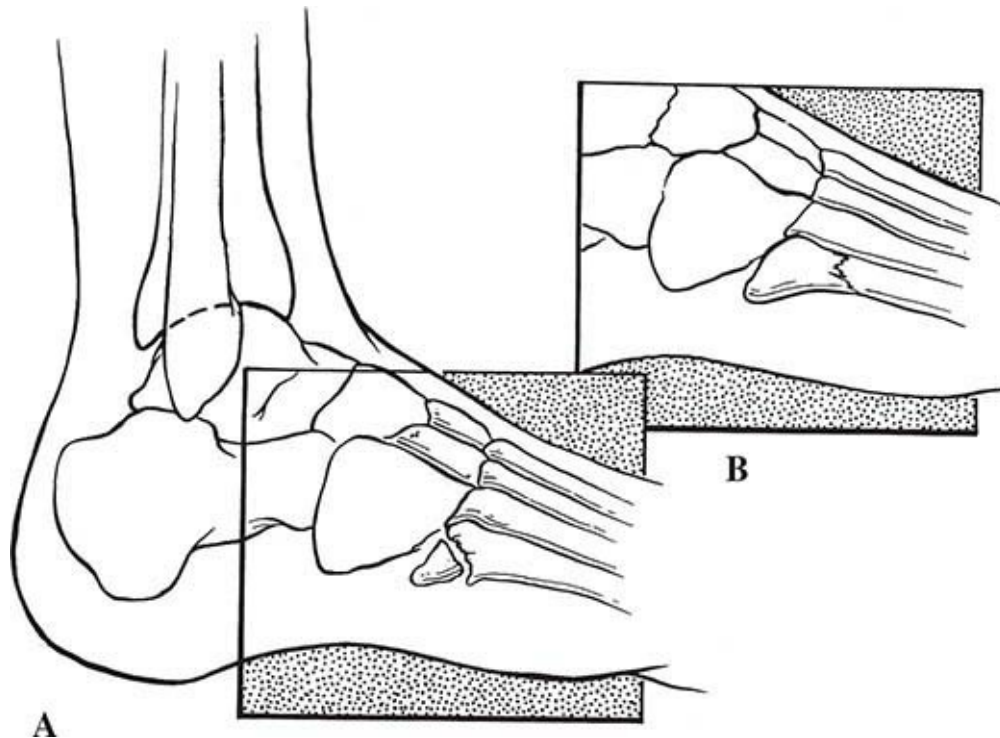


FIGURE 11.104 ■ Fifth Metatarsal Base Fractures. This illustration depicts an avulsion fracture (**A**) and a classic Jones fracture (**B**).

Management and Disposition

The patient with a Jones fracture should be discharged in a posterior splint and crutches and be non-weight bearing for 6 to 8 weeks. Surgical treatment is sometimes required since there is a risk of nonunion.

The avulsion fracture usually heals rapidly and seldom leads to permanent disability. This patient can be discharged in a hard-soled shoe or walking cast for 2 to 3 weeks and can bear weight as tolerated. A significantly displaced fracture may require operative intervention. Both types of patients should be referred to a musculoskeletal specialist as an outpatient.

Pearls

1. The original description was by Sir Robert Jones, who personally sustained this injury while dancing. The avulsion fracture is sometimes referred to as a dancer's fracture.
2. The classic Jones fracture has a high incidence of delayed healing and nonunion.



FIGURE 11.105 ■ Jones Fracture. Radiograph with typical appearance for a diaphyseal fracture of the fifth metatarsal base. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.106 ■ Fifth Metatarsal Avulsion Fracture. Radiograph illustrating an avulsion-type fracture of the fifth metatarsal base. (Photo contributor: Alan B. Storrow, MD.)

LISFRANC FRACTURE-DISLOCATION

Clinical Summary

The Lisfranc joint (tarsometatarsal joint) connects the midfoot and forefoot. It is defined by the articulation of the bases of the 1st three metatarsals with the cuneiforms and the 4th and 5th metatarsals with the cuboid. The Lisfranc ligament anchors the *second metatarsal base* to the medial cuneiform. Disruption of the Lisfranc joint is typically associated with high-energy mechanisms; however, they may occur with less force. Clinical presentation is variable, but severe midfoot pain and the inability to bear weight are usually present. Radiographs may reveal displacement of the metatarsals in one direction

(homolateral) or a split, usually between the 1st and 2nd metatarsals (divergent).

Management and Disposition

Meticulous evaluation of foot radiographs is key to diagnosis. There are three radiographic findings that suggest a Lisfranc injury. Normally, the medial aspect of the 2nd metatarsal should align with the medial borders of the middle cuneiform on the dorsoplantar (DP) foot x-ray. Second, on a DP and an oblique view, there should not be any widening between the 1st and 2nd metatarsals. Third, on the oblique view of the foot, the medial aspect of the 4th metatarsal should align with the medial aspect of the cuboid. A disruption of these relationships is suggestive of a Lisfranc injury and warrants orthopedic evaluation in the ED. Closed reduction can be attempted; however, due to significant ligamentous disruption, the injury is often unstable and necessitates operative stabilization. Tenderness over the Lisfranc complex with normal radiographs can reflect a strain of the complex. Weight-bearing radiographs may unmask joint instability but are often not tolerated in the acute setting.

Pearls

1. Early recognition of Lisfranc fracture-dislocations is facilitated by assessing for normal bony alignments on x-ray.
2. Fractures of the 2nd metatarsal base are considered pathognomonic of a Lisfranc injury since these fractures are often associated with Lisfranc ligament disruption.



FIGURE 11.107 ■ Lisfranc Fracture-Dislocation. This patient presented with extreme midfoot pain and swelling. (Photo contributor: Lawrence B. Stack, MD.)

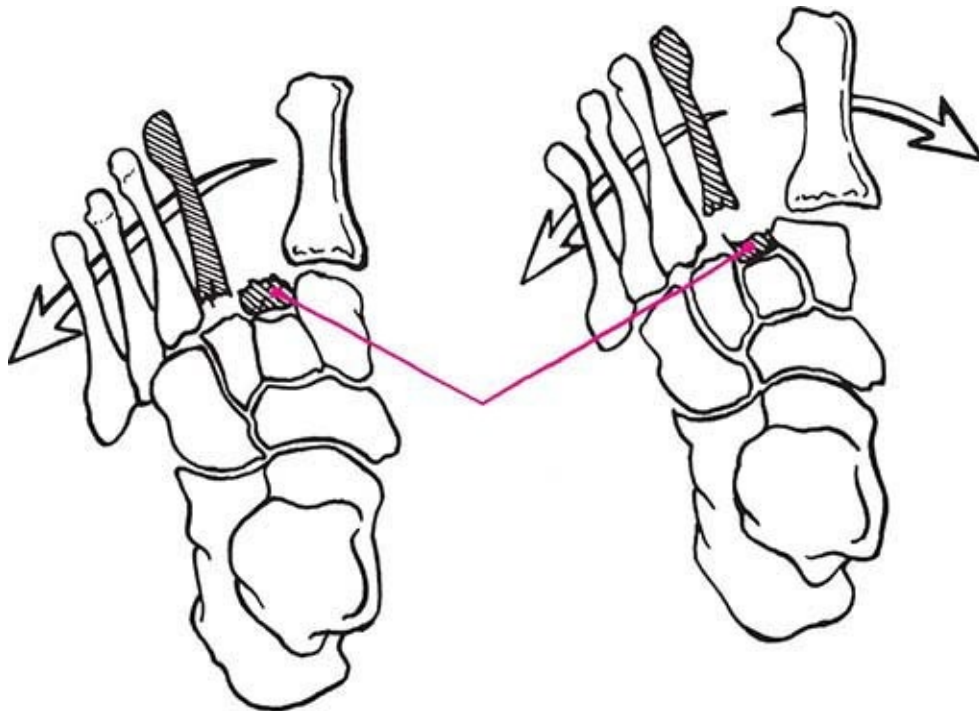


FIGURE 11.108 ■ Lisfranc Fracture-Dislocations. Two different types of Lisfranc fracture-dislocations. Homolateral (*left*) and divergent (*right*).



FIGURE 11.109 ■ Homolateral Lisfranc Fracture-Dislocation. (A-C) DP, oblique, and lateral views demonstrate the radiographic hallmark: lateral subluxation of the second metatarsal base in relationship to the middle cuneiform. The first and third metatarsal bases have also migrated laterally. (C) The lateral view demonstrates superior subluxation in relationship to the cuneiform bones. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. *The Atlas of Emergency Radiology*. New York, NY: McGraw Hill; 2013.)



FIGURE 11.110 ■ Lisfranc Fracture-Dislocation. A divergent Lis-franc fracture-dislocation. Note the disruption of the alignment of the second metatarsal and the medial cuneiform. Sometimes these injuries are not as apparent and comparison radiographs are necessary. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.111 ■ Lisfranc Fracture-Dislocation, Weight-Bearing View. Weight-bearing DP view of both feet reveals a subtle malalignment of the right midfoot with subtle lateral subluxation of the second metatarsal base. The alignment was normal on the non-weight bearing view. Notice the normal alignment of the medial cortices of the second cuneiform and second metatarsal on the left. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. *The Atlas of Emergency Radiology*. New York, NY: McGraw Hill; 2013.)

AMPUTATIONS

Clinical Summary

Major trauma may result in partial or complete removal of a limb. The high kinetic injury, crush, or avulsion forces required often cause extensive damage and heavy contamination. Other significant injuries to the thorax and abdominal cavities should be considered and may be life-threatening.

Management and Disposition

Advanced trauma life support protocols should be rapidly initiated. The injury should be managed as an open fracture with appropriate antibiotics and tetanus prophylaxis. The amputated part should be kept clean, irrigated with sterile saline, wrapped in a sterile dressing, placed in a plastic bag, and put on ice. Except for minor digit amputations, patients should be admitted under trauma and orthopedic consultation for close monitoring of neurologic and vascular status.

While reimplantation is often not possible due to tissue loss and contamination, all patients should receive consideration. Young, healthy individuals with sharp, guillotine injuries without significant avulsion or crushing damage are the best candidates. Radiographs may help delineate the exact spot of injury and reveal associated dislocations or fractures.

Pearls

1. Cooling the amputated part will increase viability from approximately 6-8 hours to 12-24 hours.
2. Postreplantation limb shortening may create significant disability. Proper use of postinjury prosthetics may be the better option.



FIGURE 11.112 ■ Lower Extremity Amputation. A below-the-knee amputation from a motorcycle accident. These injuries are often associated with significant, and potentially life-threatening, abdominal or thoracic trauma. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 11.113 ■ Lower Extremity Amputation. Amputation from a motor vehicle accident. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 11.114 ■ Finger Amputation. Digit avulsion and tendon rupture. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 11.115 ■ Table Saw Amputation. The high kinetic injury of a table saw can produce significant avulsion forces and contamination. (Photo contributor: Selim Suner, MD, MS.)

The authors recognize the contributions of Neha P. Raukar, George J. Raukar, and Daniel L. Savitt to previous editions.

Chapter 12

EXTREMITY CONDITIONS

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Paronychia. (Photo contributor: Lawrence B. Stack, MD.)

CELLULITIS

Clinical Summary

Cellulitis is a common skin or subcutaneous tissue infection with characteristic findings: erythema with poorly defined borders, edema, warmth, pain, and limitation of movement. Fever and constitutional symptoms may be present and are associated with bacteremia. Predisposing factors include trauma, lymphatic or venous stasis, immunodeficiency (including diabetes mellitus), and foreign bodies. Common organisms include group A β -hemolytic *Streptococcus* and *Staphylococcus aureus* in nonintertriginous skin, and gram-negative organisms or mixed flora in intertriginous skin and ulcerations. In immunocompromised hosts, *Escherichia coli*, *Klebsiella* species, *Enterobacter* species, and *Pseudomonas aeruginosa* may be present. There has been an increase in community-acquired methicillin-resistant *S aureus* (CA-MRSA), particularly in cellulitis associated with a cutaneous abscess. Differential diagnosis includes deep venous thrombosis (DVT), venous stasis, erythema nodosum, septic or inflammatory arthritis/bursitis, and allergic reactions.



FIGURE 12.1 ■ Cellulitis. Cellulitis of the left leg characterized by erythema and mild swelling. (Photo contributor: Frank Birinyi, MD.)

Management and Disposition

Treatment of minor cases commonly consists of elevation, analgesia, and oral β -lactam antibiotics with reevaluation in 48 hours. The increase in CA-MRSA has prompted some, especially in highly endemic areas, to advocate coverage with trimethoprim/sulfamethoxazole, clindamycin, doxycycline, or other agents in

addition to conventional β -lactam antibiotics. Admission and parenteral antibiotic administration may be necessary for immunocompromised or toxic-appearing patients or those who do not respond to outpatient therapy.

Pearls

1. Rapidly progressive cellulitis or one that progresses despite treatment with β -lactam antibiotics should raise suspicion for CA-MRSA or deeper infections such as fasciitis.
2. Known risk factors for CA-MRSA include military personnel, prison inmates, and competitive sports players.
3. Routine blood or leading-edge cultures in nontoxic patients are generally low yield.



FIGURE 12.2 ■ Cellulitis. Cellulitis of the right lower extremity characterized by sharply demarcated erythema and edema. (Photo contributor: Robert Tubbs, MD.)

Clinical Summary

A felon is a distal digital pulp space pyogenic infection. Pus collects in spaces formed by the vertical septa anchoring the pad to the distal phalanx. It is characterized by severe pain, exquisite tenderness, erythema, and tense swelling of the distal digit. Visible pus or palpable fluctuance may be present. Complications include deep ischemic necrosis, osteomyelitis, septic arthritis, and suppurative tenosynovitis. The differential includes paronychia, herpetic whitlow, and traumatic hematoma.

Management and Disposition

Incision and drainage is necessary. To ensure complete drainage of the abscess cavity, all affected compartments should be entered. The packing of the abscess space is made with a small, loose-fitting wick to facilitate drainage. Oral antibiotics directed against gram-positive organisms should be used for 10 days, and the packing should be removed or replaced after 24 to 48 hours. Consider treatment for CA-MRSA in addition to standard coverage in highly endemic areas or in at-risk populations.

Pearls

1. Incisions should be made on the ulnar aspects of the index, middle, and ring fingers and along the radial aspects of the thumb and small finger. Incisions should be made dorsal to the neurovascular bundle and not be extended proximal to the distal flexion crease.
2. “Hockey stick” and “fish mouth” incisions are associated with increased occurrence of unnecessary sequelae and are not recommended.
3. If there is radiographic evidence of osteomyelitis or concern for tenosynovitis, consultation with a hand surgeon is required.

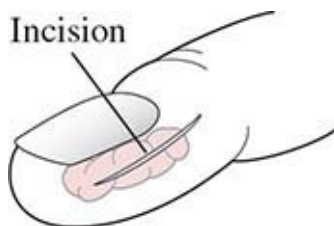


FIGURE 12.3 ■ Felon. Purulence, swelling, and erythema at the center of the palmar pad. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 12.4 ■ Felon. Incision and drainage of a felon. (Reproduced with permission from Stone C, Humphries RL. *Current Diagnosis & Treatment: Emergency Medicine*. 8th ed. New York, NY: McGraw Hill; 2017.)

GANGRENE

Clinical Summary

Gangrene denotes tissue with lost blood supply and undergoing necrosis. The term *dry gangrene* is used for tissues undergoing sterile ischemic coagulative necrosis. Patients with atherosclerotic disease and diabetes are at risk for the development of dry gangrene, usually because of embolization to the forefoot or toe. *Wet gangrene* is associated with bacterial proteolytic decomposition and is characterized by its moist appearance, frequently with blistering.

Management and Disposition

Hospitalization is usually required. The treatment consists of amputation, debridement, and antibiotic therapy as needed. Wet gangrene requires emergent surgical consultation. Underlying vascular pathology must be evaluated and corrected surgically or endovascularly. Patients with systemic toxicity should be resuscitated aggressively.



FIGURE 12.5 ■ Dry Gangrene. Dry gangrene of the toes showing the areas of total tissue death, appearing as black and lighter shades of discoloration of the skin demarcating areas of impending gangrene. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Obtain radiographs to help rule out clostridial myonecrosis (gas gangrene, see related item) and osteomyelitis.
2. Aggressive surgical debridement is necessary for cure in most cases of wet gangrene.



FIGURE 12.6 ■ Dry Gangrene. Complete tissue death characterized by black, desiccated tissue, and lighter areas demarcating areas of impending gangrene. (Photo contributor: David Effron, MD.)



FIGURE 12.7 ■ Wet Gangrene. Note the moist appearance and blistering due to bacterial proteolytic decomposition of gangrenous tissue. (Photo contributor: Robert Tubbs, MD.)

GAS GANGRENE (MYONECROSIS)

Clinical Summary

This infection causes rapid necrosis and liquefaction of fascia, muscle, and tendon. Most cases involve *Clostridium perfringens*; *Streptococcus pyogenes* accounts for most of the remaining cases. Myonecrosis is classically associated with trauma (including surgery) and diabetes. There is edematous bronze or purple discoloration, flaccid bullae with watery brown nonpurulent fluid, and a foul odor. The classic presentation is pain out of proportion to physical findings. Systemic signs are also typically present and may develop rapidly into shock.

Crepitus and appearance of gross air pockets in the tissue are appreciated but may not be present early. The incubation period for clostridia ranges between 1 and 4 days, but can be as early as 6 hours. Decreased tissue oxygen tension and wound contamination are required for the infection to progress. Crepitant cellulitis, synergistic necrotizing cellulitis, acute streptococcal hemolytic gangrene, and streptococcal myositis are some conditions that may be mistaken for clostridial myositis. Often, surgical exploration of the fascia and muscle is required to make the correct diagnosis.



FIGURE 12.8 ■ Gas Gangrene. A foot with significant areas of gangrene and underlying gas. (Photo contributor: R. Jason Thurman, MD.)

Management and Disposition

Treatment includes aggressive resuscitation, broad-spectrum antibiotics (including clindamycin), and tetanus prophylaxis. Surgical debridement or amputation is the mainstay of therapy. Hyperbaric oxygen may have a

synergistic effect in preventing the progression of infection and toxin production.

Pearls

1. Shock and multiorgan failure may be rapidly progressive. Mortality is 80% to 90% if untreated and 10% to 25% when treated appropriately.
2. Clindamycin may improve survival by inhibiting toxin production.
3. Gram stain of gram-positive bacilli with a relative lack of leukocytes may rapidly confirm suspected clostridial myonecrosis.



FIGURE 12.9 ■ Gas Gangrene. Radiograph of the foot in [Fig. 12.8](#) exhibiting pockets of soft-tissue gas tracking up the dorsal surface. (Photo contributor: R. Jason Thurman, MD.)

NECROTIZING FASCIITIS

Clinical Summary

This uncommon, severe infection involves the subcutaneous soft tissues, including the superficial and deep fascial layers. It is usually seen in the lower extremities, abdominal wall, and perianal or groin area. It is commonly spread

from a trauma site, surgical wound, abscess, or decubitus ulcer. Alcoholism, parenteral drug abuse, and diabetes are predisposing factors. Pain, tenderness, erythema, swelling, warmth, shiny skin, lymphangitis, and lymphadenitis are early findings. Later, there is rapid progression of bullae with clear pink or purple fluid and cutaneous necrosis; the skin becomes anesthetic, and subcutaneous gas may be present. Systemic toxicity may be manifest by fever, dehydration, leukocytosis, and frequently positive blood cultures. Type I is polymicrobial and includes anaerobic species. Type II includes group A streptococci.



FIGURE 12.10 ■ Necrotizing Fasciitis. Markedly swollen, dusky, erythematous arm with necrotizing fasciitis in an IV drug user. (Photo contributor: Alexis Lawrence, MD.)



FIGURE 12.11 ■ Necrotizing Fasciitis. Radiograph of the forearm in [Fig. 12.10](#) showing extensive subcutaneous gas. (Photo contributor: Alexis Lawrence, MD.)



FIGURE 12.12 ■ Necrotizing Fasciitis. Large cutaneous bullae on the leg of this patient with necrotizing fasciitis. Note the dark purple fluid in the bullae. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 12.13 ■ Necrotizing Fasciitis. Necrotizing fasciitis with cutaneous necrosis in the inner thigh. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Prompt diagnosis is critical; if made within 4 days from symptom onset, the mortality rate is reduced from approximately 50% to approximately 10%. Initial treatment involves resuscitation with volume expansion, operative debridement, and prompt initiation of broad-spectrum antibiotics.

Pearls

1. Plain radiographs or computed tomography (CT) imaging may detect nonpalpable subcutaneous gas.
2. Hemolysis and disseminated intravascular coagulation may be present.



FIGURE 12.14 ■ Necrotizing Fasciitis. Involvement of the abdominal wall and genitals. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 12.15 ■ Necrotizing Fasciitis. Lower extremity CT demonstrating extensive soft tissue gas.
(Photo contributor: Lawrence B. Stack, MD.)

INGROWN TOENAIL (ONYCHOCRYPTOSIS)

Clinical Summary

Ingrown toenail occurs from impingement and puncture of the medial or lateral nail fold epithelium by the nail plate, allowing growth into the dermis. Granulation tissue causes sharp pain, erythema, and further swelling. The granulation tissue may become epithelialized, preventing elevation of the nail above the nail groove. There is often secondary bacterial or fungal infection. Risk factors include cutting nails short, tightly fitting shoes, and trauma. Differential includes paronychia, felon, and tumor.

Management and Disposition

Elevation of the nail out of the fold and placement of gauze under it to prevent contact, in conjunction with warm soaks, is the initial therapy. If infected, removal of part of the nail and sometimes destruction of the involved nail matrix is necessary. The nail section is removed followed by paronychia fold packing with petroleum gauze or other nonadherent dressing. Consider follow-up with a podiatrist until growth of the nail plate is complete. The destruction of the nail matrix is required for recurrent infected ingrown toenails and is not part of routine emergency care.

Pearls

1. Ingrown toenail is most common in the great toe.
2. Use of antibiotics is not a substitute for surgical excision.



FIGURE 12.16 ■ Ingrown Toenail and Removal. Typical appearance including granulation tissue and erythema. After digital block, a hemostat is paced under the nail in the affected area to the matrix. The portion of the nail under the cuticle (wing) is flipped out and the nail is cut. The use of silver nitrate is optional. Nonadherent gauze or bacitracin should be used before bandaging. (Photo contributor: Lawrence B. Stack, MD.)

LYMPHANGITIS

Clinical Summary

Inflammation of lymphatic channels in subcutaneous tissue is commonly caused by spread of local bacterial infection; group A β -hemolytic streptococcal species are the most frequently implicated. Lymphangitis is characterized by red linear streaks extending, within 24 to 48 hours, from a primary site of infection (eg, abscess, cellulitis) to regional lymph nodes (eg, axilla, groin). The lymph nodes are often enlarged and tender. The differential diagnosis includes cellulitis, trauma, and superficial thrombophlebitis.

Management and Disposition

Rest, elevation, immobilization, and antibiotics are the initial treatment. Coverage for *Streptococcus* and *Staphylococcus* is appropriate. Toxic-appearing patients require admission for parenteral antibiotics. Any patient sent home with oral antibiotics should be followed up in 24 to 48 hours. Patients who subsequently do not show improvement require admission for parenteral antibiotic therapy. Consider treatment for CAMRSA in addition to standard skin coverage in highly endemic areas or in at-risk populations.

Pearls

1. Consider *Pasteurella multocida* with cat and dog bites, *Spirillum minus* with rat bites, and *Mycobacterium marinum* in association with swimming pools and aquaria.
2. Chronic lymphangitis may be associated with mycotic, mycobacterial, and filarial infection.
3. Aspiration of the leading edge is generally not helpful for acute management.



FIGURE 12.17 ■ Lymphangitis. Severe lymphangitis extending from metacarpophalangeal wound up the arm from a “fight bite.” The red streak extends from the hand to the axilla, extending along lymphatic channels. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 12.18 ■ Lymphangitis. The red streak extends from ankle to groin along lymphatic channels. The site of infection was the great toe. (Photo contributor: Liudvikas Jagminas, MD.)



FIGURE 12.19 ■ Lymphangitis. Lymphangitis in the upper extremity, in this case from wrist to upper arm, commonly arises from nail biting. (Photo contributor: R. Jason Thurman, MD.)

LYMPHEDEMA

Clinical Summary

Lymphedema occurs from obstruction of lymphatic channels and is associated

with malignancy, radiation, trauma, surgery, inflammation, infection, parasitic invasion, DVT, paralysis, renal insufficiency, heart failure, cirrhosis, and malnutrition. Lymphedema is characterized by painless pitting edema, fatigue, increase in limb size (particularly during the day while upright), and presence of lymph vesicles. The skin becomes thickened and brown in the late stages.

Management and Disposition

Elevation, pneumatic compression boots and firm elastic stockings, maintenance of healthy skin, and avoidance of cellulitis and lymphangitis are the mainstays of symptomatic treatment. Treatment of the underlying disease may be curative.

Pearls

1. The dorsum of the toes and feet are always involved in lymphedema, unlike other causes of edema.
2. Careful examination for heart failure and screening for renal insufficiency should be completed for all patients.



FIGURE 12.20 ■ Lymphedema. Pitting edema is seen in a woman with lymphedema of the lower extremities. Note how the impression of the thumb remains on the foot in this patient with lymphedema. (Photo contributor: Selim Suner, MD, MS.)

OLECRANON AND PREPATELLAR BURSITIS

Clinical Summary

Bursitis is an inflammatory reaction in a fluid-filled synovial sac, commonly over the subacromial, prepatellar, olecranon, or trochanteric bursa. It is associated with repetitive motion, trauma, or infection. The fluid collection may be bacterial (septic bursitis), gouty, or, most commonly, inflammatory. It produces pain, tenderness, swelling, warmth, and limited range of motion. It is critical to differentiate septic from benign inflammation.

Because bursitis does not involve the intra-articular space, signs and symptoms should be isolated to the bursal area. Typically, intra-articular involvement is associated with pain on minor range of motion, while the discomfort of bursitis occurs with skin and synovial sac stretching at extreme ranges of joint movement. When this differentiation is difficult, fluid aspiration and analysis for cell count, Gram stain, protein, glucose, and polarized microscopy (see Gout in Rheumatologic Conditions) may be helpful. Fluid with greater than 50,000 cells per cubic millimeter, polymorphonuclear neutrophil predominance, increased protein, reduced glucose, and a positive Gram stain are associated with bacterial infection.

Management and Disposition

Rest, compression dressings, and nonsteroidal anti-inflammatory drugs (NSAIDs) are initially used. Bursal injection of local anesthetics mixed with corticosteroids can be considered if septic bursitis has been ruled out, usually in patients who have failed treatment with NSAIDs. Reducing the effusion volume by aspiration may provide temporary relief, although it often recurs. Septic bursitis requires aspiration, gram-positive antibiotic coverage, and consideration of open incision and drainage by orthopedic surgery. Most patients can be treated as outpatients with follow-up.

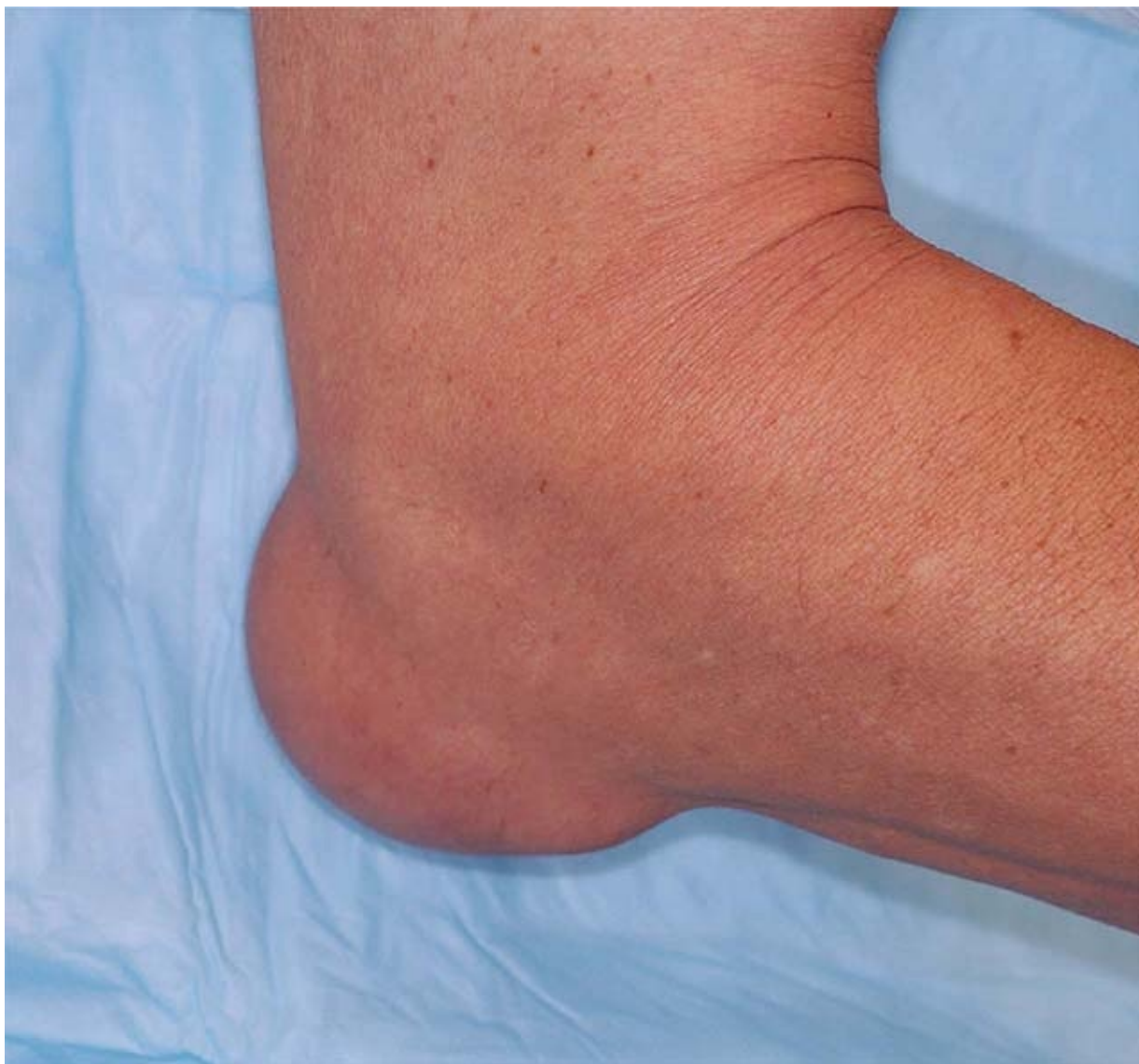


FIGURE 12.21 ■ Olecranon Bursitis. Enlarged olecranon bursa. (Photo contributor: David Effron, MD.)



FIGURE 12.22 ■ Prepatellar Bursitis. Local bursal swelling is evident over the left knee. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 12.23 ■ Septic Prepatellar Bursitis. Erythematous, enlarged bursa in this patient with septic prepatellar bursitis. (Photo contributor: R. Jason Thurman, MD.)

Pearls

1. Septic joint infections in patients who are immunocompromised may have lower synovial fluid leukocyte counts ($<30,000/\text{mm}^3$) than usual ($>50,000/\text{mm}^3$).
2. Bursal fluid from a septic bursitis typically has a lower nucleated cell count than septic joint fluid; lower limits of $2000/\text{mm}^3$ have been proposed.
3. *S aureus* is the most common etiologic agent of septic bursitis.
4. Ultrasound can be used to differentiate between prepatellar and joint effusions.

PALMAR SPACE INFECTION

Clinical Summary

Palmar space infections occur within the deep fascial spaces, commonly from puncture wounds or hematogenous spread. The palm loses its concavity; tenderness, erythema, warmth, and fluctuance are evident. A *thenar space* infection is characterized by swelling over the thenar eminence and pain with thumb movement. With a *midpalmar space* infection, motion is limited and painful for the middle and ring fingers. Cellulitis, local traumatic injury, fractures, and soft-tissue mass are included in the differential.

Management and Disposition

All deep space infections of the hand should be managed by a hand surgeon. Prompt incision and drainage in the operating room is necessary for the best outcome. Parenteral antibiotics against *S aureus* (including CA-MRSA) as well as anaerobes should be started urgently.

Pearls

1. Palmar space infections may cause swelling on the dorsal hand due to the dorsal location of hand lymphatics.
2. There may be minimal signs of swelling over the palmar surface.

3. Infections may be associated with compartment syndrome of the hand.



FIGURE 12.24 ■ Thenar Space Infection. Thenar space infection following injury to the thumb. In this palmar view, erythema and swelling in the right thenar area are evident. (Photo contributor: Richard Zienowicz, MD.)

TENOSYNOVITIS

Clinical Summary

Tenosynovitis, an inflammation of the tendon and surrounding synovial sheath, is characterized by pain and tenderness. *Pyogenic flexor tenosynovitis* is a serious tendon sheath infection resulting from puncture wounds, local extension, or hematogenous spread. It is characterized by the four cardinal Kanavel signs (see [Fig. 12.28](#)). Tenosynovitis may be complicated by fibrosis and adhesions, leading to stiffness, loss of function, and tendon necrosis. *Inflammatory flexor tenosynovitis* is typically due to rheumatoid arthritis, overuse, diabetes mellitus, or connective tissue disorders. The time course is typically more indolent than

pyogenic flexor tenosynovitis, although the sequelae can be similar. *Intersection syndrome* is tenosynovitis of the radial wrist extensors. It causes pain, swelling, and crepitus of these muscle bellies in the distal third of the dorsoradial forearm.



FIGURE 12.25 ■ Flexor Tenosynovitis. Pyogenic flexor tenosynovitis of the fourth finger with fusiform swelling, tenderness along the flexor tendon sheath, and pain on flexion. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 12.26 ■ Tenosynovitis. Pyogenic tenosynovitis of the middle finger with fusiform swelling, tenderness along the tendon sheath, and pain on movement. (Photo contributor: Edmond A. Hooker, MD,

Management and Disposition

It is difficult to distinguish infectious and noninfectious causes early (24-48 hours). Management of noninfectious tenosynovitis includes immobilization and NSAIDs. Broad-spectrum parenteral antibiotics and emergent consultation with a hand surgeon for incision and drainage are mandated with pyogenic flexor tenosynovitis.

Pearls

1. *S aureus* is the most common organism, but *Streptococcus* as well as gram-negative and anaerobic organisms may occur.
2. The most specific sign of tenosynovitis is pain with passive digit extension.
3. Patients with immunocompromised states or recently administered antibiotics may not exhibit the classic tetrad of Kanavel signs.
4. Palpable crepitus may be present in flexor tenosynovitis.
5. Intersection syndrome can be diagnosed by palpable crepitus with wrist flexion/extension over the distal third of the dorsal forearm.



FIGURE 12.27 ■ Inflammatory Flexor Tenosynovitis. The forefinger exhibits redness, uniform swelling, pain to palpation along the flexor sheath, and pain with passive extension. (Photo contributor: Lawrence B. Stack, MD)

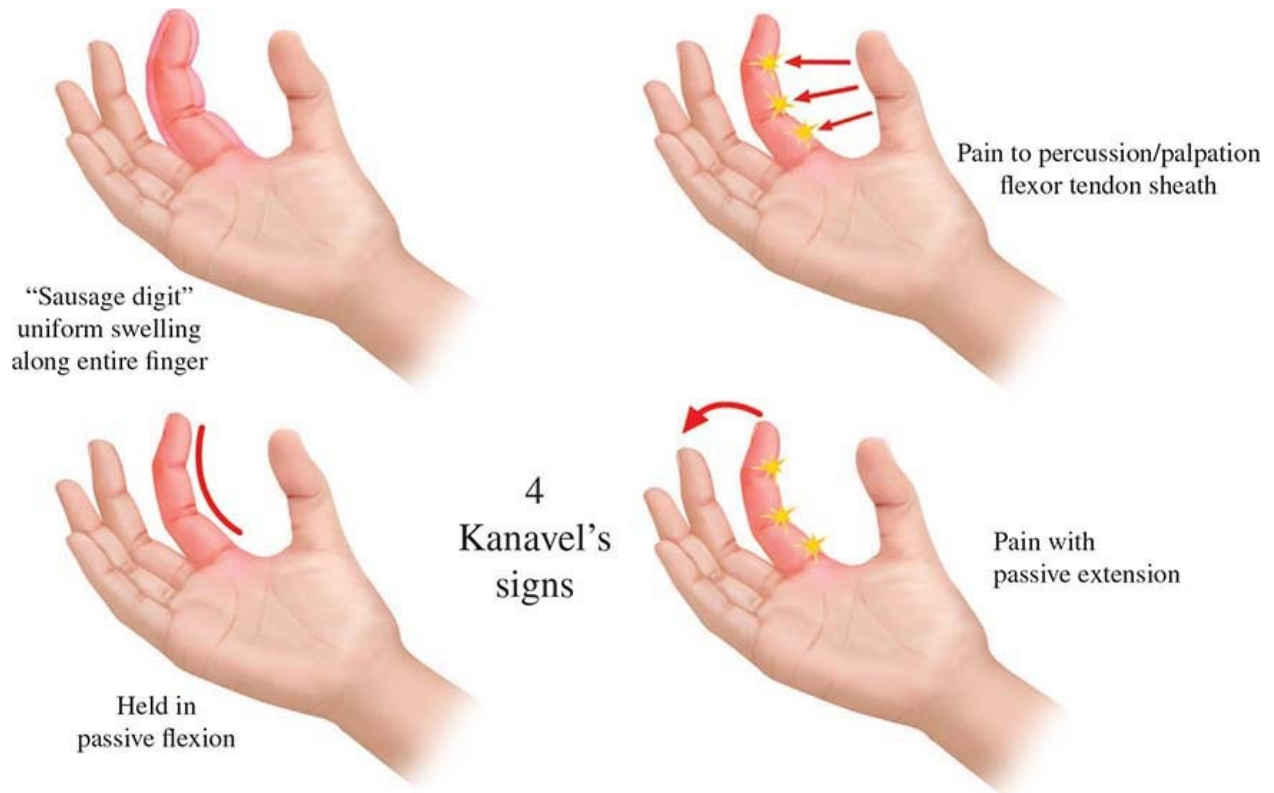


FIGURE 12.28 ■ Kanavel Signs. (Adapted from EM in 5. <https://emin5.com/>)



FIGURE 12.29 ■ de Quervain Tenosynovitis. Swollen and markedly tender area over radial styloid in de Quervain tenosynovitis. (Photo contributor: Robert Tubbs, MD.)

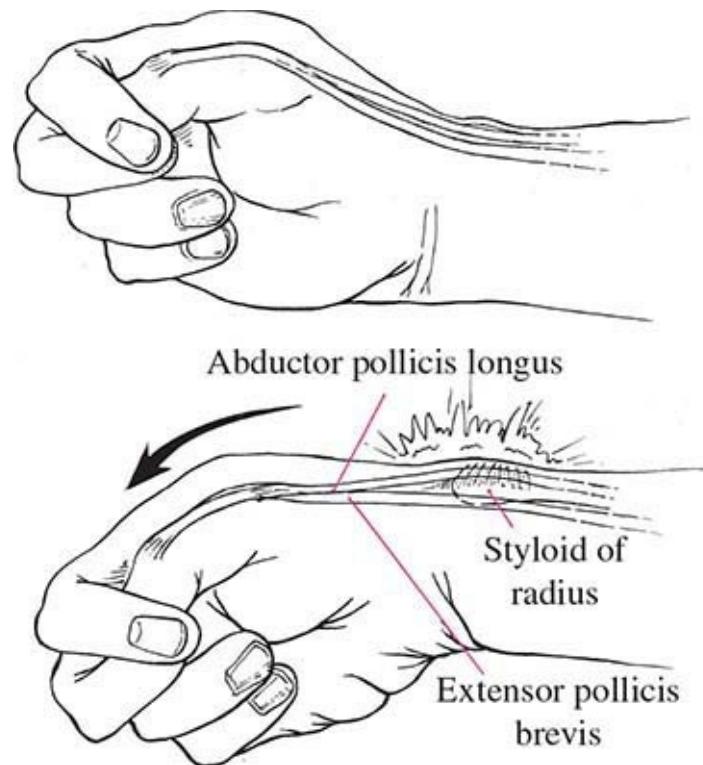


FIGURE 12.30 ■ Finkelstein Test for de Quervain Tenosynovitis. Pain over the radial styloid is elicited with ulnar deviation of the wrist as shown.



FIGURE 12.31 ■ Intersection Syndrome. Discrete swelling at the intersection of muscles that connect to the thumb and underlying wrist tendons. (Photo contributor: Larry Mellick, MD, MS.)

THROMBOPHLEBITIS

Clinical Summary

Thrombophlebitis is superficial thrombosis and inflammation of veins or varicosities characterized by redness, tenderness, and palpable, indurated, cordlike venous segments. Common associations are intravenous (IV) line insertion, irritant IV solutions, trauma, pregnancy, and recent postpartum states. There is little risk of pulmonary embolism when associated with varicose veins or superficial veins distal to the popliteal fossa. However, pulmonary embolism can occur secondary to thrombus propagation to more proximal veins of the deep venous system, particularly with greater saphenous vein involvement. Lymphangitis, DVT, and cellulitis are in the differential.



FIGURE 12.32 ■ Thrombophlebitis. Thrombophlebitis of the superficial leg veins. The thrombosed veins are erythematous, close to the surface, and palpable. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 12.33 ■ Thrombophlebitis. Linear erythema from the popliteal fossa to the mid-calf in thrombophlebitis. (Reproduced with permission from Wolff K, Johnson R, Saavedra AP, Roh EK. *Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology*. 8th ed. New York, NY: McGraw Hill; 2017.)

Management and Disposition

Elevation with warm compresses, rest, and analgesia is sufficient treatment for uncomplicated superficial thrombophlebitis. Superficial thrombophlebitis of the saphenofemoral or iliofemoral system requires treatment as a DVT. Admission is warranted if there are septic signs, progression of symptoms despite treatment, or severe inflammatory reactions.



FIGURE 12.34 ■ Septic Thrombophlebitis. Purulence associated with erythematous thrombosed veins. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Thrombophlebitis of the greater saphenous vein may be confused with lymphangitis, since the lymphatic drainage from the leg runs along the vein.
2. The superficial femoral vein, despite its name, is considered a deep vein, and thrombosis involving this requires standard DVT treatment.
3. Anticoagulation may be considered for lower extremity thrombophlebitis involving the greater saphenous vein close to the femoral junction.
4. Thrombophlebitis of the upper extremity is unlikely to progress to DVT.
5. Suppurative thrombophlebitis should be suspected with high fevers, signs of extensive erythema or purulent drainage, or track marks indicating IV drug use.

PARONYCHIA

Clinical Summary

Paronychia is the most common hand infection and is characterized by

inflammation and pus accumulation along a lateral nail fold. It may spread to involve the eponychium at the base of the nail and the opposite nail fold if untreated. *S aureus* is most frequently isolated, although the infection is generally mixed flora. Felon, dactylitis, herpetic whitlow, hydrofluoric acid burn, and traumatic injury should be considered in the differential.

Management and Disposition

If paronychia is recognized early, prior to frank abscess formation, warm soaks with or without oral antibiotics may be sufficient. After 2 to 3 days, there may be enough pus accumulation along the eponychial fold to warrant incision and drainage. After digital block, a #11 blade or 18-gauge needle is advanced parallel to the nail and under the eponychium at the site of maximal fluctuance. If pus has collected under the nail (subungual abscess), then a portion must be removed to provide drainage. Oral antibiotics should be prescribed; the patient should be reevaluated in 2 to 3 days.



FIGURE 12.35 ■ Paronychia. A paronychia involving one lateral fold and the eponychium. There is swelling, erythema, and tenderness on the dorsum of the distal phalanx. (Photo contributor: Frank Birinyi, MD.)

Pearls

1. Paronychia is associated with nail biting, manicure trauma, and foreign bodies.
2. Germinal matrix damage during nail plate excision may result in nail deformity.
3. It is important to distinguish a paronychia from herpetic whitlow, where incision and drainage is contraindicated.



FIGURE 12.36 ■ Paronychia. A paronychia involving the medial fold and eponychium. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 12.37 ■ Paronychia. Incision and drainage of a paronychia with considerable purulent drainage.

SUBCLAVIAN VEIN THROMBOSIS

Clinical Summary

Thrombosis of the subclavian vein (Paget–von Schrötter syndrome) is an uncommon condition, typically occurring in young patients following exercise and compression injury to the subclavian or axillary vein from a narrow thoracic outlet (effort thrombosis). Pain, tightness, and arm swelling are manifest within a day. Pitting edema develops in the fingers, hand, and forearm. There is no arterial insufficiency, and the pulses are palpable. This syndrome is separate from iatrogenic upper extremity thrombosis, generally as a result of vascular access catheters. There is a 15% risk of developing pulmonary embolism from thrombosis of upper extremity veins; however, large or fatal emboli are very rare. Ultrasound has become the screening test of choice. Superior vena cava syndrome, upper extremity trauma, heart failure, angioedema, and lymphatic obstruction are in the differential.

Management and Disposition

Treatment consists of hospital admission, elevation, local heat, analgesia, and anticoagulation for patients presenting with long-term thrombosis. In cases of acute thrombosis (within 5 days of symptom onset), thrombolysis with direct catheter infusion of thrombolytic agents or mechanical clot retrieval may be considered. Surgical thrombectomy has also been employed. In cases of effort-dependent thrombosis, operative correction of anatomic abnormalities should be accomplished to prevent long-term morbidity.

Pearls

1. Swelling of the neck and face may signify thrombosis or compression of the superior vena cava.
2. The superficial veins in the upper extremity are often dis-tended and do not collapse when the arm is elevated.
3. There is a greater incidence of subclavian vein thrombosis in men and in the

right arm.

4. Ultrasound may be limited in visualizing nonocclusive mural thrombi or those located in the proximal subclavian or innominate veins. In these situations, a CT venogram should be considered.



FIGURE 12.38 ■ Subclavian Vein Thrombosis. Left subclavian vein thrombosis manifested by swelling of the upper extremity. (Photo contributor: Frank Birinyi, MD.)



FIGURE 12.39 ■ Subclavian Vein Thrombosis. Diffuse arm swelling and dilated superficial veins. Note that the veins remain dilated even when the arm is elevated. (Photo contributor: Robert Tubbs, MD.)

CERVICAL RADICULOPATHY

Clinical Summary

Cervical radiculopathy is caused by compression of a nerve root by a laterally

bulging or herniated intervertebral disk, osteoarthritis, or degenerative spondylosis. Pain results from injury to the nerve roots and nerves innervating the dura, ligaments, facet joints, and bone. Common clinical features include pain, paresthesia, and root signs (sensory loss, lower motor neuron muscle weakness, impaired reflexes, and trophic changes). Frequently, there is numbness and tingling following a dermatomal distribution. Magnetic resonance imaging (MRI) is the test of choice to distinguish cervical radiculopathy from disk and bone disease. Electromyography studies may also be helpful in ruling out other disease processes. Trauma, myelopathy, plexopathy, neurofibromatosis, metastatic tumor infiltration of nerve roots, neoplasm, shingles, and central cord syndrome should be considered in the differential.

Management and Disposition

Emergency treatment includes pain control and referral to an orthopedic surgeon or neurosurgeon. Although management may require opioid analgesics, appropriate doses of NSAIDs should also be initiated in patients without contraindications. Oral steroids and gabapentin can also be considered. Since prolonged nerve root compression can lead to permanent deficits, immediate referral is necessary for progressive neurologic signs. Patients with intractable pain, progressive weakness, and myelopathy should be admitted.

Pearls

1. Most radiculopathies resulting from cervical disk disease are seen in the 30- to 60-year age group and in the C5 to C7 region.
2. Patients with acute cervical radiculopathy may present with their upper extremity supported by their head to counteract the cervical root distraction caused by the weight of their dependent extremity.
3. CT myelography may be the next most appropriate study in patients with a contraindication to MRI.

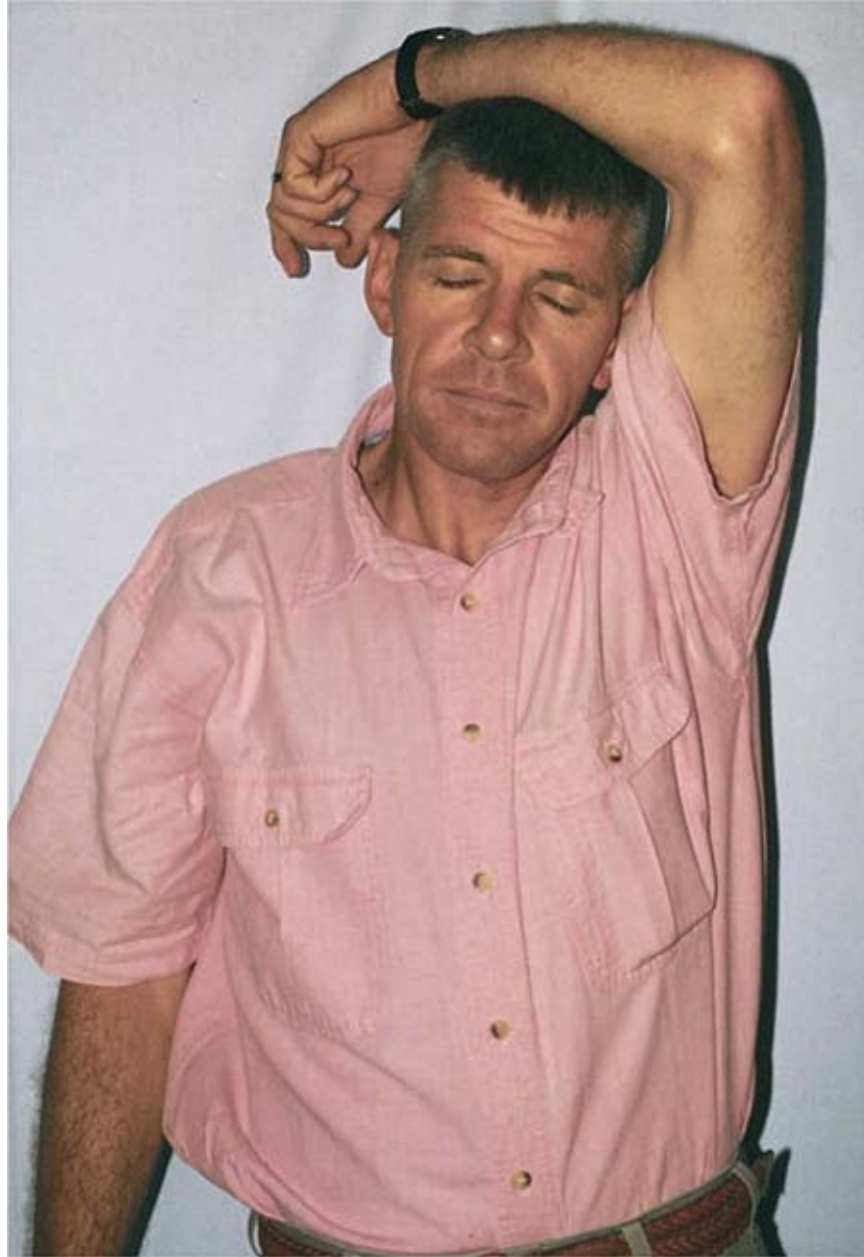


FIGURE 12.40 ■ Cervical Radiculopathy. This is the classic position of relief for cervical radicular pain. This patient presented with severe neck pain with radiation to the extremity. The only way the patient was able to get relief was by holding his arm over his head in the position shown. This patient has a C5-C6 herniated nucleus pulposus. (Photo contributor: Kevin J. Knoop, MD, MS.)

DIGITAL CLUBBING

Clinical Summary

Digital clubbing is characterized by bulbous fusiform enlargement of the distal portion of a digit with loss of the angle between the proximal nail fold and the nail plate (Lovibond angle). The mechanism underlying clubbing is not known. It is associated with multiple medical conditions including carcinoma, intrathoracic sepsis, bacterial endocarditis, cyanotic congenital heart disease, esophageal disorders, cirrhosis, inflammatory bowel disease, pulmonary disorders, atrial myxoma, multiple pregnancies, and pachydermoperiostosis. The incidence of clubbing with each of these conditions is variable. It may be reversible in certain disease processes.

Management and Disposition

Treatment of the underlying condition is indicated.



FIGURE 12.41 ■ Clubbing. Marked digital clubbing can be seen in this patient. Note the hyperemia in the skin folds around the nail. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 12.42 ■ Clubbing. Pronounced digital clubbing. Note the loss of angle between the proximal nail fold and the nail plate (Lovibond angle). (Photo contributor: Robert Tubbs, MD.)

Pearls

1. Patients rarely recognize clubbing in their own fingers even if the condition is marked.
2. Pseudoclubbing is an overcurvature of the nails in both longitudinal and transverse axes, with preservation of the angle between the proximal nail fold and nail plate.

PHLEGMASIA DOLENS

Clinical Summary

Phlegmasia alba dolens (painful white leg, or milk leg) is caused by massive thrombosis of the iliofemoral veins and is characterized by pitting edema of the entire lower extremity, inguinal area tenderness, and a pale extremity secondary to arterial occlusion. *Phlegmasia cerulea dolens* (painful blue leg) arises from massive thrombosis of the lower extremity veins, including the perforators and

collaterals, resulting in venous ischemia with a cool, painful, swollen, tense, and cyanotic-appearing lower extremity. Occasionally, there is bullae formation; compartment syndrome and gangrene may follow. The differential includes arterial insufficiency or thrombosis, aortic dissection, abdominal aortic aneurysm, cellulitis, and lymphedema. Doppler ultrasound and CT venography (most accurate for determining extent) are used for diagnosis.



FIGURE 12.43 ■ Phlegmasia Alba Dolens. Pale discoloration of the lower extremity. (Reproduced with permission from Wolff K, Johnson R, Saavedra AP, Roh EK. *Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology*. 8th ed. New York, NY: McGraw Hill; 2017.)



FIGURE 12.44 ■ Phlegmasia Cerulae Dolens. Bluish discoloration and swelling of the left lower extremity. (Photo contributor: Daniel L. Savitt, MD.)

Management and Disposition

Systemic anticoagulation with heparin should be initiated immediately; vascular surgery or interventional radiology should be consulted. There is a significant rate of pulmonary embolization and a high incidence of postphlebitic syndrome

due to venous valvular incompetence. Endovascular techniques for pharmacomechanical clot dissolution are showing promising results for treating these patients.

Pearls

1. Pregnancy is one risk factor for phlegmasia alba dolens.
2. About 40% of patients with phlegmasia cerulea dolens have an underlying malignancy.
3. Hypotension may result from venous pooling of blood in the lower extremity and diminished venous return.

DEEP VENOUS THROMBOSIS

Clinical Summary

Deep venous thrombosis (DVT) is often associated with intrinsic coagulopathy, impaired fibrinolysis, recent surgery, trauma, immobilization, increased estrogen (pregnancy, oral contraceptives) with smoking, malignancy, prior DVT, inflammatory disease processes, or coronary artery disease. Intravenous catheters are also a major cause of DVT, particularly in the upper extremity. Unilateral swelling and tenderness, classically in the calf and thigh, are characteristic. Doppler ultrasonography is the screening test of choice in most institutions. In selected low-risk patients, a quantitative D-dimer study may be used to rule out DVT. Cellulitis, lymphedema, heart failure, compartment syndrome, myositis, arthritis, and superficial phlebitis should also be considered.

Management and Disposition

Classic treatment is heparin anticoagulation and admission for warfarin loading. However, low-molecular-weight heparin (LMWH) and direct oral anticoagulants (DOACs) have allowed outpatient management in selected patients. In cases of large DVT, consultation with interventional radiology or vascular surgery may be indicated for endovascular thrombectomy. Although DVTs in the calf and superficial veins of the lower extremity do not typically embolize, they can propagate into the deep venous system and may eventually lead to pulmonary

emboli. Serial diagnostic studies are performed to follow the course.



FIGURE 12.45 ■ Deep Venous Thrombosis. This patient has some classic findings of left lower extremity DVT: swelling, erythema, pain, and tenderness. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 12.46 ■ Ruptured Baker Cyst. Comparison of this patient's ankles reveals circumferential swelling around the right side. MRI revealed a ruptured Baker cyst in the right popliteal fossa. Such a presentation may mimic acute lower extremity DVT. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. A Baker cyst, herniation of the synovial membrane through the posterior knee capsule, may rupture, causing unilateral calf swelling like DVT.
2. Patients with unexplained DVT should be screened for occult malignancy.
3. Homan sign (calf pain during passive dorsiflexion) is unreliable in the

diagnosis.

4. Patients with an unclear diagnosis of cellulitis should have an objective study to rule out DVT.
5. The superficial femoral vein is, despite the name, a part of the deep venous system; thrombosis there requires systemic anticoagulation.



FIGURE 12.47 ■ Upper Extremity DVT. Left-sided arm swelling consistent with DVT. (Photo contributor: Lawrence B. Stack, MD.)

DUPUYTREN CONTRACTURE

Clinical Summary

Dupuytren contracture results from shortening and fibrotic changes in the subcutaneous palm tissue and longitudinal bands of the palmar aponeurosis. It may begin as a nodule and then progress to contracture of a finger or fingers. Usually, this is noted at the metacarpophalangeal joint, but the proximal interphalangeal (PIP) or distal interphalangeal (DIP) joint may be involved.

Management and Disposition

Surgery is an effective treatment option when patients lose the ability to extend their fingers. Patient education and referral to a hand surgeon are recommended. Recurrence and development of a contracture in other areas may occur.

Pearls

1. The flexor tendons are not involved.
2. The ring and small fingers are the most commonly involved.
3. Alcoholic liver disease is associated with an increased risk of development.
4. Thickening of the plantar aponeurosis may occur in the foot.



FIGURE 12.48 ■ Dupuytren Contracture. This chronic problem is seen at the most common site: the ring finger. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 12.49 ■ Dupuytren Contracture. Note the contracture at the fifth metacarpophalangeal joint. (Photo contributor: Vineet Mehan, MD.)

ACHILLES TENDONITIS

Clinical Summary

Achilles tendonitis refers to a spectrum of disease ranging from nonpainful nodules to painful swelling of the tendon and paratendon sheath. It most typically develops from overuse, usually after sudden changes in activity or training level. It often occurs in older recreational athletes, who are generally more sedentary and deconditioned. Multiple factors contribute to the condition, including inappropriate footwear, training on poor surfaces, or prolonged running or jumping. High-risk factors include anatomic abnormalities, such as cavus feet, tibia vara, and heel or forefoot varus deformities. A tight Achilles tendon may develop in patients who frequently wear high-heeled shoes.

The most common area of pain is typically 2 to 6 cm proximal to the insertion

site. This is due to a relative paucity of blood vessels in that region, making it more susceptible to inflammation and degeneration from repetitive microtrauma. Pain and tenderness increase with dorsiflexion of the foot. In some cases, a tendon friction rub may be palpable.

Management and Disposition

Careful examination should be performed to distinguish between Achilles tendonitis and tendon rupture. The Thompson test (see Achilles tendon rupture) and palpation of the tendon for gaps or discontinuity should be performed. Ultrasound can also be used. Any patients with suspicion of partial or complete tendon rupture should be splinted and urgently referred to orthopedic surgery. In cases of tendonitis, gentle progressive stretching and lengthening exercises are helpful. In athletes, a reduction in activity is recommended. Use of gel heel inserts may be helpful in the short term, by cushioning and raising the heel, thereby decreasing tendon excursion. Use of ice and NSAIDs is useful in reducing pain and inflammation.

Pearls

1. Fluoroquinolone use has been reported to increase the risk of Achilles tendonitis and possible rupture. Patients presenting with pain, who are currently taking a fluoroquinolone, should have alternative antibiotic therapy considered.
2. Corticosteroid injection for Achilles tendonitis is very controversial and should not be performed in the emergency department.
3. Bilateral tendon involvement, especially at the insertion site, suggests a systemic inflammatory condition such as ankylosing spondylitis, reactive arthritis (Reiter syndrome), or psoriatic arthritis.



FIGURE 12.50 ■ Achilles Tendonitis. Note the significant erythema and thickening of the Achilles tendon on the patient's left compared to the unaffected right side. (Photo contributor: Kevin J. Knoop, MD, MS.)

GANGLION (SYNOVIAL) CYST

Clinical Summary

Ganglion (synovial) cysts are a cystic swelling overlying a joint or tendon sheath. They are the most common soft-tissue tumors of the hand and wrist, although they can arise over any joint. The etiology is currently debated; the most commonly accepted theory is the cyst forms secondary to mucoid degeneration of collagen and connective tissues. It is unclear whether repetitive motion leads to causation, although it does appear to provoke symptoms and possibly lead to cyst enlargement. They may occur in any age, although the majority arise between the 2nd and 4th decades of life.

Ganglion cysts are composed of collagen fiber walls with clear, highly viscous mucin content. They may be unilocular or multilocular. The most common presentations include swelling over or near a joint, as well as pain, limitation of motion, weakness, and paresthesias. They are rarely greater than 2 cm in diameter. The most common location is dorsally over the scapholunate ligament of the wrist (60%-70%), with the volar wrist the next most common site (20%).

Management and Disposition

Ganglion cysts may spontaneously regress; therefore, treatment is generally reserved for symptomatic lesions. Treatments include both nonsurgical and surgical options. The most common nonsurgical option is cyst aspiration, generally using a 16-gauge needle, followed by steroid injection. For recurrent lesions, referral to a hand surgeon for excision is warranted.



FIGURE 12.51 ■ Ganglion Cyst. This large ganglion cyst is located over the scapholunate ligament, the most common location for this entity. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Patients with chronic dorsal wrist pain of unknown etiology should be screened for occult ganglion cysts.
2. MRI or ultrasound may be useful in detecting occult ganglion cysts.
3. Never aspirate volar cysts due to the high risk of injury to neurovascular structures.



FIGURE 12.52 ■ Ganglion Cyst. Classic example of a ganglion cyst located over the scapholunate ligament of the wrist. (Photo contributor: Lawrence B. Stack, MD.)

RAYNAUD DISEASE

Clinical Summary

Raynaud disease refers to reversible ischemia of peripheral arterioles, usually in response to cold exposure or emotional stress. It is more prevalent in women and most often affects the fingers, although the toes, face, ears, nose, and nipples may be involved. Secondary Raynaud phenomenon occurs in association with a related disease process, such as systemic lupus erythematosus or scleroderma.

A typical episode usually starts suddenly with the onset of cold digits associated with sharply demarcated, blue or white, color changes. With rewarming, erythema develops due to a reactive hyperemia. The vasospasm may last for several hours but usually resolves with removal of the initial stimulus.

Management and Disposition

Treatment involves removal of the inciting stimulus. As many cases are induced by going from a warm to a cold environment, active rewarming with warm water soaks or placing in the axilla is generally effective. Sympathetic stimulation may also trigger an episode, so calming patients who are anxious or removing them from stressful situations may be efficacious.

A thorough history and examination should be performed with careful attention paid to signs and symptoms of connective tissue disorders. *Acrocyanosis* (Crocq disease) is a circulatory disorder in which the hands and, less commonly, the feet are persistently cold and blue; some forms are related to Raynaud phenomenon. Counseling patients about methods for reducing the frequency and duration of attacks is helpful, including avoiding sudden cold exposure, minimizing stress, keeping the digits warm, avoiding cigarette smoking, and avoiding sympathomimetic drugs.



FIGURE 12.53 ■ Raynaud Disease. A patient with sharply demarcated color changes consistent with an acute attack of Raynaud phenomenon due to cold exposure. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. A history of cool skin and sharply demarcated color changes is essential for diagnosis.
2. Routine ordering of blood tests, such as erythrocyte sedimentation rate (ESR) or antinuclear antibody (ANA), is not recommended unless other symptoms of connective tissue disorders are present. In these cases, close primary care or rheumatology follow-up is recommended.



FIGURE 12.54 ■ Raynaud Disease. A patient with sharply demarcated color changes characteristic of an acute attack of Raynaud phenomenon. (Photo contributor: Katherine Farmer, MD.)



FIGURE 12.55 ■ Acrocyanosis. Persistently blue and cold fingers. (Photo contributor: Lawrence B. Stack, MD.)

ARTERIAL EMBOLUS

Clinical Summary

Most emboli result from a detached piece of thrombus, often originating from left ventricular thrombus after myocardial infarction or from atrial fibrillation. Other sources include atheroemboli from ruptured plaque, tumor, or foreign bodies such as venous or arterial catheters.

Acute arterial embolization usually occurs at branch points, due to the abrupt change in diameter, and results in distal tissue infarction. The most frequent site is the bifurcation of the common femoral artery (35%-50%). Patients generally present with some or all the “six Ps”: pain, pallor, pulselessness, paresthesias, poikilothermia, and paralysis. Predictors of ischemic insult degree include collateral circulation amount, vessel size, and embolus size. Patients with longstanding peripheral vascular disease often have a greater amount of collateral circulation and can tolerate an acute occlusion better than a patient with normal arteries.

Management and Disposition

Acute arterial embolus is a surgical emergency. Immediate initiation of IV heparin is indicated to prevent further clot propagation. Prompt consultation with a vascular surgeon is imperative, as the rate of limb salvage drastically decreases after 4 to 6 hours. In clear-cut cases, treatment is generally Fogarty catheter embolectomy without prior angiography. Preoperative angiography only prolongs ischemic time and decreases salvage. If it is difficult to distinguish between acute embolic occlusion and in situ thrombosis, preoperative angiography may be considered. Emergent surgical intervention may aggravate thrombosis for in situ thrombus formation.

Pearls

1. Acute arterial embolus generally presents with sudden onset of severe pain. In contrast, in situ thrombosis tends to be more subacute.
2. Aortic dissection may mimic acute arterial embolus. Involvement of multiple sites suggests dissection.



FIGURE 12.56 ■ Arterial Embolus. Note right foot pallor, consistent with an acute arterial embolus, in this case secondary to femoral artery occlusion. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 12.57 ■ Arterial Embolus. Pallor in the right hand secondary to arterial embolus, likely from atrial fibrillation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 12.58 ■ Blue Toe Syndrome. Atherothrombotic micro-embolism, causing transient focal ischemia, occasionally with minor apparent tissue loss, but without diffuse forefoot ischemia. (Photo contributor: R. Jason Thurman, MD.)

MOREL-LAVALLÉE LESION

Clinical Summary

Morel-Lavallée lesions are closed degloving injuries. They occur when the shearing forces of a high-energy trauma cause separation of the skin and subcutaneous tissue from the muscle fascia. This creates a dead space, allowing blood, lymph, and necrotic fat to collect. Drainage of this fluid is impaired since the vascular and lymphatic supply has been injured. Morel-Lavallée lesions are most commonly seen in the hip or proximal thigh.

As time passes after the injury, the collection of fluid becomes encapsulated, and this further prevents spontaneous drainage of the lesion. The diagnosis of a Morel-Lavallée lesion is typically made clinically. A swollen area with fluctuance of the soft tissue can be palpated. There may be overlying skin abrasions and ecchymosis. MRI is the most detailed study to characterize these lesions.

Management and Disposition

Initial management may be conservative with supportive care and compression dressings. However, once the collection of fluid becomes encapsulated, these patients should be referred to surgery for drainage. Complications of untreated lesions include infection and skin necrosis.



FIGURE 12.59 ■ Morel-Lavallée Lesion. Hematoma associated with a Morel-Lavallée lesion in a lateral compression pelvic injury. (Reproduced with permission from Hall JB, Schmidt GA, Kress JP. *Principles of Critical Care*. 4th ed. New York, NY: McGraw Hill; 2015.)



FIGURE 12.60 ■ Morel-Lavallée Lesion. Soft-tissue mass in the proximal calf. (Photo contributor: Chang Hyun Kim, MD. Reproduced with permission from Kim WJ et al. A Morel-Lavallée lesion of the proximal calf in a young trauma patient: A case report. *Medicine (Baltimore)*. 2018;97(41):e12761.)



FIGURE 12.61 ■ Morel-Lavallée Lesion. Intraoperative photograph showing the fibrous capsule of the Morel-Lavallée lesion. (Photo contributor: Karen M. Myrick, DNP. Reproduced from Myrick KM, Davis S. Morel-Lavallee injury a case study. *Clin Case Rep.* 2018;6(6):1033.)

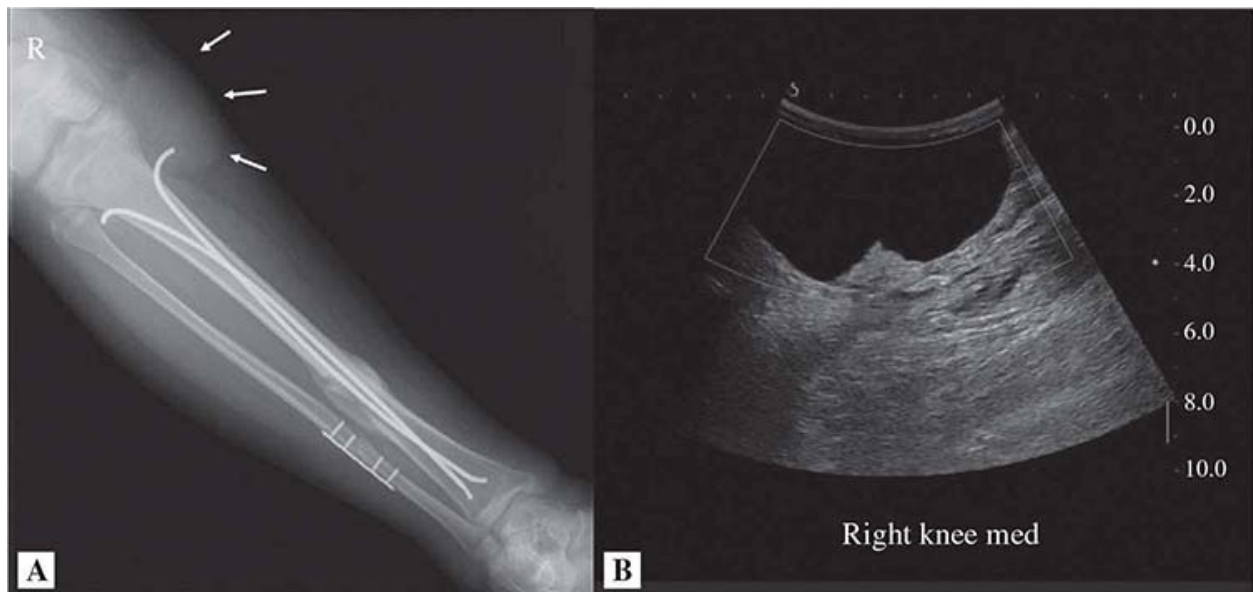


FIGURE 12.62 ■ Morel-Lavallée Lesion. Radiograph (A) showing soft-tissue opacity in the proximal calf, and ultrasound (B) demonstrating a hypoechoic fluid collection. (Photo contributor: Chang Hyun Kim, MD.)

Reproduced with permission from Kim WJ et al. A Morel-Lavallée lesion of the proximal calf in a young trauma patient: A case report. *Medicine (Baltimore)*. 2018;97(41):e12761.)

Pearls

1. On bedside ultrasound, these lesions will appear as a homogenous anechoic or hypoechoic fluid collection. However, echogenic foci within the lesion may be seen if fat globules are present.
2. It is important to differentiate this condition from a hematoma. A hematoma is typically less well defined, and it lacks the fluctuance that is characteristic of the Morel-Lavallée lesion.



FIGURE 12.63 ■ Morel-Lavallée Lesion. MRI showing hyperintensity in the interfascial plane. (Photo contributor: Karen M. Myrick, DNP. Reproduced from Myrick KM, Davis S. Morel-Lavallee injury a case study. *Clin Case Rep*. 2018;6(6):1033.)

CONTRAST EXTRAVASATION

Clinical Summary

Extravasation of IV contrast material into surrounding soft tissues is reported in only 0.5% of CT scans. When this occurs, patients may develop erythema,

ecchymosis, blistering, and swelling of the affected site. If the volume of extravasated contrast material is greater than 50 mL, patients are at increased risk for serious complications including skin ulceration or necrosis. Compartment syndrome is the most feared sequela; it is important to monitor these patients for pain out of proportion on exam, paresthesias, poikilothermia, pallor, paralysis, or pulselessness.

Management and Disposition

When contrast extravasation occurs, the IV infusion should be stopped immediately. Elevation of the affected extremity and application of ice packs can help to relieve pain. If the patient does not have any signs of complications 2 hours after the extravasation, then the patient may be discharged from the emergency department with strict return precautions. The patient should also follow-up within 2 to 3 days to assess resolution of symptoms.

Pearls

1. The risk of contrast extravasation can be minimized by avoiding using small veins distal to the antecubital fossa and by choosing 18- or 20-gauge catheters instead of smaller catheters.
2. If a patient starts to develop symptoms of compartment syndrome, the patient must undergo emergent fasciotomies.



FIGURE 12.64 ■ Contrast Extravasation. Tense swelling and blistering of the dorsal hand after contrast extravasation. (Photo contributor: Ömer Salt, MD. Reproduced with permission from Yurdakul E et al. Compartment syndrome due to extravasation of contrast material: A case report. *Am J Emerg Med*. 2014;32(9):1155. e3. Copyright © Elsevier.)



FIGURE 12.65 ■ Contrast Extravasation. Radiograph demonstrating extravasation of contrast into the soft tissues of the hand. (Photo contributor: Ömer Salt, MD. Reproduced with permission from Yurdakul E et al. Compartment syndrome due to extravasation of contrast material: A case report. *Am J Emerg Med.* 2014;32(9):1155.e3. Copyright © Elsevier.)

LEGG-CALVÉ-PERTHES DISEASE

Clinical Summary

Legg-Calvé-Perthes disease is idiopathic avascular necrosis of the femoral head due to disruption of blood flow. It typically occurs in children aged 4 to 10 years and is more common in males. Patients typically present with hip pain and a limp, which is exacerbated by activity. Legg-Calvé-Perthes may be diagnosed on radiographs. X-rays may show widening of the cartilage space or a subchondral

stress fracture. If left untreated, this will progress to femoral head deformity and subluxation from the acetabulum.

Management and Disposition

It is crucial patients have adequate orthopedic follow-up scheduled. Treatment depends on the phase of the disease. Patients with deformity of the femoral head will typically need surgery to prevent progression of malalignment of the hip joint.

Pearls

1. Radiographs may still be normal early during the disease. Bone scan or MRI should be considered.
2. Legg-Calvé-Perthes disease is an idiopathic condition, so it is important to rule out other potential causes of avascular necrosis including leukemia, sickle cell disease, or corticosteroids.



FIGURE 12.66 ■ Legg-Calvé-Perthes. Radiograph showing deformity of the right femoral head. (Reproduced with permission from Doherty GM. *Current Diagnosis & Treatment: Surgery*. 14th ed. New York, NY: McGraw Hill; 2015.)

SLIPPED CAPITAL FEMORAL EPIPHYSIS

Clinical Summary

Slipped capital femoral epiphysis (SCFE) typically affects adolescents during a growth spurt. This condition is more common in boys, and it typically occurs between ages 13 and 16. Patients present with hip pain or referred pain to the knee. These patients commonly have a limp. On physical exam, the affected leg may be externally rotated and shortened.

Klein's line can be drawn on a plain anteroposterior radio-graph to diagnose this condition. This line is drawn along the superior edge of the femoral neck on a radiograph. In a normal hip, Klein's line should intersect the epiphysis. In an SCFE, Klein's line will not intersect the epiphysis.

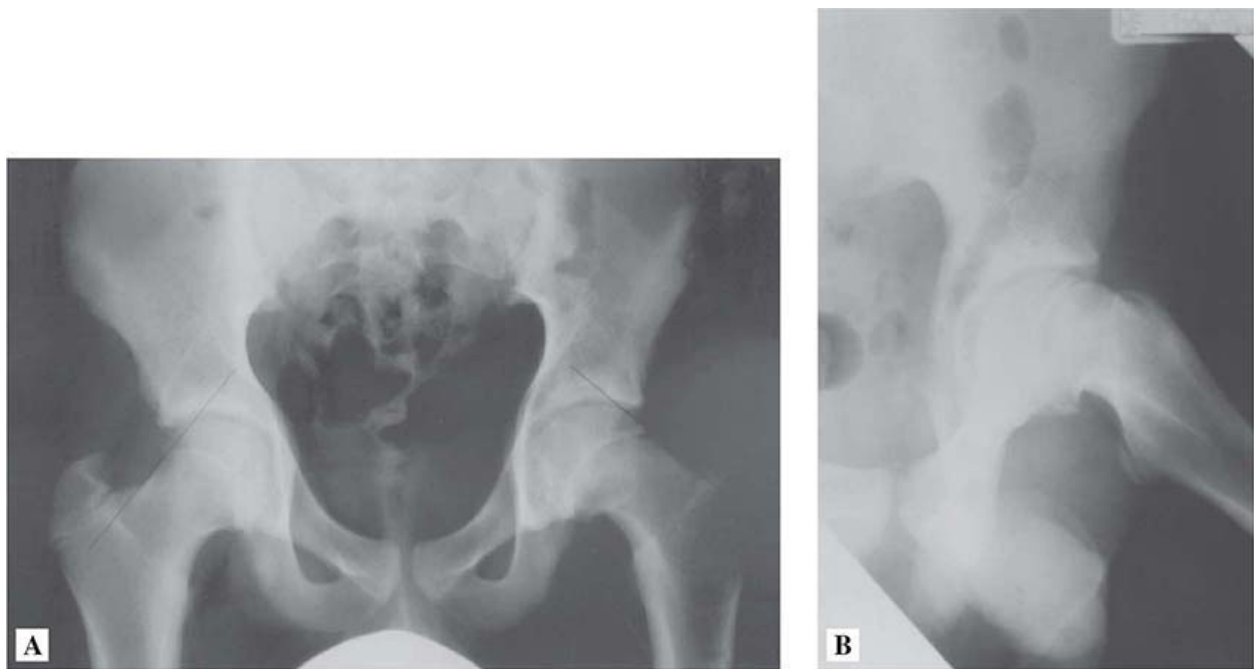


FIGURE 12.67 ■ Slipped Capital Femoral Epiphysis. Anteroposterior radiograph (A) demonstrating left SCFE. Frog-leg radiograph (B) showing posterior displacement in SCFE. (Reproduced with permission from Skinner HB, McMahon PJ. *Current Diagnosis & Treatment in Orthopedics*, 5th ed. New York, NY: McGraw Hill; 2014.)

Management and Disposition

Orthopedic surgery should be consulted for operative management. If left untreated, complications include osteoarthritis, limb length discrepancy, and

avascular necrosis of the femoral head. Patients with an SCFE need to be made non–weight bearing immediately in the emergency department to prevent further deformity.

Pearls

1. The differential diagnosis includes septic arthritis, but patients with SCFE will have normal labs and absence of systemic symptoms.
2. Obesity and hypothyroidism increase the risk of SCFE development.
3. SCFE can be bilateral in 20% of patients.



FIGURE 12.68 ■ Slipped Capital Femoral Epiphysis. Klein's lines (white lines) are drawn along the superior edge of the femoral neck in this radiograph. On the right hip (normal), Klein's line intersects the epiphysis. On the left hip (SCFE), Klein's line is lateral to the epiphysis. (Reproduced with permission from Elsayes KM, Oldham SA. *Introduction to Diagnostic Radiology*. New York, NY: McGraw Hill; 2014.)

The authors acknowledge the contributions of Daniel L. Savitt, Selim Suner, and Robert J. Tubbs to previous editions.

Chapter 13

CUTANEOUS CONDITIONS

J. Matthew Hardin



Kerion. (Photo contributor: Alan B. Storrow, MD.)

STEVENS-JOHNSON SYNDROME/TOXIC
EPIDERMAL NECROLYSIS

Clinical Summary

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are two ends of a continuum of life-threatening, reactive diseases. SJS involves two or more mucous membranes and epidermal detachment of less than 10% body surface area (BSA). TEN involves greater than 30% BSA and often has mucous membrane involvement. TEN and SJS “overlap” occurs between 10% and 30% BSA. The overall mortality of SJS is 1% to 5%, while TEN approaches 30%.

SJS/TEN begins with a nonspecific prodrome of upper respiratory tract symptoms, fever, fatigue, myalgia, headache, and mucous membrane and skin sensitivities. A rash appears 1 to 3 days later, with SJS presenting with mucosal lesions first (erythema, erosions, and hemorrhagic crusting), whereas TEN may have similar or less pronounced mucous membrane involvement. The rash of SJS is generalized, erythematous, and target-like (the center of the target lesions can be dusky and/or bullous). TEN may begin as purpuric, dusky, and target-like patches but then becomes confluent with larger BSA involvement; bullae can form within the rash, and large sheets of epidermis easily separate from the dermis. The involved skin and mucous membranes of TEN are exquisitely tender and may demonstrate the *Nikolsky* sign (lateral pressure on unblistered skin causes the epidermis to slide off; see Video). Progression of involved skin can occur over a single day or slowly evolve over 14 days. In addition to the generalized “skin failure,” life-threatening sepsis, respiratory failure, metabolic derangements, and gastrointestinal hemorrhage may occur. These serious complications may be compounded by underlying comorbidities.



FIGURE 13.1 ■ Stevens-Johnson Syndrome. Moderate hemorrhagic crusting on the lips and target-like macules on the palms and fingers—like erythema multiforme. (Photo contributor: Alan B. Storrow, MD.)

With a few exceptions, SJS/TEN results from a drug exposure, generally within 7 to 21 days before the prodrome. Sulfonamide antibiotics, aromatic anticonvulsants (phenytoin, phenobarbital, and carbamazepine), penicillins, nonsteroidal anti-inflammatory drugs (NSAIDs), allopurinol, lamotrigine, and antiretrovirals are common causes, although over 200 medications, including over-the-counter (pseudoephedrine) and herbal remedies, have been implicated. SJS can also be associated with *Mycoplasma pneumoniae* and rarely herpes simplex virus (HSV) infections.

Management and Disposition

The most critical intervention is to consider a medication cause and stop it (usually requiring withholding all medications acutely). Assess and secure airway status and intravenous access for potentially high-volume replacement. Emergently consult and prepare patient for transfer to an experienced burn unit. Dress denuded skin with nonadherent bandages and normal saline-moistened gauze (transition to antibacterial dressings per burn unit protocol). Meticulous supportive care is the foundation of SJS/TEN treatment. Emergently consult dermatology to help confirm the diagnosis and guide systemic treatments (potential treatments include cyclosporin, etanercept, and intravenous

immunoglobulin [IVIG]). In addition to burn unit and dermatology consultations, early consultation of ophthalmology, pulmonology, gynecology, urology, and gastroenterology should be considered to avoid long-term complications.

Pearls

1. Always examine and document all mucous membranes in SJS/TEN patients.



FIGURE 13.2 ■ Toxic Epidermal Necrolysis. Widespread desquamation. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.3 ■ Toxic Epidermal Necrolysis. Extensive epidermal desquamation. In darker skinned patients, the initial macules/patches may be hyperpigmented and erythema difficult to appreciate. (Photo contributor: Keith Batts, MD.)

2. If a patient notes continued skin pain, even in areas of normal skin, expect additional sloughing and progression of affected BSA.
3. High-risk groups to develop TEN include patients with cancer/hematologic malignancies, HIV/AIDS, immuno-suppression, slow acetylators, genotypes, anticonvulsant use associated with radiotherapy, and specific human leukocyte antigen alleles (HLA-B*1502 associated with carbamazepine and HLA-B*5801 associated with allopurinol).
4. The high mortality associated with TEN should be discussed with the patient and relevant family or designated medical decision makers.



FIGURE 13.4 ■ Bullous SJS/TEN. A bullous form of TEN. (Photo contributor: J. Matthew Hardin, MD.)

ERYTHEMA MULTIFORME

Clinical Summary

Erythema multiforme (EM) begins with symmetric, erythematous, sharply defined extremity or trunk macules, and evolves into a “targetoid” or “bull’s eye” morphology (a flat, dusky, central area with two concentric, erythematous rings). Bullae may appear in the central dusky area (bullous EM). The mucous membranes, typically oral, may become involved and, when severe, raise concern for SJS. The typical targetoid lesions allow a diagnosis to be made clinically (bullae, purpura, and mucosal involvement should prompt a dermatology consultation). The rash usually persists for 1 to 4 weeks.

HSV (frequently labialis) is strongly associated but may not be clinically apparent. Other viruses, bacteria (*M pneumoniae*, *Chlamydia*, *Salmonella*,

Mycobacterium), and fungi (*Histoplasma capsulatum*, dermatophytes) are also associated. Medications account for < 10%; NSAIDs, sulfonamides, anti-convulsants, allopurinol, and antibiotics are responsible for the majority. Physical factors such as trauma, ultraviolet light exposure, and cold have been reported to elicit EM.

Management and Disposition

Prevention of HSV recurrences is essential. Antivirals administered after lesions present have minimal clinical impact, but patients should be referred for future prophylaxis consideration. Use of facial sunscreens and lip balms may help prevent UVB-induced recurrences. With the distinctive clinical findings and no systemic symptoms, patients may be discharged home. Systemic symptoms and atypical presentations with mucous membrane involvement (suggestive of SJS/TEN require admission and dermatologic consultation). Systemic steroids are generally discouraged but can be considered in atypical or severe presentations.



FIGURE 13.5 ■ Erythema Multiforme. Wrist, hand, and fingers with typical central dusky centers surrounded by the concentric “bull’s eye” rings. (Reproduced with permission from Prose N, Kristal L.

Pearls

1. EM does not progress to TEN.
2. The dusky centers help differentiate EM from typical morbilliform drug reactions (no dusky centers) and giant annular urticaria (normal central zone skin).
3. Reassure patients the lesions will completely resolve without scarring.
4. Eye involvement requires a slit-lamp examination and ophthalmologic consultation to exclude active HSV infection.
5. Patients with immunosuppression are more prone to recurrent and prolonged episodes.



FIGURE 13.6 ■ Erythema Multiforme. Symmetric distribution of targetoid macules and plaques. The dusky central zone is more obvious on the left waistline lesions. (Photo contributor: Michael Redman, PA-C.)



FIGURE 13.7 ■ Bullous Erythema Multiforme. Atypical targetoid appearance (only two rings apparent) and bulla formation. (Photo contributor: J. Matthew Hardin, MD.)

DRUG ERUPTIONS

Clinical Summary

Exanthematous drug eruptions present 7 to 14 days after a new medication but may appear sooner if the patient is rechallenged with the culprit medication. A symmetric, erythematous, macular and papular eruption is most frequently encountered. Typically, pruritus and low-grade fever are present. The macules and papules usually become confluent and may progress to an exfoliative dermatitis (rarely to erythroderma). The eruption is progressive over the first few days and, if the culprit medication is stopped, completely resolves over 7 to 14 days.

Acute generalized exanthematous pustulosis (AGEP), a type of drug eruption, presents 1 to 2 days after starting a new medication (typically, a β -lactam or a

macrolide antibiotic). A high fever is usually noted with neutrophilia (90% of patients) and eosinophilia (30%). The rash begins on the face and inter-triginous areas with edematous erythema studded with nonfollicular, small pustules (1-5 mm). Within hours, the pustules become generalized and progressively change to larger flaccid, flat pustules. With the cessation of the offending medication, the pustules slowly resolve over 2 to 3 weeks and are followed by superficial desquamation.

Management and Disposition

While exanthematous drug eruptions may resolve despite the medication's continued use, cessation of the causative agent is paramount. Symptomatic management includes antihistamines and topical corticosteroids. The appearance of AGEF, with pustules, fever, and neutrophilia, is difficult to distinguish from an infectious etiology. Wound and blood cultures should be obtained early. Consult dermatology to help differentiate from pustular psoriasis, cellulitis, EM, bullous diseases, SJS, and TEN. Treatment consists of supportive care and may require systemic steroids. The large surface area of desquamation makes secondary infection a major concern.

Pearls

1. Exanthematous drug eruptions are usually symmetric and pruritic as opposed to viral eruptions, which are usually asymmetric and asymptomatic.
2. Mononucleosis patients taking amoxicillin or HIV patients taking sulfa drugs frequently experience this reaction (augmented by viral infections).
3. The desquamation seen in AGEF is much more superficial than the full-thickness desquamation seen in SJS or TEN.



FIGURE 13.8 ■ Exanthematous Drug Eruption. Coalescing macules and papules—typically, this is a symmetric exanthem. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.9 ■ Acute Generalized Exanthematous Pustulosis. Note the large, flaccid pustule (sterile) and surrounding smaller pustules. The smaller pustules are typical of the initial AGEP presentation. These will eventually slough off and leave a superficial, erythematous erosion. (Photo contributor: J. Matthew Hardin, MD.)

FIXED DRUG ERUPTION

Clinical Summary

Fixed drug eruptions (FDEs) appear 3 to 14 days after 1st exposure. The lesions can appear anywhere, including mucous membranes, but are most common on the face, lips, hands, feet, and genitalia. Single or multiple annular, edematous, well-demarcated plaques are typical. A central vesicle, bulla, or erosion may occur. After stopping the offending medication, the lesion(s) fade over several days to weeks. Residual hyperpigmentation is common. Within 24 hours of reexposure to the culprit medication, the exact rash reappears. The most common offending medications are sulfonamides, NSAIDs, barbiturates, tetracyclines, and carbamazepine.

Management and Disposition

Identify all potential medications (prescription, herbal, and over-the-counter) and

stop the offending drug. Symptomatic treatment with antihistamines and analgesics is sufficient. Refer to dermatology for further evaluation.



FIGURE 13.10 ■ Fixed Drug Eruption. This red to violaceous, pruritic, sharply demarcated patch is a cutaneous reaction to a drug. Repeated exposure will cause a similar reaction in the same location. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.11 ■ Fixed Drug Eruption. Recurring reaction to acetaminophen. (Photo contributor: J. Matthew Hardin, MD.)

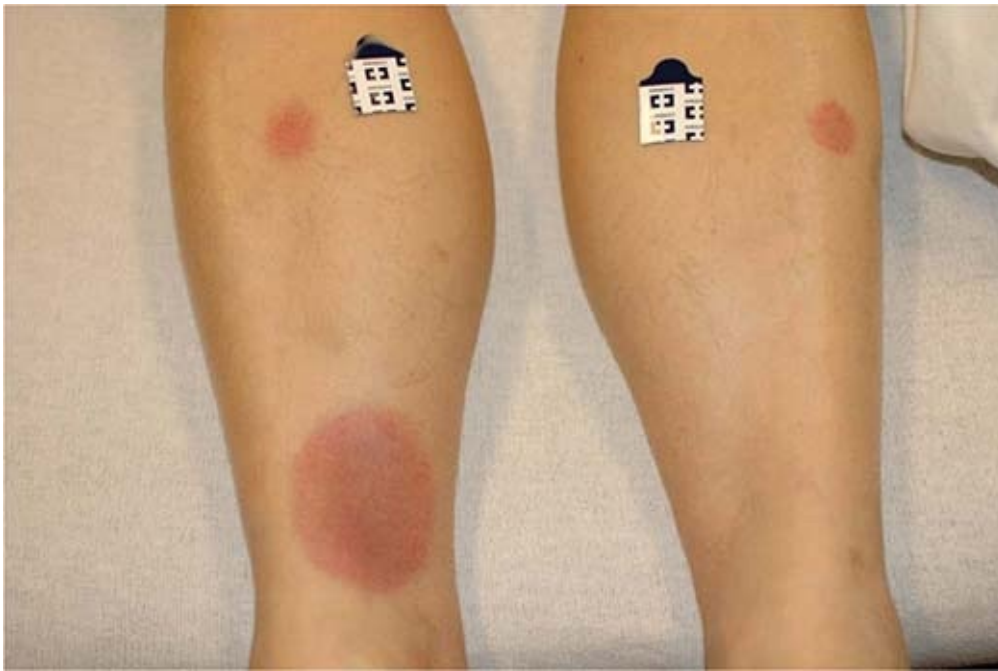


FIGURE 13.12 ■ Fixed Drug Eruption. Recurring reaction to sulfamethoxazole and trimethoprim. (Photo contributor: Edmond A. Hooker, MD, DrPH.)

Pearls

1. Without a thorough history including medication use, FDEs are difficult to identify.
2. Pseudoephedrine, a common over-the-counter medication, is a frequent cause of FDEs.
3. Prolonged hyperpigmentation follows the acute lesions and can be further treated by dermatology.

AUTOIMMUNE BULLOUS DISEASES

Clinical Summary

Autoimmune bullous diseases are uncommon but have dramatic presentations. *Bullous pemphigoid* (BP) results from auto-antibodies to the epidermal basement

membrane and creates tense bullae, frequently located on the proximal extremities. In *pemphigus vulgaris* (PV), the autoantibodies are directed against the epidermal keratinocytes. This results in flaccid bullae (more superficial bullae that easily slough off to form erosions). The delicate bullae and subsequent erosions of PV commonly present in the pharynx, scalp, and trunk. *Paraneoplastic pemphigus* presents with severe oral ulcerations (like SJS/TEN; see related item) and resolves with treatment of the associated malignancy.

Management and Disposition

Considering an autoimmune bullous disease in the differential is the 1st step. Admission with early dermatologic consultation for histologic and immunofluorescent studies should be considered. Systemic corticosteroids and immunosuppressant therapy are required for control. Patients with significant BSA involvement should be treated in a burn unit.

Pearls

1. Oral erosions and ulcerations should always raise the suspicion of autoimmune bullous diseases.
2. The high morbidity and mortality of this disease is now significantly lower due to modern steroid-sparing immunosuppressants and wound care.
3. Always consider medication history and signs of underlying malignancies when evaluating a patient with extensive bullae or erosions.



FIGURE 13.13 ■ Bullous Pemphigoid. Tense blister formation among confluent erosions and plaques. (Photo contributor: Ben Heavrin, MD.)



FIGURE 13.14 ■ Bullous Pemphigoid. Tense bullae on in the distal extremities are a common presentation of BP. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 13.15 ■ Pemphigus Vulgaris. Multiple flaccid bullae now de-roofed and at risk for secondary infection. (Photo contributor: J. Matthew Hardin, MD.)

IMMUNE THROMBOCYTOPENIA

Clinical Summary

Immune thrombocytopenia (formerly idiopathic thrombocytopenic purpura [ITP]) occurs because of platelet injury and destruction. Pinpoint, red, nonblanching petechiae or nonpalpable purpura and ecchymoses are found on the skin and mucous membranes, either spontaneously (platelets $< 10,000/\text{mm}^3$) or at the site of minimal trauma (platelets $< 40,000/\text{mm}^3$). Petechiae are commonly found on dependent areas. Gingival bleeding, melena, hematochezia, menorrhagia, and severe intracranial hemorrhages may also occur. The newly diagnosed form affects children (peak incidence in 2- to 4-year-olds, equal gender distribution) after a viral illness or vaccination and completely resolves in 3 months. The chronic form occurs most often in adults, with women outnumbering men and a prolonged course of thrombocytopenia.

Management and Disposition

Consult hematology for medical management guidance. Treatment can include observation, oral or systemic corticosteroids, IVIG, rituximab, thrombopoietin receptor agonists, and platelet transfusions alone or in combination.

Pearls

1. Isolated thrombocytopenia is the hallmark finding; white blood cell, hemoglobin, and coagulation levels should be normal (if no major hemorrhage).
2. Patients with advanced age, comorbidities, and prior history of hemorrhage have a high risk of associated bleeding (eg, central nervous system). In addition, adults have a higher incidence of thromboembolism. Keep a low threshold for working up these patients and insist on appropriate disposition.
3. The newly diagnosed form of immune thrombocytopenia (symptoms < 3 months in duration) has an excellent prognosis, whereas chronic immune thrombocytopenia (symptoms > 12 months in duration) has varying severity.



FIGURE 13.16 ■ Immune Thrombocytopenia. This thrombocytopenic patient with splenomegaly has pinpoint, nonblanching, nonpalpable petechiae. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 13.17 ■ Immune Thrombocytopenia. Nonpalpable purpura. (Photo contributor: Lawrence B. Stack, MD.)

THROMBOTIC THROMBOCYTOPENIC PURPURA

Clinical Summary

Thrombotic thrombocytopenic purpura (TTP) was previously defined by the following pentad of symptoms: (1) microangiopathic hemolytic anemia, (2) thrombocytopenia, (3) renal abnormalities, (4) fever, and (5) neurologic abnormalities. However, at initial presentation, only 10% of acute TTP patients have all of these symptoms. Most TTP patients will have severe thrombocytopenia (usually $< 30,000/\text{mm}^3$), microangiopathic hemolytic anemia (schistocytes in the blood smear), and a constellation of clinical findings (petechia, purpura, neurologic symptoms, myocardial ischemia, mesenteric ischemia, and renal abnormalities). Approximately 50% of TTP presentations have an associated comorbidity that may trigger the disease (autoimmune diseases, antiphospholipid syndrome, pregnancy, medications, HIV infection, pancreatitis, malignancy, and organ transplantation), whereas the other 50% are idiopathic.

Management and Disposition

The cornerstone of primary TTP therapy is total plasma exchange transfusion (plasmapheresis) with fresh frozen plasma. Emergent consultation with hematology will establish the most expeditious management strategy available.

Pearls

1. Total plasma exchange transfusions have increased TTP survival rate to 80% to 90%.
2. Understanding the varied presentation of TTP patients will lower your threshold to identify them earlier and expedite definitive treatment.
3. Pediatric TTP cases represent only 10% of all TTP cases and are often misdiagnosed.



FIGURE 13.18 ■ Thrombotic Thrombocytopenic Purpura. Bleeding at initial presentation is seen in about 30% to 40% of patients with TTP. (Photo contributor: James J. Nordlund, MD.)

INFECTIVE ENDOCARDITIS

Clinical Summary

Infective endocarditis (IE) is characterized by infection of the endocardium (including the valves, mural endocardium, or septal defect) manifested by many cutaneous findings. Causes are diverse and can be broadly categorized as (1) acute and subacute native valve IE, (2) early and late prosthetic valve IE, (3) intravenous (IV) drug abuse IE, and (4) iatrogenic IE. The latter is associated with recent hospital admission or a procedure causing bacteremia or endocardial damage. *Staphylococcus aureus* is the most common causative organism, but many other bacteria and fungi are implicated. Janeway lesions (septic emboli forming microabscesses) are nontender, small, erythematous (occasionally with central hemorrhage) macules on the palms or soles. Osler nodes (immune complex deposition resulting in small-vessel vasculitis) consist of transient, tender, purplish nodules on the pulp of the fingers and toes. Subungual splinter hemorrhages are black, linear discolorations beneath the conjunctiva and nail plate. Murmurs, retinal hemorrhages, septic arthritis, and significant embolic episodes such as pulmonary embolism or cerebral vascular embolism may also be present.

Management and Disposition

Admission and empiric IV antibiotic therapy are the mainstay of treatment. Obtain blood cultures prior to antibiotic administration. Consult cardiology for emergent, echocardiographic assessment of the cardiac valves. Consult infectious disease for empiric antibiotic recommendations. Emergent surgical intervention may be required for valvular dysfunction or associated abscess.

Pearls

1. Acute bacterial endocarditis presents with fever (> 90% of patients) and a toxic patient; this contrasts with a more insidious presentation of subacute bacterial endocarditis.
2. Previous episode of IE, congenital heart disease, IV drug abuse, prosthetic heart valves, recent medical procedures, and cardiac transplants with valvulopathies are risk factors.

3. Although splinter hemorrhages in the nail bed have long been associated with IE, the most common causes are trauma, psoriasis, and onychomycosis. Proximal nail bed splinter hemorrhages have a higher likelihood of systemic disease (including IE, drug reactions, vasculitis, antiphospholipid antibody syndrome, and trichinosis).



FIGURE 13.19 ■ Janeway Lesions. Peripheral embolization to the sole, resulting in a cluster of nontender, erythematous macules known as Janeway lesions. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.20 ■ Janeway Lesion. Embolization to the hand in a patient with infectious endocarditis.
(Photo contributor: Alan B. Storrow, MD.)



FIGURE 13.21 ■ Osler Nodes. Tender, subcutaneous, violaceous nodules in the pulp of the fingers known

as Osler nodes. (Photo contributor: Armed Forces Institute of Pathology, Bethesda, MD.)

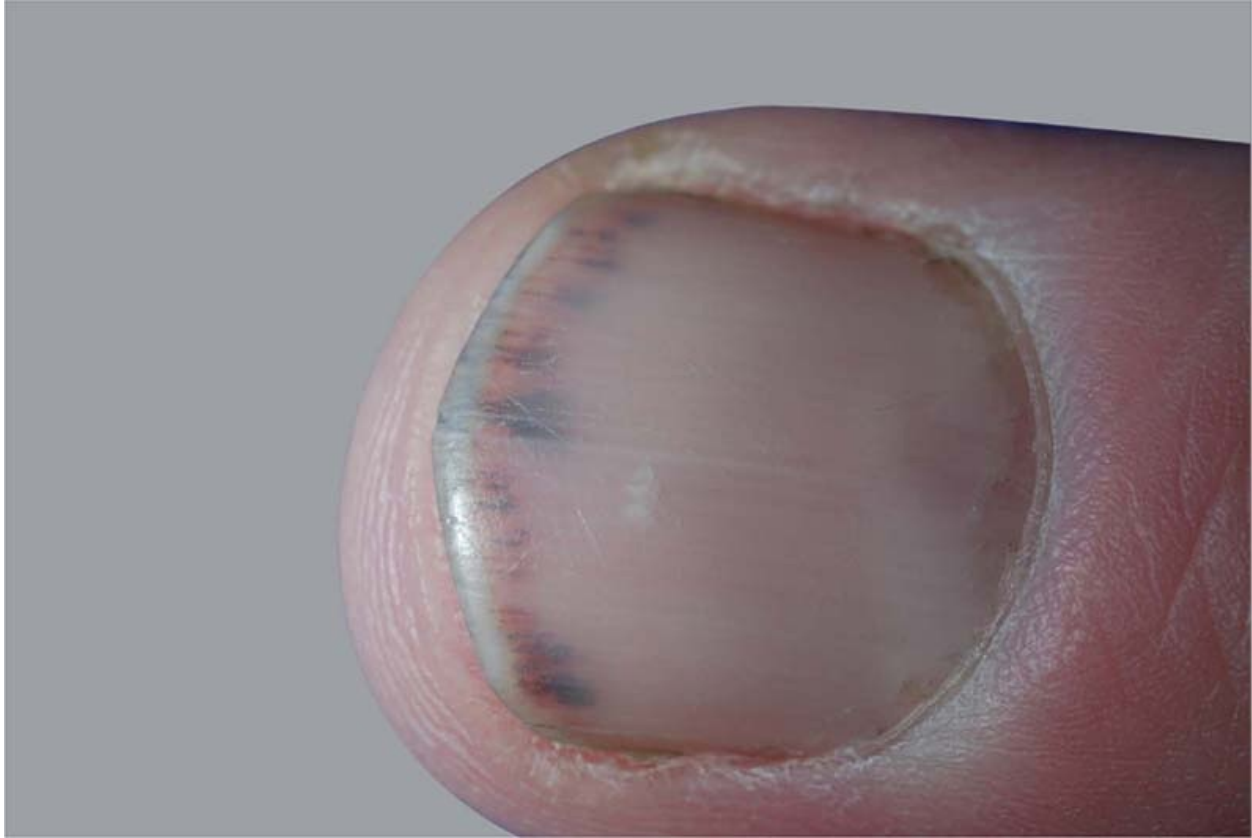


FIGURE 13.22 ■ Splinter Hemorrhages. Note the splinter hemorrhages along the proximal and distal aspect of the nail plate—emboli from subacute bacterial endocarditis. (Photo contributor: R. Jason Thurman, MD.)

ERYSIPELAS

Clinical Summary

Erysipelas is a group A streptococcal cellulitis (groups G, B, C, and D can be implicated as well as other bacteria causing an erysipelas-like infection) involving the skin; the infection is more superficial than cellulitis. The most affected sites are the face and the lower extremities. Sudden onset of fever, chills, and malaise is followed by the appearance of an erythematous, edematous, and painful plaque. The characteristic border is sharp and elevated. This nonpitting edematous plaque is different from typical cellulitis due to dilation of the superficial lymphatics. Regional lymphadenopathy and

lymphangitis may be present. Patients with chronic lymphedema are prone to repeated infections.

Management and Disposition

All infections require rest, elevation, and antibiotics. Mild presentations may be treated on an outpatient basis with oral penicillins. More severe illness or toxicity requires hospitalization and IV antibiotics.

Pearls

1. The sharp elevated border is diagnostic of erysipelas.
2. A similar shiny, erythematous (although more violaceous) plaque on the face of a febrile child may be caused by *Haemophilus influenzae* type B in nonimmunized or immunosuppressed individuals.
3. Consider this diagnosis in any patient with chronic lymphedema.



FIGURE 13.23 ■ Erysipelas. Sharply demarcated and elevated erythema. (Photo contributor: David Effron, MD.)



FIGURE 13.24 ■ Erysipelas. Note the well-demarcated, edematous, erythematous, shiny plaque. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

ERYTHEMA MIGRANS

Clinical Summary

Borrelia burgdorferi is the tick-borne spirochete responsible for Lyme disease, and erythema migrans is the 1st cutaneous sign. Erythema migrans typically

presents 1 to 2 weeks after the bite. The initial prodromal symptoms of fever, myalgias, arthralgias, and headache are followed by a macule or papule progressing to a plaque at the bite site. This plaque expands (usually to 5 cm or larger) its red, raised border as it clears centrally, leading to an annular appearance (“bull’s eye”). The plaque may burn and is rarely pruritic. Less frequently, secondary erythema migrans–like lesions can appear due to multiple bites or spirochetemia. Erythema migrans is seen in 60% to 90% of patients and represents the early localized stage.

Management and Disposition

The duration and choice of antibiotic depend on the features (presence of erythema migrans, early disseminated disease with mild or severe symptoms, cranial nerve palsies, heart block, meningitis, and radiculopathy). Doxycycline is the drug of choice for adults and children over 8 years of age. Pregnant or lactating females and children younger than 8 years of age should be treated with amoxicillin. Patients with minimal symptoms may be treated and followed up on an outpatient basis. Those patients with significant toxicity (severe disseminated rash, systemic symptoms, meningitis, radiculopathy, or third-degree heart block) require admission, supportive care, and IV antibiotics. Consult the Centers for Disease Control and Prevention (CDC) for the most up-to-date treatment regimen.

Pearls

1. Over 50% of untreated cases of erythema migrans can progress to an asymmetric, episodic, oligoarticular arthritis weeks to months after the initial infection.
2. Facial nerve palsies are the most common neurologic manifestation of untreated Lyme disease.
3. Early serology testing may not demonstrate elevated anti-*Borrelia* antibodies, and this does not rule out the diagnosis. Follow current CDC case definitions and recommendations.
4. A similar erythema migrans–like rash has been described with the southern tick-associated rash illness (STARI).
5. Avoid Lyme disease single-dose doxycycline prophylaxis in areas endemic for Rocky Mountain spotted fever (RMSF); this inadequate treatment for RMSF could delay eventual RMSF diagnosis and treatment.



FIGURE 13.25 ■ Erythema Migrans. The eruption of Lyme disease forms at the site of the tick bite. The initial papule forms into an expanding oval of erythema. There is central “bull’s eye” clearing as the erythema progresses. (Photo contributor: James Gathany, Public Health Image Library, US Centers for Disease Control and Prevention.)



FIGURE 13.26 ■ Erythema Migrans. An atypical erythematous patch of erythema migrans—always have a high level of suspicion. (Photo contributor: David Effron, MD.)

ROCKY MOUNTAIN SPOTTED FEVER

Clinical Summary

Rickettsia rickettsii, the causative organism of RMSF, is transmitted by the bite of an infected tick. Fever, headache, rigors, abdominal pain, myalgias, and malaise occur 2 to 14 days after inoculation. Three to 5 days after the onset of symptoms, the rash begins with erythematous, blanching macules on the distal extremities (wrists and ankles). This is followed by centripetal spread to the trunk and the palms and soles. The lesions evolve into papules and petechia. Without treatment, RMSF has a 25% mortality; delayed diagnosis and treatment result in 3% to 4% mortality.

Management and Disposition

Prompt initiation of doxycycline is recommended for adults and children. Consultation with an infectious disease specialist should be initiated to help guide treatment; this is especially critical for pregnant females. Mildly ill patients may be treated with oral antibiotics on an outpatient basis if close follow-up can be confirmed. More severely ill patients require admission.

Pearls

1. Typical distribution of extremity lesions should be treated as RMSF until proven otherwise. Treatment should never be delayed for laboratory confirmation.
2. Most cases occur between April and October, with the highest US incidence occurring in the Southeast and South-Central states (not Rocky Mountain states).
3. Forty percent of patients do not recall the inciting tick bite.
4. Up to 15% of cases present without any cutaneous manifestations. Patients with darker skin types may have less obvious cutaneous findings.
5. In pediatric patients less than 9 years old, the recommended course of doxycycline has a negligible effect on permanent tooth discoloration. Previously recommended chloramphenicol has a higher mortality rate than doxycycline.



FIGURE 13.27 ■ Rocky Mountain Spotted Fever. These erythematous macular lesions will evolve into a petechial rash that will spread centrally. (Photo contributor: Daniel Noltkamper, MD.)



FIGURE 13.28 ■ Rocky Mountain Spotted Fever. Early erythematous and hemorrhagic macules and papules appeared initially on the ankles of an adolescent. (Used with permission from Wolff K, Johnson RA, Saavedra AP. *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*. 7th ed. New York, NY: McGraw Hill; 2013: Fig. 25-49.)

DISSEMINATED GONOCOCCUS

Clinical Summary

Disseminated gonococcus (gonococcemia) is a systemic infection that presents in 1% to 3% of untreated mucosal (urethral, endocervical, pharyngeal, and rectal) gonorrhea infections. The hematogenous dissemination of *Neisseria*

gonorrhoeae results in fever, arthralgias, and scattered pustules. The initial lesion is an erythematous macule that evolves into a papule and hemorrhagic pustule. Petechial or purpuric macules can occur. These lesions are few, asymmetric, painful, and predominantly located on the distal extremities. The spectrum varies from skin lesions alone to associated tenosynovitis, septic arthritis, endocarditis, and meningitis.

Management and Disposition

Initiation of IV ceftriaxone and a single dose of azithromycin can be started in the emergency department (ED) with subsequent admission. Confirmatory testing with nucleic acid amplification techniques can be obtained to quickly confirm infection, but blood cultures should be obtained (prior to antibiotic initiation) to elucidate potential antibiotic-resistant strains. Dermatology consultation may help confirm the diagnosis.

Pearls

1. Pregnant or menstruating females have a higher risk of disseminated gonococemia.
2. Disseminated gonococemia is the most common cause of septic arthritis in young, sexually active adults.
3. Obtain blood cultures prior to giving antibiotics as antimicrobial resistance is increasing.



FIGURE 13.29 ■ Disseminated Gonococcus. Erythematous macules of disseminated gonococcus—these will evolve into hemorrhagic pustules. (Photo contributor: David Effron, MD.)



FIGURE 13.30 ■ Disseminated Gonococcus. Vesiculopustule (**A**) and hemorrhagic pustule with surrounding erythema (**B**). (Photo contributors: A: Stephan E. Russ, MD, B: David Effron, MD.)

ECTHYMA GANGRENOSUM

Clinical Summary

Ecthyma gangrenosum is a cutaneous manifestation of *Pseudo-monas aeruginosa* septicemia occurring in an immunocompromised or neutropenic patient. A toxic appearance with fever, abnormal vital signs, and altered consciousness is common. The initially erythematous macules develop bullae or pustules surrounded by violaceous halos. Bullae become hemorrhagic and rupture (pustules breakdown), becoming necrotic ulcers with central black or gray eschars. The time course from macule to eschar can occur within 12 to 24 hours. The lesions primarily affect the anogenital areas (> 50%) but may appear anywhere on the body. Ecthyma gangrenosum is seen in up to 13% of patients with *P aeruginosa* septicemia and rarely in nonbacteremic patients. Other bacteria causing septicemia (*Aeromonas hydrophila*, *Aspergillus*, *Burkholderia cepacia*, *Candida*, *Citrobacter freundii*, *Escherichia coli*, *Fusarium*, *Pseudomonas stutzeri*) may present with similar ecthyma gangrenosum–like lesions.

Management and Disposition

Ecthyma gangrenosum is associated with life-threatening *P aeruginosa* sepsis and a high mortality. Appropriate supportive care measures and rapid initiation of antipseudomonal antibiotics are required; delaying antibiotics can increase mortality. Dermatology consultation for diagnosis confirmation and infectious disease for antibiotic guidance can be helpful. Patients with oncologists should consult the oncology service and follow oncology protocols.

Pearls

1. Patients with ecthyma gangrenosum are severely immuno-suppressed, and many other bacteria and fungi can cause similar lesions.
2. Without examining the entire patient, these lesions can be completely missed.
3. Multiple lesions, delayed diagnosis, and delayed institution of appropriate antibiotics portend a poor prognosis.



FIGURE 13.31 ■ Ecthyma Gangrenosum. A typical hemorrhagic bulla of ecthyma gangrenosum secondary to pseudomonal sepsis. (Photo contributor: James Mensching, DO.)



FIGURE 13.32 ■ *Pseudomonas aeruginosa* Infected Ulcerations. Note the green colored crusting. (Photo contributor: J. Matthew Hardin, MD.)

NONGENITAL HERPES SIMPLEX INFECTIONS

Clinical Summary

Primary or recurrent HSV infection presents as grouped vesicles on an erythematous base. Recurrent HSV is typically less severe than the primary infection. A prodrome is frequently noted with fever, malaise, anorexia, and regional lymphadenopathy. The vesicles progress to pustules and crusted erosions. They heal over 2 to 3 weeks. Mucocutaneous involvement of the mouth and lips are the most common sites. *Herpetic whitlow* is a painful HSV infection of a distal finger seen primarily in children and adolescents. *Herpes gladiatorum* spreads via direct skin-to-skin contact in sports such as rugby and wrestling.

Management and Disposition

Oral antivirals in addition to analgesics and antipyretics are useful. To be most effective, antivirals should be started within 72 hours of eruption or during the typical prodrome. Immunocompromised patients may require admission and IV antivirals. Dermatology consultation is recommended for complicated presentations.

Pearls

1. Always wear protective gloves when examining open wounds; herpetic infections are an occupational hazard in the medical and dental professions.
2. Wrestlers (or participants in any skin-to-skin contact sport) with vesicles and ulcers may not participate in organized sports until completely healed. Consult the National Collegiate Athletic Association (NCAA) and the National Federation of State High School Associations (NFHS) return-to-play guidelines.
3. Recurrent, same-site infections on the fingers should alert the clinician to consider herpetic whitlow. These infections are often misdiagnosed as cellulitis, blistering dactylitis, or paronychia.



FIGURE 13.33 ■ Herpetic Whitlow. Painful, grouped, confluent vesicles and an erythematous base on the distal finger. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 13.34 ■ Herpetic Whitlow. Vesicles on an erythematous base. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.35 ■ Herpes Gladiatorum. Primary herpes gladiatorum in a wrestler. Note the varied stages of lesions, from vesicular to dried crusts. (Photo contributor: Lawrence B. Stack, MD.)

HERPES ZOSTER

Clinical Summary

Herpes zoster is a dermatomal, unilateral reactivation of the varicella zoster virus. All ages, including infants, can be affected. The eruption may occur anywhere but most commonly occurs on the face and trunk. Pruritus, pain, tenderness, and dysesthesias may present 4 to 5 days prior to an eruption composed of umbilicated, grouped vesicles on an erythematous, edematous base. The vesicles may become purulent or hemorrhagic. Occasionally, nerve involvement may occur without cutaneous involvement. Rare presentations involve multiple dermatomes or cross midline. Ophthalmic zoster (see related item) involves the nasociliary branch of the 5th cranial nerve and presents with vesicles on the nose and cornea (Hutchinson sign). Ramsay Hunt syndrome (see related item) is a herpes zoster infection of the geniculate ganglion with tinnitus, decreased hearing, facial palsy, and vesicles on the tympanic membrane, pinna, and ear canal.

Management and Disposition

Antiviral medication (acyclovir, famciclovir, or valacyclovir) hastens healing. If started within 72 hours of vesicle appearance, the duration, intensity, and associated pain are significantly decreased (benefit seen at least up to 7 days after vesicle appearance).

Admission for IV acyclovir is usually reserved for complicated cases involving multiple dermatomal distributions, involvement of the ophthalmic branch of the trigeminal nerve, disseminated disease, or immunocompromised patients. Herpes zoster keratitis requires emergent ophthalmologic consultation to avoid any potential scarring or vision loss.



FIGURE 13.36 ■ Herpes Zoster. Dermatomal distribution of vesicles and crusted erosions. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.37 ■ Herpes Zoster. Trigeminal nerve (V1) dermatomal distribution with upper eyelid involved; ophthalmologic evaluation is needed to rule out corneal involvement. (Photo Contributor: Kevin J. Knoop, MD, MS.)



FIGURE 13.38 ■ Herpes Zoster. Unilateral T4 distribution with grouped erythematous vesicles. (Photo Contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Avoid contact with nonimmune or immunocompromised contacts from the prodrome stage until complete reepithelialization of the lesions.
2. Herpes zoster during pregnancy confers no risk to a healthy mother or fetus (as opposed to primary varicella virus infection, which causes morbidity and mortality in both mother and fetus).
3. Postherpetic neuralgia (intense, chronic pain in the affected dermatome) affects 10% to 20% of patients and is more common with advancing age, family history, and immunosuppression.
4. Immunocompromised patients can have unusual presentations with diffuse cutaneous and visceral involvement.

SCABIES

Clinical Summary

Human scabies is caused by *Sarcoptes scabiei* var. *hominis*, a mite within the epidermal layers. Transmission occurs after skin contact with an infected individual or possibly from infested clothing and bedding. The female mite burrows into the skin and deposits two to three eggs daily. Fecal pellets (scybala) are deposited in the burrow and may be responsible for localized pruritus, often nocturnal. The pink white, slightly elevated burrows are typically seen in the web spaces of the hands and feet, penis, buttocks, scrotum, or extensor surfaces of the elbows and knees. Crusted, or Norwegian, scabies seen in immunosuppressed or debilitated patients (see HIV chapter) usually present with asymptomatic acral crusting, but can occur anywhere.

Management and Disposition

Topical 5% permethrin cream is commonly used. Apply from the neck to the toes overnight (8-12 hours) and then wash off. Repeat in 7 days. Bedding, towels, and clothing should be washed in hot water and dried on high heat (an alternative is to place items into a plastic bag for 10 days).



FIGURE 13.39 ■ Scabies. Note the scaling, erythema, and thickening of the skin at the finger bases. This describes chronic infection and potential to develop crusted scabies. (Photo contributor: David Effron, MD.)



FIGURE 13.40 ■ **Scabies.** Burrows and erosions in an infant. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.41 ■ **Scabies.** Extensive burrows and erosions. (Photo contributor: David Effron, MD.)

Pearls

1. Intimate contacts and all family members in the same household should be treated.
2. Patients often experience “postscabietic itch,” which can last for 2 to 4 weeks. This is not a new infestation but rather an immunologic reaction to the dead mite. Confirm completion of an appropriate treatment.

HIDRADENITIS SUPPURATIVA

Clinical Summary

Hidradenitis suppurativa (HS) most commonly affects obese, postpubertal individuals with a genetic predisposition. This unremitting disease involves intertriginous sites: axillae, inguinal folds, gluteal fold, perianal area, and inframammary folds. Lesions begin with erythematous nodules that become tender and fluctuant. The sterile abscess ruptures with a suppurative discharge, and multiple abscesses can eventuate into sinus tracts and/or fistulae. HS is primarily a noninfectious, inflammatory response to the hair follicle; however, secondary infections are common. Severe hypertrophic scar formation may be dramatic and disfiguring. In addition, chronic discharge is difficult to control and commonly malodorous. The differential diagnosis includes bacterial furunculosis, granuloma inguinale, mycetoma, cutaneous tuberculosis, and fistulas associated with inflammatory bowel disease.

Management and Disposition

HS should be identified and local or systemic infection ruled out. Topical and systemic antibiotics help improve lesions, especially if a secondary infection is suspected. Oral doxycycline and minocycline, with topical clindamycin, are common first-line agents. If possible, incision and drainage should be avoided as this can induce chronic sinus tract formation and worsen scarring. Referral to a dermatologist for chronic management and alternative treatments is indicated.



FIGURE 13.42 ■ Hidradenitis Suppurativa. Inflammatory abscesses and fistulae in the axilla. (Photo contributor: Stephanie Goldberg, MD.)



FIGURE 13.43 ■ Hidradenitis Suppurativa. Axillary scarring and resolving abscesses. (Photo contributor: Stephanie Goldberg, MD.)



FIGURE 13.44 ■ Hidradenitis Suppurativa. Chronic inguinal disease and scarring. (Photo contributor: Stephanie Goldberg, MD.)

Pearls

1. HS is not a primary infectious process but rather inflammatory. This often leads to delayed diagnosis, worse skin involvement, and permanent scarring.
2. Identifying presentations of HS and promptly referring them to dermatology can minimize long-term morbidity.
3. Perianal involvement should prompt gastroenterology referral to rule out inflammatory bowel disease.
4. Patients with long-term disease are at risk for developing squamous cell carcinomas in the affected areas.

DISSECTING CELLULITIS OF THE SCALP

Clinical Summary

Dissecting cellulitis of the scalp occurs predominately in young males. This

condition, along with acne conglobata, hidradenitis suppurativa, and pilonidal cysts, constitutes the “follicular occlusion tetrad.” Multiple, fluctuant abscesses on the scalp vertex and occiput are present. Sinus tracts form between the abscesses, and purulent, foul-smelling discharge may be present. Over time, disfiguring, scarring alopecia can involve the entire scalp.

Management and Disposition

Exclusion of a secondary infection is paramount. In the ED, incision and drainage of new, rapidly forming abscesses may be helpful; obtain cultures if this is attempted. Long-term anti-inflammatory antibiotics may prevent further abscesses. Urgently refer the patient to a dermatologist for further management.



FIGURE 13.45 ■ Dissecting Cellulitis of the Scalp. Many active sinus tracts and alopecia in a young Hispanic man. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. *The Color Atlas of Family Medicine*. 2nd ed. New York, NY: McGraw

Hill; 2013: Fig. 189-7.)

Pearls

1. Consider this diagnosis when presented with recurrent scalp abscesses and draining sinus tracts unresponsive to antibiotics.
2. Early diagnosis and urgent referral can prevent further scarring and alopecia.
3. Most patients are young to middle-aged males; the psychological impact is significant.



FIGURE 13.46 ■ Dissecting Cellulitis of the Scalp. Inactive but with disfiguring scars on the entire scalp. Early referral to a dermatologist may prevent this stage. (Photo contributor: J. Matthew Hardin, MD.)

LIVEDO RETICULARIS

Clinical Summary

Livedo reticularis is a reactive macular, reticulated (net-like) patch of nonpalpable cutaneous vasodilatation due to normal physiologic variation, vasospasm, vessel wall damage, or intraluminal pathology. It occurs in many systemic diseases including connective tissue disorders, vasculitis, polycythemia vera, cold agglutinins, hypercoagulability, thrombotic thrombocytopenic purpura, embolic disease, decompression sickness, and infections/sepsis. It has been associated with medications such as amantadine, quinine, and quinidine. *Physiologic livedo reticularis* is a response to cold temperatures and is a common finding in infants, children, and adults prone to acrocyanosis.

Management and Disposition

Management is dependent on identifying and treating the underlying disorder. Patients without an acute medical condition can be referred to dermatology.

Pearls

1. Physiologic livedo reticularis improves or disappears with warming, whereas secondary causes usually do not.
2. Patchy, nonsymmetrical distribution of livedo reticularis should elicit concern for more serious underlying diseases.



FIGURE 13.47 ■ Livedo Reticularis. A netlike, arborizing pattern defined by violaceous, erythematous streaks resembling lightning. The skin within the erythematous areas is normally pale. (Used with permission from Wolff K, Johnson RA, Saavedra AP. *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*. 7th ed. New York, NY: McGraw Hill; 2013: Fig. 14-42.)



FIGURE 13.48 ■ Livedo Reticularis. Reticulated (lacylike) branching erythema symmetrically distributed over the lower extremities (Photo contributor: James J. Nordlund, MD.)

CUTANEOUS SMALL-VESSEL VASCULITIS

Clinical Summary

Cutaneous small-vessel vasculitis (CSVV) represents the deposition of immune complexes in small blood vessels with subsequent vessel damage and blood extravasation (pathologically described as leukocytoclastic vasculitis). Nonblanching, purpuric macules and initially erythematous papules frequently coalesce into violaceous plaques (“palpable purpura”). The lower extremities and dependent areas of the back and buttocks are frequently involved. Pruritus can be significant or not present. Vesicles, ulcers, and necrosis may evolve within the purpuric lesions. Lesions appear over a few days and usually resolve with hyperpigmentation over 4 to 6 weeks. Symptoms may be minimal or include fever, arthralgias, myalgias, and malaise.

CSVV is associated with connective tissue diseases, malignancies,

medications (cephalosporins, penicillins, sulfonamides, minocycline, thiazides, allopurinol, phenytoin, NSAIDs, oral contraceptives, antithyroid agents), infections (group A β -hemolytic streptococci, *Mycobacterium leprae*, viral hepatitis, HIV), and idiopathic subtypes (Henoch-Schönlein purpura [HSP], acute hemorrhagic edema of childhood).

HSP is a unique form of CSVV with palpable purpura of the lower extremities and buttocks. Occasionally, the lesions may be found on the upper extremities, trunk, and face. A recent respiratory infection, arthralgias, abdominal pain, and hematuria are common. Renal vasculitis can occur in up to 40%, but chronic renal impairment occurs in only 1% to 3%. Adults tend to have necrotic lesions (rare in children), as well as a higher incidence of renal impairment.

Management and Disposition

Recognition that a diverse group of entities can trigger CSVV is the 1st step. Evaluation for systemic symptoms (fever or other signs of infection, hematuria, gastrointestinal bleeding, and neurologic symptoms) requiring admission and appropriate consultation should be undertaken. Most cases are self-limited and only require supportive care (rest, elevation of extremities, antihistamines, and analgesics). Systemic symptoms require admission and consideration of corticosteroids and other immunosuppressants. Dermatologic referral for mild CSVV cases is indicated, and if systemic symptoms are present, ED consultation may help expedite the diagnosis. HSP requires dermatologic consultation as well as appropriate specialty consultations.



FIGURE 13.49 ■ Leukocytoclastic Vasculitis. Lower extremities with erythematous papules and dorsal foot with erythematous plaques. If you were to run your finger across these lesions, they would be raised and not blanch. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.50 ■ Leukocytoclastic Vasculitis. Acute necrotic leukocytoclastic vasculitis. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.51 ■ Leukocytoclastic Vasculitis. Note the erythematous papules beginning to coalesce. These would not blanch with pressure. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.52 ■ Henoch-Schönlein Purpura. Note the classic acral distribution of HSP (both upper and lower extremities, raising concern for renal involvement). (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 13.53 ■ Urticarial Vasculitis. Urticarial plaques present for over 24 hours. (Photo contributor: J. Matthew Hardin, MD.)

Pearls

1. CSVV affects all ages and has equal incidence in males and females. The etiologies are often idiopathic, but scrutinize each patient for associated diseases and medicine exposures.
2. HSP associated renal involvement and lesions above the waist are more common in adults.

SWEET SYNDROME

Clinical Summary

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, is characterized by fever, peripheral neutrophilia, and a nonvasculitic neutrophilic cutaneous eruption. It can occur at any age but most commonly from 30 to 60 years old. Lesions can occur anywhere, but most frequently on the upper extremities, neck, and face; they are typically tender, well-demarcated erythematous plaques with an edematous periphery (pseudovesiculation). Some progress to ulcerations and hemorrhagic crusting. Variants have been isolated to the face (erysipelas-like) and the dorsal hands. The plaques generally cause a

burning pain and are nonpruritic. Sweet syndrome is associated with preceding upper respiratory infections, vaccinations, medications, malignancies, inflammatory bowel disease, autoimmune connective tissue diseases, and pregnancy.

Management and Disposition

The associated diseases need to be considered. A dermatologist should be consulted for diagnosis confirmation and further evaluation.

Pearls

1. Unless a specific infection is identified (*Streptococcus*, *Yersinia*, or *Staphylococcus*), antibiotics are not indicated.
2. Malignancies associated with Sweet syndrome account for 20% of cases; acute myeloid leukemia is the most common.
3. Although preceding infections can cause this rash, the lesions are not infectious.



FIGURE 13.54 ■ Sweet Syndrome. This young woman complained of sudden onset of fever and painful skin lesions. WBC = 22,000 with neutrophilia. This constellation of symptoms suggests Sweet syndrome and was confirmed by histology. There is a hint of peripheral vesiculation on some lesions. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.55 ■ Sweet Syndrome. Less obvious erythematous plaques on the foot. (Photo contributor: J.

Matthew Hardin, MD.)



FIGURE 13.56 ■ Sweet Syndrome. This is a variant called neutrophilic dermatosis of the dorsal hands. (Photo contributors: Samantha Yelley, DO; Miju Kurtzweil, DO; and Veronica Tucci, MD, JD.)

PYODERMA GANGRENOSUM

Clinical Summary

Pyoderma gangrenosum (PG) is an inflammatory condition of all ages but is most common among 20- to 50-year-old females. Lesions can be located anywhere (most commonly on the lower extremities) and begin as a papulopustule surrounded by erythema. This pustule erodes and expands to form a necrotic ulcer. Similar satellite pustules and ulcers form around the original lesion and can rapidly coalesce into a larger ulcer. The surrounding border is “rolled,” due to the convex elevation, and has a violaceous hue. The ulcers are exquisitely tender to movement and palpation. On the extremities, the ulcers can involve muscles and tendons. Ostomy sites are a common location and can make management very difficult.

Half of cases are idiopathic; the other half are associated with systemic processes such as vascular diseases, inflammatory diseases, malignancies, infections, necrobiosis lipoidica diabetorum, and trauma. Since the diagnosis is based on exam, dermatopathology, and exclusion of other causes, PG is difficult to confirm, and delayed treatment is common.



FIGURE 13.57 ■ Pyoderma Gangrenosum. Rolled and violaceous borders. This lesion can rapidly enlarge and become secondarily infected. (Photo contributor: J. Matthew Hardin, MD.)

Management and Disposition

Appropriate cultures should be obtained. Broad-spectrum antibiotics are indicated for secondary infections. Consult dermatology for biopsy, tissue culture, and initiation of immunosuppressant therapy.

Pearls

1. With the large number of ulcers seen in the ED, early diagnosis of PG is difficult and requires a high level of suspicion; think of this in chronic ulcers.
2. PG is often not diagnosed until late-stage ulcer formation and after multiple failed ulcer treatments and skin grafts. Without identification and control of the underlying disease process, any skin-directed treatment is unlikely to succeed.
3. Half of PG cases have associated diseases; each case needs a thorough specialty evaluation.



FIGURE 13.58 ■ Pyoderma Gangrenosum. Early ulcer formation at the site of an ileostomy dressing. (Photo contributor: Lawrence B. Stack, MD.)

ERYTHEMA NODOSUM

Clinical Summary

Erythema nodosum (EN) can present at any age but is most common in young, adult females. Most typical is bilateral, erythematous, subcutaneous, tender nodules on the pretibial and lateral lower extremities (usually spares the posterior calves). Rarely, the nodules can be found on the thighs, upper extremities, and face. Concomitant symptoms include lower extremity edema and arthralgias. Systemic symptoms can include fever, headache, and gastrointestinal complaints. Generally, the nodules resolve over days to weeks with flattening and a change in color to a blue-green (like a deep bruise). There is no ulceration, and the skin slowly returns to normal. Recurrence occurs in up to one-third of cases.



FIGURE 13.59 ■ Erythema Nodosum, Acute. Pretibial, erythematous, and subcutaneous nodules. (Photo contributor: Gianina Best, MD.)



FIGURE 13.60 ■ Erythema Nodosum, Resolving. Bruise-like appearance of the resolving phase. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.61 ■ Erythema Nodosum. Tender subcutaneous nodules with overlying erythematous and hyperpigmented macules, indicating a new crop of lesions. (Photo contributor: J. Matthew Hardin, MD.)

Multiple etiologies exist for EN, although over one-third of cases are idiopathic. Infectious causes include streptococcal, tuberculosis, *Yersinia*, *Salmonella*, *Shigella*, coccidioidomycosis, histoplasmosis, sporotrichosis, blastomycosis, and toxoplasmosis. EN has also been associated with pregnancy,

sarcoidosis, and inflammatory bowel disease. Oral contraceptives, sulfonamides, bromides, and iodides are known to be common causative agents, among many others.

Management and Disposition

With the many etiologies of EN, it is critical to exclude and treat an infectious, systemic, or medication cause. Supportive care with elevation of the extremity, rest, and NSAIDs is helpful. Recurrences do occur and should prompt a further workup for occult infection or persistent medication. Refer patients to a dermatologist for a confirmatory biopsy, additional laboratory testing, and definitive treatment.

Pearls

1. The patient's history is very helpful in determining possible etiologies. A complete medication, travel, and past medical history must be performed.
2. Systemic glucocorticoids can be considered, but only when the etiology is clearly known and infectious agents are excluded.
3. EN is considered a good prognostic sign in sarcoidosis and pregnancy-associated coccidioidomycosis.

SQUAMOUS CELL CARCINOMA

Clinical Summary

Squamous cell carcinoma (SCC) is the 2nd most common skin cancer. It is associated with a higher incidence in males, increased age, chronic sun exposure, immunosuppressive treatment, and chronic burns or scars. Initially, SCC presents with erythematous macules that develop into firm papules and plaques. Most are located on the sun-exposed sites of the head, neck, and upper extremities, but can occur anywhere.

Management and Disposition

After ensuring a secondary infection is not present, prompt outpatient

dermatologic referral is indicated.



FIGURE 13.62 ■ Squamous Cell Carcinoma. The lower lip is exposed to more sunlight and involved more frequently. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.63 ■ Squamous Cell Carcinoma. This nodule with central ulceration slowly developed over 1 year. (Photo contributor: J. Matthew Hardin, MD.)

Pearls

1. There is a higher risk of metastasis with SCC versus basal cell carcinoma (although still very low). As with basal cell carcinoma, metastatic potential is higher on the ears, periocular area, nose, and lips; do not miss the opportunity to refer patients with questionable lesions.
2. Any persistent nodule, plaque, or ulcer should be referred to dermatology for potential SCC.



FIGURE 13.64 ■ Squamous Cell Carcinoma. This nodule has a central keratogenous core. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.65 ■ Squamous Cell Carcinoma. Ulcerated nodule on the forehead. (Photo contributor: J. Matthew Hardin, MD.)

BASAL CELL CARCINOMA

Clinical Summary

Basal cell carcinoma (BCC) is the most common nonmelanoma skin cancer. BCC can present anywhere but is most common on sun-exposed areas. The typical lesion begins as a pearly papule with telangiectasias (nodular BCC). The lesion may ulcerate and bleed. Other forms of BCC include superficial BCC (pink, scaly plaque with pearly border), pigmented BCC (appears as a nodular or superficial BCC with dark brown to black center), and morpheaform BCC (appears as a rapidly expanding scar).

Management and Disposition

After ensuring a secondary infection is not present, prompt outpatient dermatologic referral is indicated.



FIGURE 13.66 ■ Basal Cell Carcinoma. Nodular basal cell carcinoma consists of a firm, centrally ulcerated (rodent ulcer) nodule with a raised, pearly, telangiectatic border. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.67 ■ Pigmented Basal Cell Carcinoma. Translucent, brownish-black, flat papule, easily confused with melanoma. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

Pearls

1. The metastatic potential of BCC is very low (0.1%) but higher on the ears, periocular area, nose, and lips. Do not miss the opportunity to refer patients with questionable lesions.
2. BCC occurs in darker skinned persons (a common misperception).
3. Early identification and treatment of basal cell carcinomas, especially around the eyes, ears, and nose, will reduce potential morbidities.



FIGURE 13.68 ■ Basal Cell Carcinoma. A superficial basal cell carcinoma is frequently disregarded. Note the flat, erythematous, scaly plaque with its elevated, irregular border. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.69 ■ Basal Cell Carcinoma. This chronic, erythematous papule and crusted ulcer appeared 2 years prior to presentation. (Photo contributor: J. Matthew Hardin, MD.)

MELANOMA

Clinical Summary

Melanoma is a potentially fatal cutaneous tumor derived from epidermal melanocytes. Any age can be affected; peak incidence is in 20- to 45-year-old patients (much younger than BCC or SCC). The most significant risk factor is a primary relative with melanoma. Evaluation of any pigmented lesion should include the ABCDE rule (A for asymmetry, B for irregular borders, C for color variegation, D for diameter > 6 mm, and E for elevation and evolving). Any lesion with these characteristics is considered suspicious for melanoma.



FIGURE 13.70 ■ Melanoma. This lesion demonstrates asymmetry, color variegation, and a diameter greater than 6 mm. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.71 ■ Nodular Melanoma. This has progressed to an exophytic tumor, which was deeply invasive histopathologically. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

Management and Disposition

Prompt outpatient dermatologic referral is indicated. Simply acknowledging suspicious lesions seen during emergency care may encourage earlier follow-up.

Pearls

1. The palms, soles, and nail areas are the most common sites in dark-skinned individuals.
2. Melanoma can occur in sites not exposed to the sun (genitalia/buttocks/scalp).
3. Any growing pigmented or nonpigmented lesion should be referred to dermatology.
4. Most patients will not have new moles after 35 years old. A new mole in this setting should be referred.



FIGURE 13.72 ■ Melanoma. Slow-growing, pigmented plaque on the arch of the foot. (Photo contributor: J. Matthew Hardin, MD.)

URTICARIA AND DERMATOGRAPHISM

Clinical Summary

Acute urticaria develops over days to weeks and presents with transient wheals. Generally, acute urticaria resolves within 6 weeks, whereas chronic urticaria lasts longer. Common triggers include medications (penicillin, aspirin, and NSAIDs), foods (chocolate, shellfish, nuts, eggs, milk, and others) infections (streptococcal, hepatitis B and C, mononucleosis, and helminths), and physical

factors (exercise, pressure, cold, vibratory, and solar induced).

Dermatographism is the production of linear urticarial lesions and surrounding erythematous flare after stroking the skin. Simple dermatographism, seen in up to 5% of the population, is considered an exaggerated physiologic response to friction. In contrast, symptomatic dermatographism presents without a previous history. Wheals are typically seen with scratching and friction from tight clothing. Children and young adults are commonly affected, and fortunately, no associated systemic disease, autoimmunity, or food allergy exists. Symptomatic dermatographism can persist for several years.



FIGURE 13.73 ■ Acute Urticaria. Classic raised plaques on the lower extremity. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.74 ■ Acute Urticaria. Preschool child with annular, raised pruritic lesions with central clearing and tense edema. The lesions disappeared approximately 5 minutes after presentation. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 13.75 ■ Acute Urticaria and Anaphylaxis. Raised facial plaques in a patient severely allergic to bee stings. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 13.76 ■ Cold Urticaria. Plaque formation after placement of an ice cube on the skin confirms cold urticaria. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.77 ■ Dermatographism. Linear wheal with surrounding erythematous flare after scratching and stroking. (Photo contributor: J. Matthew Hardin, MD.)

Management and Disposition

Triggers of urticaria should be investigated and, if present, stopped. H₁ and H₂ blockers usually help. Systemic steroids and epinephrine are used for severe reactions and anaphylaxis.

Pearls

1. More than 50% of chronic urticaria is idiopathic; consider an infection, medication, or physical factor first.
2. Wheals present for over 24 hours and in the same location are concerning for urticarial vasculitis (see related item) and should prompt dermatology consultation. Draw a circle around the lesions and have the patient monitor changes.

Clinical Summary

Allergic contact dermatitis occurs after previously sensitized skin is rechallenged with the same allergen and represents a delayed-type hypersensitivity reaction. Papules and vesicles first develop; they can become a generalized morbilliform eruption (autosensitization). Pruritus is a dominant feature. The most common causes are nickel, toxicodendrons (poison ivy, poison oak, and poison sumac), neomycin, fragrances, balsam of Peru (common in perfumes), formaldehyde, bacitracin, and rubber compounds.

Management and Disposition

Identification of the causative agent and prevention of further contact is critical. Supportive care is given with antihistamines and topical corticosteroids. Systemic corticosteroids may be needed for generalized eruptions. Refer patients to dermatology for further evaluation and possible patch testing.



FIGURE 13.78 ■ Contact Dermatitis. Erythematous eruption in a waist-band distribution (elastic allergy from underwear). (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.79 ■ Contact Dermatitis. The erythematous, edematous base of the eruption corresponds to the posterior watch surface. Superimposed on the erythematous base are multiple vesicles with exudate and crust. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.80 ■ Contact Dermatitis. Allergy to tattoo pigment. Note erythema, scale, and erosions contained within the tattoo image. (Photo contributor: J. Matthew Hardin, MD.)

Pearls

1. Obtaining a complete history of all exposures is critical; pay attention to personal hygiene products.
2. Toxicodendron allergic contact dermatitis (poison ivy, poison sumac, poison oak) requires a minimum 3-week taper of oral prednisone; a shorter course allows rash reappearance.

ATOPIC DERMATITIS

Clinical Summary

Atopic dermatitis presents in three overlapping stages: infantile, childhood, and adult. *Infantile* begins after 2 months of age and is symmetrically distributed on the cheeks, scalp, neck, forehead, and extensor surfaces of the extremities. The lesions begin as erythema or papules, but with persistent itching and rubbing, they become thin plaques, exudative, and crusted. *Childhood* atopic dermatitis presents with flexural involvement. Other areas frequently involved are the face, neck, and trunk. The scratching induces plaque lichenification and potential for secondary infection. *Adult* atopic dermatitis is less specific but can present with a childhood-like distribution, papular lesions that coalesce into plaques, and chronic hand dermatitis. Uncontrolled atopic dermatitis can become a generalized exfoliative erythroderma. Differential diagnoses include seborrheic dermatitis, psoriasis, irritant or allergic contact dermatitis, nummular eczema, and scabies.

Management and Disposition

If a severe flare, suprainfection, punched-out lesions (eczema herpeticum), or a generalized exfoliative erythroderma is present, an ED dermatologic consultation is indicated. Mild cases can be sent home with referral. Patients (caregivers) should avoid soaps, detergents, or any personal products with fragrances. After bathing, pat dry the skin and smear a thin film of petrolatum or mild corticosteroid over the affected areas. A short course of systemic steroids may be indicated. Ensure follow-up with dermatologist.

Pearls

1. Atopic dermatitis is often called the “itch that rashes” since pruritus precedes clinical disease.
2. If dispensing corticosteroids, use appropriate classes for the affected site and patient age.
3. Frequent relapses are common and require an astute clinician to differentiate associated complications (eg, HSV infection or developing cellulitis).



FIGURE 13.81 ■ Atopic Dermatitis. A typical localization in children is the region around the mouth, with lichenification, fissuring, and crusting. (Used with permission from Wolff K, Johnson RA, Saavedra AP. *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*. 7th ed. New York, NY: McGraw Hill; 2013: Fig. 2-14.)



FIGURE 13.82 ■ Atopic Dermatitis. (A) One of the hallmarks is lichenification in the flexural regions. Note the thickening of the skin with exaggerated skin lines and erosions. (B) Atopic dermatitis in black child. Pruritic follicular papules on posterior leg. Follicular eczema pattern is more common in African and Asian children. (Used with permission from Wolff K, Johnson RA, Saavedra AP. *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*. 7th ed. New York, NY: McGraw Hill; 2013: Fig. 2-15 A and B.)



FIGURE 13.83 ■ Adult Atopic Dermatitis. Lichenified plaques, erosions, and fissures are characteristic. (Photo contributor: James J. Nordlund, MD.)

NUMMULAR/XEROTIC ECZEMA

Clinical Summary

Nummular eczema is characterized by an erythematous, edematous, vesicular, and crusted plaque. These commonly present on the upper and lower extremities. The lesions enlarge by forming satellite papulovesicles that coalesce with the original lesion. Pruritus is the dominant symptom.

Xerotic eczema (also called winter itch, eczema craquele, and asteatotic eczema) presents on the anterior shins, extensor arms, and flanks. The lesions are erythematous patches with fine, cracked fissures and adherent scaling. The edema and exudate present in nummular eczema is absent. Pruritus can be severe. This is a common finding in the winter and in the elderly.

Management and Disposition

Treatment consists of mid- to high-potency topical steroid. Prevention of secondary infection is important as patients frequently cannot resist scratching. Difficult to control, recurrent, and chronic cases should be referred to a dermatologist for advanced treatment. Xerotic eczema is treated with topical emollients (petrolatum), three to four applications per day. Topical steroids may be required for areas with inflammation.



FIGURE 13.84 ■ Eczema. Nummular eczema of the wrist. Note the satellite lesions on the periphery. (Photo contributor: J. Matthew Hardin, MD.)

Pearls

1. Nummular eczema should be considered with lesions unresponsive to antibiotic and antifungal medications.
2. Both entities are associated with significant pruritus and secondary infections, especially in the young and elderly.



FIGURE 13.85 ■ Eczema. Nummular eczema on the extensor surface of the forearms and elbows. Thickened, scaly lesions resemble psoriatic plaques. A biopsy was performed to confirm the diagnosis. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. *The Color Atlas of Family Medicine*. 2nd ed. New York, NY: McGraw Hill; 2013: Fig. 148-6.)

DYSHIDROTIC ECZEMA

Clinical Summary

Dyshidrotic eczema (also called pompholyx or acute vesiculobullous hand eczema) is the abrupt appearance of deep-seated, 1- to 2-mm vesicles on the sides of the fingers, palms, and soles. The vesicles are extremely pruritic, may coalesce into larger bullae, and may rupture to become dry or fissured. The outbreak usually resolves over a few weeks unless secondary infection develops. The differential includes bullous tinea, id reaction, scabies infestation, or allergic contact dermatitis.

Management and Disposition

Treatment includes a high-potency topical steroid and prevention of secondary infection. Refer to a dermatologist for long-term treatment; this is often chronic with the potential for significant disability.

Pearls

1. The initial lesions resemble tapioca pudding.
2. These lesions wax and wane and are often exacerbated by stressful life events.



FIGURE 13.86 ■ Dyshidrotic Eczema. In most cases dyshidrotic eczematous dermatitis starts with tapioca-like vesicles on the lateral aspects of the fingers. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. *The Color Atlas of Family Medicine*. 2nd ed. New York, NY: McGraw Hill; 2013: Fig. 147-7.)



FIGURE 13.87 ■ Dyshidrotic Eczema. The vesicles show confluence and spread to the palm, but also to the wrist and dorsal aspect of the hand when the eruption progresses. (Used with permission from Wolff K, Johnson RA, Suurmond D. *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*. 5th ed. New York, NY: McGraw Hill; 2005: 45.)

ID REACTION (DISSEMINATED ECZEMA)

Clinical Summary

Id reactions are seen in response to a variety of disorders, including fungal infections (tinea capitis and tinea pedis), scabies infestation, pediculosis capitis, molluscum contagiosum, bacterial and mycobacterial infections, and arthropod bites. The rash appears days to weeks after the instigating rash and consists of erythematous papules (sometimes crusted at the apices) as well as eczematous patches and plaques. The rash can be local to the instigating lesions/rash, distant, or generalized. The id reaction usually presents on the extremities, commonly on the sides of fingers, but may occur on the face and trunk. Pruritus is intense. The id reaction will not demonstrate infectious organisms and may not respond to topical steroids.

Management and Disposition

Recognition and treatment of the initial infection or infestation is curative. Refer to a dermatologist for follow-up to confirm diagnosis and resolution.

Pearls

1. Repeated ED evaluation for fungal infection or eczematous rash should prompt further investigation for a distant, untreated, or occult fungal infection.
2. Id reactions are intensely pruritic; make sure secondary bacterial infections do not develop from excoriations.
3. Recurrences are common, especially if the primary source is not treated or treated adequately.

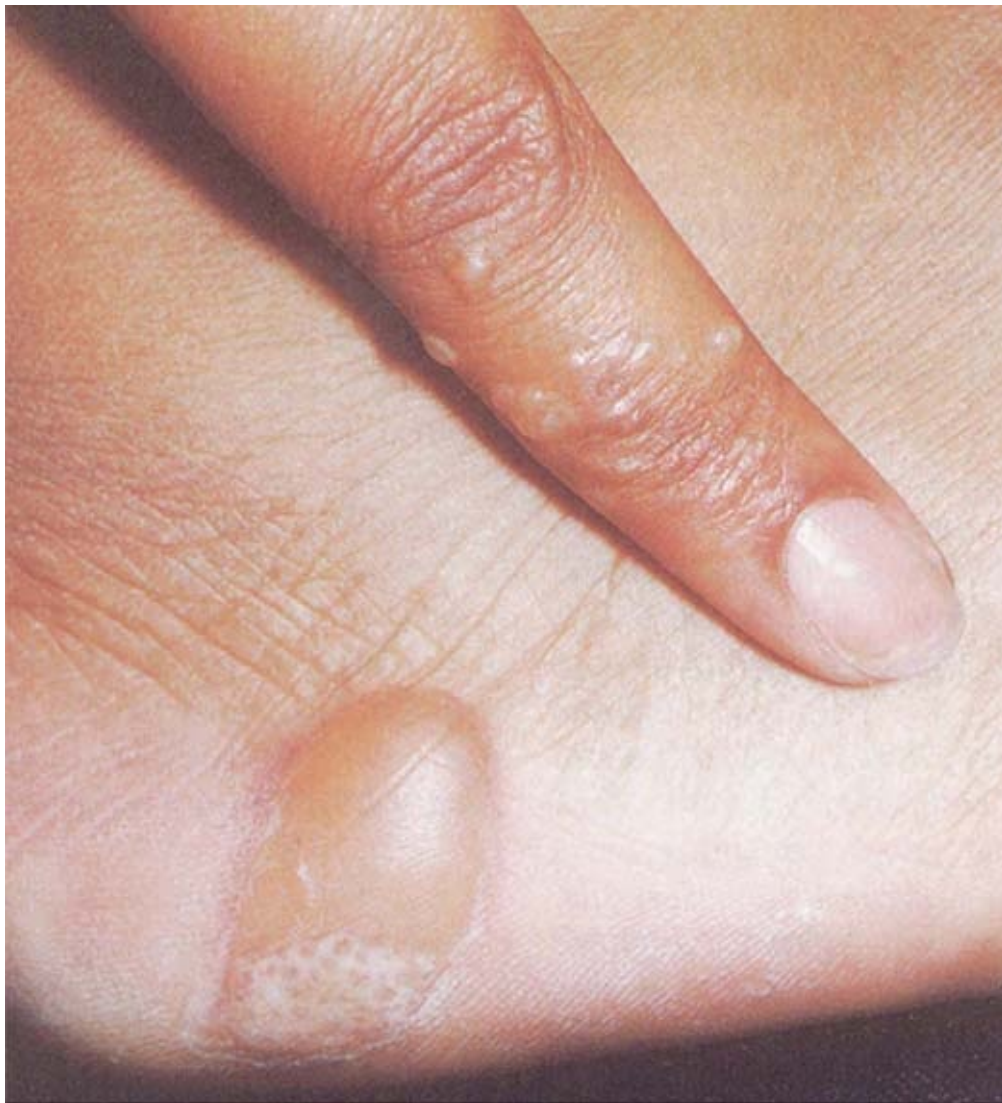


FIGURE 13.88 ■ Id Reaction. Bullous tinea pedis causing id reaction on the fingers. (Used with

permission from Wolff K, Johnson RA, Suurmond D. *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*. 5th ed. New York, NY: McGraw Hill; 2005: 48.)



FIGURE 13.89 ■ Id Reaction. Reaction to tinea pedis. Erythematous, partially dried up, pruritic vesicles on the foot. (Used with permission from Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, Wolff K. *Fitzpatrick's Dermatology in General Medicine*. 8th ed. New York, NY: McGraw Hill; 2012: Fig. 16-7.)

LIP LICKER'S DERMATITIS

Clinical Summary

Lip licker's dermatitis is caused by repeated exposure of the vermillion border and the cutaneous lips to saliva. Children and young adults are more commonly affected, but older adults, including the elderly, can be afflicted. Irritants in topical lip preparations can initiate this process or can worsen a presentation. Winter is the most common time to see lip licker's dermatitis due to the constant exposure to cold air and low humidity, but presentations occur throughout the year.

The dermatitis typically starts with red papules that slowly progress into an erythematous, thin plaque. As the cycle of licking continues, the area becomes confluent circumferentially around the vermillion border and the cutaneous lips. The skin begins to break down with fissuring and scaling, and the area can become very painful. The rash does not extend beyond the tongue's reach on the

cutaneous lips.

Management and Disposition

Treatment is focused on identifying and preventing repetitive lip licking. Many children are not aware they are contributing, and care should be taken to not blame them for this predicament (especially younger children). Use of 100% petrolatum (Vaseline) applied all day (any time the area becomes dry or the patient desires to lick) will help. Stop the use of any other topical products. The addition of 1% hydrocortisone ointment may hasten the resolution.

Pearls

1. Never approach a young child blaming them for their condition; it is best to use indirect questioning to make them aware of the cause (“Do you think you ever lick your lips?”).
2. The use of over-the-counter lip preparations or flavored lip products may cause or exacerbate the condition.
3. Lip licker’s dermatitis never ends until the lip licking behavior ends!



FIGURE 13.90 ■ Lip Licker's Dermatitis. Circumoral dryness and pain. (Photo contributor: Aubrey Mowery, MSN, MPH, CPNP.)

DERMATOPHYTE INFECTIONS

Clinical Summary

Tinea corporis includes all dermatophyte infections excluding the scalp, face, hands, feet, and groin. The dermatophytosis is pruritic and consists of a well-circumscribed scaly plaque with a slightly elevated border and central clearing. This annular configuration is most commonly found on the trunk and neck. Skin scrapings examined with a KOH preparation demonstrate hyphae.



FIGURE 13.91 ■ Tinea Corporis, Ringworm. A well-defined, annular, pruritic plaque with a raised, scaly border and central clearing. KOH preparation is positive. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

Tinea faciale (dermatophyte infection of the facial skin) commonly appears as a well-circumscribed scaling and erythematous patch. *Tinea manuum* (hands) presents with long-term scaling of the palms. *Tinea cruris*, or “jock itch,” is a pruritic dermatophytosis of the intertriginous areas, usually, but not always, sparing the penis and scrotum. The scaly, erythematous plaque spreads peripherally with well-defined borders. *Tinea pedis*, or “athlete’s foot,” consists of erythema and scaling of the sole and interdigital spaces, frequently with maceration, vesiculation, and fissure formation. The toenails may also be affected (*tinea unguium*). *Tinea capitis* (scalp) presents as a pruritic, erythematous, scaly plaque. This may develop into a delayed-type hypersensitivity reaction, where the initial erythematous, scaly plaque becomes boggy with inflamed, purulent nodules and plaques (*kerion*). The hair follicle is frequently destroyed by the inflammatory process in a kerion, leading to a scarring alopecia.



FIGURE 13.92 ■ Tinea Faciale. Note the sharply margined, annular plaques with central clearing. KOH preparation is positive. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.93 ■ Tinea Manuum and Tinea Pedis. The involvement of both the hands and feet are common. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.94 ■ Tinea Cruris. Erythematous plaque with accentuated and well-defined border. The scale may not be appreciated in this anatomic site. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.95 ■ Tinea Pedis. The interdigital spaces are characteristically involved. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.96 ■ Tinea Capitis. Multiple, well-defined, scaly plaques on the occiput. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.97 ■ Kerion. Occipital boggy swelling with hair loss consistent with kerion. (Photo contributor: Anne W. Lucky, MD.)

Management and Disposition

Systemic antifungals are required to treat *tinea capitis* and *tinea unguium* infections. Due to the long-term treatment requirement and potential side effects, referral to a dermatologist is recommended. Topical antifungal medications are used for *tinea corporis*, *tinea faciale*, *tinea cruris*, and *tinea pedis* infections.

Rarely, systemic antifungals may be required.

Pearls

1. The scale is usually located at the leading edge of erythema and provides the best yield for positive scrapings seen on KOH examination (see related item for slide preparation).
2. Macerated areas may become secondarily infected by bacteria. Discuss the importance of good wound care.
3. When using topical antifungals, it is important to treat for 1 to 2 weeks beyond the point of clinical cure to ensure success.

TINEA (PITYRIASIS) VERSICOLOR

Clinical Summary

Tinea versicolor, or pityriasis versicolor, is a chronic, superficial fungal infection that involves the trunk and extremities with rare facial involvement. The fungus is part of normal skin flora. Finely scaling brown macules are present in fair-skinned patients, whereas scaly hypopigmented macules are often noted in dark-skinned patients.

Management and Disposition

Treatment consists of short applications of selenium sulfide lotion, topical antifungal creams, or topical ketoconazole. Resistant cases require referral and consideration of oral antifungal.

Pearls

1. Tinea versicolor is more common in adolescents and young adults.
2. Clinically active areas or areas colonized with the fungus may be identified by orange fluorescence noted on the Wood lamp examination.
3. Normal pigmentation may take months to return.



FIGURE 13.98 ■ Tinea Versicolor. Multiple, small-to-medium-sized, well-demarcated hypopigmented macules on the back of a tanned individual with white skin. (Used with permission from Wolff K, Johnson RA, Saavedra AP. *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*. 7th ed. New York, NY: McGraw Hill; 2013: Fig. 26-19.)



FIGURE 13.99 ■ Tinea Versicolor. An example of hypopigmented areas on dark skin. (Photo contributor: James J. Nordlund, MD.)

ONYCHOMYCOSIS

Clinical Summary

Onychomycosis is an invasion of the nails by any fungus. Four clinical subtypes are described. *Distal subungual* presents as discolorations of the free edge of the nail with hyperkeratosis, leading to subungual accumulation of friable keratinaceous debris. *White superficial* consists of sharply outlined white areas on the nail plate, which leave the surface friable. *Proximal subungual* presents as discolorations that start proximally at the nail fold. *Candidal onychomycosis* encompasses the entire nail plate, leaving the surface rough and friable.

Management and Disposition

Oral antifungals are required to eradicate the fungus. Refer to a dermatologist or primary care physician.

Pearls

1. All that causes the nail plate to separate from the nail bed is not necessarily fungus. Psoriasis, lichen planus, and various other nail dystrophies, such as distal onycholysis (caused by excessive water exposure or drugs) must be differentiated from this fungal infection.
2. Treatment requires proper monitoring and long-term follow-up.



FIGURE 13.100 ■ **Onychomycosis.** Invasion of the nail bed by fungus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.101 ■ **Onychomycosis.** Note that multiple nail beds have been invaded by the fungus, leading

to chronic hyperkeratosis and subungual accumulation of friable keratinaceous debris. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.102 ■ Onychomycosis. Multiple nail beds with dystrophy and yellow subungual debris. (Photo contributor: J. Matthew Hardin, MD.)

INTERTRIGO

Clinical Summary

Intertrigo is a dermatitis occurring on opposed surfaces of skin, such as the creases of the neck, folds of the groin and armpit, or a panniculus. It is characterized by a tender, red patch or plaque with a moist, macerated surface. The juxtaposed skin surfaces create a chronic friction, and this can easily become suprainfected with candida, fungal, or bacterial infections.

Management and Disposition

Local care, empiric topical antifungal treatment, and good personal hygiene are recommended.

Pearls

1. The nature of the intertriginous areas makes them a high risk for secondary infection.
2. Potent topical corticosteroids should be avoided because of the risk for striae and atrophy.



FIGURE 13.103 ■ Intertrigo of the Panniculus. In this dramatic case, the weight of the pannus creates constant friction on the abdominal wall. This leads to erythema and tissue breakdown. This patient also had fever, suggesting secondary infection. (Photo contributor: Lawrence B. Stack, MD.)

HOT TUB FOLLICULITIS/PSEUDOMONAS HOT-FOOT SYNDROME

Clinical Summary

Hot tub folliculitis is a pruritic, pustular eruption confined to the hair follicle and

secondary to a cutaneous infection with *P aeruginosa*. Patients have a history of hot tub, whirlpool, or swimming pool exposure within 24 to 72 hours of eruption. The lesions have a predilection for areas covered with bathing suits. Headache, sore throat, earache, and fever may accompany the pustules (but do not necessarily indicate systemic disease).

Pseudomonas hot-foot syndrome is related to hot tub folliculitis; patients report wading in a swimming pool (later discovered to have elevated *P aeruginosa* concentrations). The weight-bearing aspects of the soles have multiple 1- to 2-cm erythematous nodules. Unlike hot tub folliculitis, these lesions are exquisitely painful and not associated with other symptoms. Like hot tub folliculitis, the lesions resolve spontaneously without treatment.

Management and Disposition

The folliculitis usually involutes in 1 to 2 weeks without treatment; however, oral ciprofloxacin or topical gentamicin may decrease recovery time. In addition, the implicated source of exposure must be decontaminated to avoid reexposure. Patients with immunosuppression, widespread lesions, or concern for systemic involvement should be treated with oral ciprofloxacin.

Pearls

1. Ensure the patient is aware of the cause and insist on decontamination of the water source.
2. Hot tub folliculitis may also result from contact with depilatory agents.



FIGURE 13.104 ■ Hot Tub Folliculitis. Note the pustules localized to the hair follicles of the trunk and proximal extremity. (Photo contributor: Jeffrey S. Gibson, MD.)



FIGURE 13.105 ■ Hot Tub Folliculitis. Folliculitis from *Pseudomonas aeruginosa* in a hot tub. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. *The Color Atlas of Family Medicine*. 2nd ed. New York, NY: McGraw Hill; 2013: Fig. 117-1.)

PYOGENIC GRANULOMA

Clinical Summary

Pyogenic granuloma presents as an eruptive, friable papule over weeks. They are frequently located on the extremities, face, or at recently traumatized sites. Children are commonly affected, but they can occur at any age. The lesion will bleed with little trauma and can be difficult to stop. If the papule is not completely removed, it will likely recur at the same site. Pregnant women have a higher incidence of pyogenic granulomas (common on the gingiva).

Management and Disposition

Most ED presentations of pyogenic granuloma will be due to prolonged, often brisk, bleeding. Silver nitrate applied to the papule base is usually effective (avoid on the face to prevent permanent staining). Refer patients to a dermatologist for possible biopsy and further treatment.

Pearls

1. An association with isotretinoin, indinavir, epidermal growth factor receptor inhibitors, and capecitabine has been described.
2. Approximately one-third of these benign lesions follow some form of minor trauma.
3. Refer patients for biopsy and histology to exclude other vascular tumors or melanoma.



FIGURE 13.106 ■ Pyogenic Granuloma. Note the violaceous color and multilobulated nodule. The hyperpigmented patches on either side are secondary to a bandage. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.107 ■ Pyogenic Granuloma. A moist, violaceous, vascular nodule formed at the site of an injury. Note that the nodule is demarcated by a thin rim of friable epidermis. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.108 ■ Pyogenic Granuloma. Friable papule with frequent bleeding. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.109 ■ Pyogenic Granuloma. Note the violaceous color and multilobulated nodule. (Photo contributor: J. Matthew Hardin, MD.)

SEBORRHEIC DERMATITIS

Clinical Summary

Seborrheic dermatitis represents a spectrum ranging from localized lesions to generalized exfoliative erythroderma. All ages are affected. Lesions have an erythematous base with a yellow, greasy scale. The scalp, external auditory canal, postauricular ear, eyebrows, eyelids, and face (especially the nasolabial folds) are common locations. Infants can have lesions at the above sites, but focal and confluent lesions are most common on the scalp and called “cradle cap.” Atypical presentations can occur in the axillae, umbilicus, chest, and inguinal folds.

Management and Disposition

Seborrheic dermatitis is a lifelong disease and has no cure; management is directed at control. Localized cases are effectively treated with topical steroids. Scalp involvement can be treated with selenium sulfide, ketoconazole, or zinc pyrithione shampoos. Parents of affected infants should be reassured that infantile seborrheic dermatitis is self-limited and can be controlled. Refer to a dermatologist for confirmation of diagnosis and chronic care.



FIGURE 13.110 ■ Seborrheic Dermatitis. Erythema and yellow-orange scales and crust on the scalp of an infant (“cradle cap”). Eczematous lesions are also present on the arms and trunk. (Used with permission from Wolff K, Johnson RA, Suurmond D. *Fitzpatrick’s Color Atlas & Synopsis of Clinical Dermatology*. 5th ed. New York, NY: McGraw Hill; 2005: 51.)



FIGURE 13.111 ■ **Seborrheic Dermatitis.** Intense erythema on the forehead and cheeks. (Photo contributor: David Effron, MD.)



FIGURE 13.112 ■ Seborrheic Dermatitis. Typical erythematous, scaling patches on the pinna and conchal bowl. Note also similar lesions on the anterior and posterior hairlines. (Photo contributor: J. Matthew Hardin, MD.)

Pearls

1. In an adult, new-onset, severe, or prolonged seborrheic dermatitis may indicate a new HIV infection or immunosuppression.
2. Although there is no cure, reassure patients the rash can be well controlled with topical medications.
3. In infants, seborrheic dermatitis can appear indistinguishable from Langerhans cell histiocytosis. Always have a clear discharge plan to follow up with a pediatrician or dermatologist.

PSORIASIS

Clinical Summary

Psoriasis has many forms. The most common is *chronic plaque* psoriasis with stable, symmetric lesions on the trunk and extremities, especially the elbows and

knees. Lesions are well-defined, erythematous plaques with silvery scales. *Inverse* psoriasis represents a form that involves the intertriginous areas, and due to the moist environment, the silvery scale is absent. *Guttate* psoriasis, common in children and young adults, presents with an abrupt eruption of 2- to 5-mm erythematous scaly papules on the trunk and extremities. A preceding respiratory infection, usually streptococcal pharyngitis, can be a precipitant of guttate psoriasis. *Pustular* forms of psoriasis can present as localized (nail bed, finger, palms, or soles) or generalized. It is characterized by erythema and “lakes of pus.” Triggers for pustular psoriasis include steroid withdrawal (as in patients with chronic obstructive pulmonary disease [COPD] and asthma exacerbations), pregnancy, infections, and topical irritants.



FIGURE 13.113 ■ Psoriasis. Annular, well-defined plaque on the shin. (Photo contributor: J. Matthew Hardin, MD.)

Management and Disposition

ED management should ensure there is no infectious etiology or systemic symptoms. Localized psoriasis typically responds to topical glucocorticoids, although the chronicity and variety of other management options, including phototherapy, should prompt referral to dermatology. Pustular forms may be challenging and require admission. Guttate psoriasis may respond to antistreptococcal antibiotics. Obtain emergent consultation with a dermatologist for patients with generalized presentations (erythrodermic patients) and referrals for localized disease.



FIGURE 13.114 ■ Psoriasis. Classic silvery scale associated with longstanding lesions. (Photo contributor: J. Matthew Hardin, MD.)

Pearls

1. The increase in biologic medications and immunosuppressants used for psoriasis can make serious infections a major concern.
2. Medication-induced psoriasis is associated with β -blockers, lithium, interferon, and antimalarials.
3. Patients with psoriasis have a higher incidence of coronary artery disease, obesity, tobacco use, and alcoholism.



FIGURE 13.115 ■ Psoriasis. Note the erythematous plaques with diffuse fissuring in this case of palmar psoriasis. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.116 ■ Psoriasis. Erythematous plaques on the upper arm and hand. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.117 ■ Psoriasis. Erythematous plaques on the forehead and scaling in the scalp. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.118 ■ **Plaque Psoriasis.** (Photo contributor: Lawrence B. Stack, MD.)

PITYRIASIS ROSEA

Clinical Summary

The 1st sign of pityriasis rosea (PR) is usually a well-demarcated, salmon-

colored macule that evolves into a larger patch (1-4 cm) with peripheral scaling (“herald patch”). Over 1 to 2 weeks, generalized, bilateral, and symmetric macules and plaques appear along skin cleavage lines (termed “Christmas tree” pattern). The macules have a peripheral collarette of fine scaling. Most will have severe itching associated with the generalized eruption. The lesions slowly resolve over 6 to 8 weeks. Atypical presentations in children include inverse PR (presentation on the face, axillae, and/or inguinal areas) and papular PR (peripheral scaling papules with central, hyperpigmented plaques). A viral etiology is postulated due to seasonal variation and case clustering.



FIGURE 13.119 ■ Pityriasis Rosea Herald Patch. The herald patch of PR, a well-demarcated salmon-colored macule with scales, frequently precedes the generalized phase by 1 to 2 weeks. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

PR is both benign and self-limited. Pruritus can be treated with oral antihistamines, topical steroids, and oatmeal baths.

Pearls

1. Patients often will not describe the herald patch unless specifically asked.
2. In patients with risk factors for syphilis and HIV, appropriate screening tests

should be performed.

3. Patients should be warned of the possible extended course of PR and given appropriate antihistamines and follow-up.
4. Atypical presentations are seen in dark-skinned individuals and children; often, lesions present in the axilla and groin.



FIGURE 13.120 ■ Pityriasis Rosea. An exanthematous, papulosquamous eruption, with the long axis of the oval papules following the lines of cleavage in a Christmas tree–like eruption. (Photo contributor: David Effron, MD.)

STASIS DERMATITIS

Clinical Summary

Stasis dermatitis results from venous insufficiency, is characteristically distributed on the distal tibia above the medial malleolus, and appears early with erythematous patches. These can progress to scaling as well as eczematous and weeping plaques. Patients will often have light brown pigmentation distributed

on the lower third of the extremity due to microvasculature blood extravasation (hemosiderin deposition secondary to increased superficial capillary pressure). Varicose veins are usually present, although they are often difficult to visualize in obese patients. Patients with heart failure, cirrhosis, nephrotic syndrome, or lower extremity trauma are at increased risk due to a chronic edematous state.

Management and Disposition

Referral to a primary care physician should be initiated to address the underlying etiology (eg, venous valvular insufficiency, thromboembolic disease, chronic edematous state), and dermatology referral should be made to ensure other diagnoses are not playing a role. Daily elevation and compression hose use should be encouraged. Emollients and mid-potency topical steroids help decrease the pruritus and promote healing.

Pearls

1. Differentiation of stasis dermatitis and early cellulitis can be difficult. Clinical history and deviation from previous presentations may be helpful. Close follow-up for reevaluation should be obtained.
2. Due to the chronic and repetitive nature of stasis dermatitis, patients use many over-the-counter topical products. These may contribute to allergic and irritant contact dermatitis.
3. An associated contact dermatitis may initiate an “autosensitization” rash, erythematous patches on the legs and arms. Stop nonessential topical medications or other products.



FIGURE 13.121 ■ Stasis Dermatitis. Erythematous patches and mild scaling in a patient with chronic venous insufficiency. Note location above the ankles. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.122 ■ Stasis Dermatitis. An example showing erythematous, scaly, and oozing patches over the lower leg. Several stasis ulcers are also present. (Used with permission from Fauci AS, Braunwald E, Kasper DL, et al. *Harrison's Principles of Internal Medicine*. 17th ed. New York, NY: McGraw Hill; 2008: 315.)

UREMIC FROST

Clinical Summary

Uremic frost is a classic manifestation of chronic renal failure; it is rarely seen today. It develops because of accumulation of urea in sweat. In advanced uremia, the accumulation may reach such a critical level that, upon its evaporation, a fine white powder is left on the skin surface. Associated hyperkalemia may also be present owing to concurrent renal failure.

Management and Disposition

Treatment of the underlying condition that resulted in the patient's uremia may prevent further accumulation of uremic frost. Typically, urgent dialysis is indicated.

Pearls

1. Although rare today, this condition may be seen in noncompliant patients or with environmental stressors, including inadequate air conditioning.
2. For patients presenting with altered mental status, attention to the airway, oxygenation, and rapid assessment and treatment of associated metabolic disorders, such as hyperkalemia, are paramount.



FIGURE 13.123 ■ Uremic Frost. Note the fine white powder on the skin of this patient with end-stage renal disease. (Photo contributor: Alan B. Storrow, MD.)

JAUNDICE

Clinical Summary

Jaundice is a light yellowing of the skin, mucous membranes, and sclera; it is generally detectable when bilirubin levels are about 3.0 mg/dL. Many patients may not be aware of the faint yellowing and present with seemingly unrelated symptoms. Up to 50% of patients with jaundice will have pruritus. The most important diagnoses to rule out are hemolytic anemias, viral hepatitis, chronic

alcohol abuse, autoimmune hepatitis, medications, primary biliary cirrhosis, primary sclerosing cholangitis, cholelithiasis, surgical strictures, and obstructive malignancies. Acetaminophen, penicillins, and oral contraceptives are some of the more common medications associated with jaundice.

Management and Disposition

As the etiology is broad, a thorough history focusing on associated symptoms (fever, pruritus, vomiting, hematochezia, melena, and abdominal pain), previous surgical procedures, and medication history (including over-the-counter medications) is essential. Physical findings of fever, abdominal tenderness, and hepatomegaly should be sought. Workup should include white blood cell count and differential, liver function tests including bilirubin levels, hepatitis viral screening, and imaging studies.



FIGURE 13.124 ■ Jaundice. Mild palmar jaundice in a dark-skinned patient. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Patients who consume large amounts of β -carotene (found in squash and carrots) may have mild yellowing of their skin (especially palms and soles) but will lack scleral icterus or elevations in bilirubin.
2. Women starting oral contraceptives may experience cholestasis in the first few

months that may cause jaundice.



FIGURE 13.125 ■ Jaundice, Scleral Icterus. Yellowing of the sclera in a patient with liver disease. (Photo contributor: R. Jason Thurman, MD.)

PORPHYRIA CUTANEA TARDA

Clinical Summary

Porphyrias are associated with enzymatic defects in heme bio-synthesis. Porphyria cutanea tarda (PCT) is the most common type. Patients have photosensitivity, skin fragility, and characteristic lesions on sun-exposed sites (most common on the dorsal hands and forearms). Typically, the skin is easily traumatized with blisters, erosions, superficial scars, milia, and hypertrichosis. PCT does not cause the life-threatening neurologic attacks associated with the acute porphyrias.

PCT may be induced by ethanol, estrogens, oral contraceptives, infections (hepatitis C and HIV), polychlorinated hydro-carbons, dialysis, and iron

overload. Pseudoporphyria, which is clinically indistinguishable from PCT, is associated with dialysis and certain medications (NSAIDs, furosemide, hydrochlorothiazides, and amiodarone, among others).

Management and Disposition

Laboratory diagnostics may begin in the ED with blood chemistries, porphyrin studies (24-hour urinary porphyrins can be ordered), and referral to a dermatologist. Long-term treatment includes phlebotomy and antimalarials (which can induce fatal hepatotoxicity in PCT patients). Discontinue any medications that might initiate PCT or pseudoporphyria.



FIGURE 13.126 ■ Porphyria Cutanea Tarda. Blisters and erosions of PCT. (Photo contributor: Selim Suner, MD, MS.)

Pearls

1. Consider PCT in a patient with fragile skin with erosions and scarring, but only on sun-exposed skin.
2. PCT does not have acute attacks of abdominal pain, neurologic deficits, psychosis, or autonomic dysfunction. Variegate porphyria, common in South Africans with Dutch ancestry, does present with the typical skin lesions and

acute attacks.

3. Examination of the urine with a Wood's lamp may reveal orange-red fluorescence.
4. Always consider medications (both prescribed and over-the-counter medications) and infections as they can worsen PCT.



FIGURE 13.127 ■ Porphyria Cutanea Tarda. The easily traumatized skin and erosions of PCT. (Photo contributor: Alan B. Storrow, MD.)

VITILIGO

Clinical Summary

Vitiligo is an acquired loss of pigmentation commonly involving the face, body folds, and backs of the hands. There is a positive family history in 30%. Initially the disease is limited, but it then slowly progresses over years. Vitiligo is secondary to the absence of epidermal melanocytes, which may be due to an autoimmune phenomenon. Approximately half of these cases begin in patients less than 20 years of age.

Management and Disposition

Refer patients to a dermatologist for further workup and long-term treatment.

Pearls

1. Vitiligo can occur at sites of trauma (Koebner phenomenon).
2. Wood lamp examination helps identify hypopigmented areas in patients with light complexions.
3. Tinea versicolor, in contrast, has a scale and positive KOH preparation.



FIGURE 13.128 ■ Vitiligo. Well-defined, hypopigmented areas are characteristic. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.129 ■ Vitiligo. Almost complete depigmentation of the hands. (Used with permission from Fauci AS, Braunwald E, Kasper DL, et al. *Harrison's Principles of Internal Medicine*. 17th ed. New York, NY: McGraw Hill; 2008: 312.)

MELASMA

Clinical Summary

Melasma is commonly seen on the face of young adult females. It consists of symmetric, well-defined, light to dark brown patches. The most common sites are on the malar cheek, lateral forehead, upper cutaneous lip, and mandible. Factors associated with accentuation of melasma include sunlight exposure, pregnancy (often called “the mask of pregnancy”), and oral contraceptives.

Management and Disposition

Most patients will be concerned about accentuation of their previously imperceptible melasma. Rule out pregnancy in new-onset or worsening melasma. Essential to any treatment is strict sun avoidance. Refer patients to a dermatologist for further treatment options.

Pearls

1. Melasma is common in young females with darker skin types, but all races can be afflicted.
2. Sun exposure on other parts of the body can cause accentuation of facial melasma.
3. Contrasted with the hyperpigmentation of melasma, Addison disease is diffuse hyperpigmentation with accentuation in sun-exposed areas.



FIGURE 13.130 ■ **Melasma.** Well-demarcated, hyperpigmented patch seen on the cheek. (Photo contributor: J. Matthew Hardin, MD.)

ABDOMINAL STRIAE (STRIAE ATROPHICAE)

Clinical Summary

Abdominal striae are linear, depressed, pink or bluish scar-like lesions that may later become silver or white. They are caused by weakening of the elastic cutaneous tissues from chronic stretching. They most commonly occur on the abdomen but are also seen on the buttocks, breasts, axilla, and thighs. Striae are commonly seen in obesity, pregnancy, rapid growth associated with puberty, Cushing syndrome, and chronic topical corticosteroid treatment.

Management and Disposition

This finding seldom presents as a condition requiring acute treatment; thus, attention is directed to determining and treating the underlying cause.

Pearls

1. Recent striae with moon facies, hypertension, renal calculi, osteoporosis, and psychiatric disorders are suggestive of Cushing syndrome.
2. The striae color (red/purple) caused by pregnancy typically fades with time, unlike striae associated with Cushing syndrome.
3. Inappropriate use of topical corticosteroids can result in permanent striae.



FIGURE 13.131 ■ Abdominal Striae. These striae are seen in a patient with recent weight gain, moon facies, and altered mental status. The patient was diagnosed with Cushing syndrome.

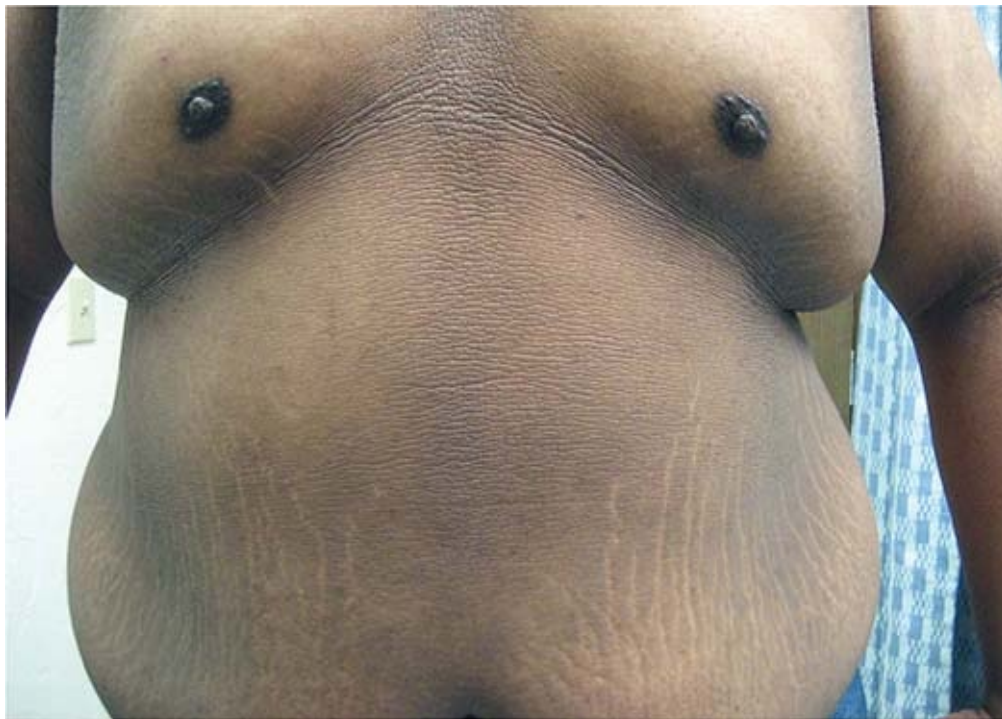


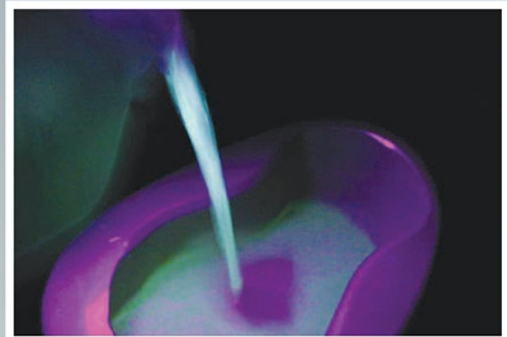
FIGURE 13.132 ■ Abdominal Striae. Obese patient with lower lateral wall striae. Also noted is thickened, hyperpigmented, abdominal skin, typical of acanthosis nigricans (associated with diabetes). (Photo

contributor: J. Matthew Hardin, MD.)

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PART 2

Specialty Areas



Chapter 14

PEDIATRIC CONDITIONS

Ashish Shah

Brad Sobolewski

Matthew R. Mittiga



Fifth Disease. Toddler with the classic slapped-cheek appearance of fifth disease caused by parvovirus B19. Note the lacy reticular macular rash on the shoulder and upper extremity. (Photo contributor: Anne W. Lucky, MD.)

Newborn Conditions

ERYTHEMA TOXICUM NEONATORUM

Clinical Summary

Erythema toxicum neonatorum is a benign, self-limited vesicopustular lesion of unknown etiology that occurs in up to 70% of term newborns. It is characterized by discrete, small, irregular erythematous macules or patches up to 2 to 3 cm in diameter with 1- to 3-mm firm pale yellow or white papules or pustules in the center. The trunk and proximal extremities are predominantly involved, but lesions can appear diffusely, only sparing the palms and soles. This rash usually appears within the first 24 to 72 hours of life but may be present at birth. The distinctive feature of erythema toxicum is its evanescence, with each individual lesion usually disappearing within 5 to 7 days. New lesions may occur in a waxing and waning fashion. The diagnosis is usually made based on the clinical appearance of the rash in an otherwise well-appearing neonate without any systemic signs of illness. Wright-stained slide preparations of a scraping from the center of the lesion demonstrate numerous eosinophils. Cultures from these lesions will be negative. The differential diagnosis includes neonatal acne, transient neonatal pustular melanosis, newborn milia, miliaria, infantile acropustulosis, neonatal herpes simplex, bacterial folliculitis, candidiasis, incontinentia pigmenti, and impetigo of the newborn.

Management and Disposition

No specific therapy is indicated in the setting of a well-appearing newborn with normal activity and appetite. Parents should be educated and reassured about the evanescence of the rash. In cases where impetigo, *Candida*, or herpes infections are suspected, a smear from the center of the lesion and bacterial and viral cultures may be necessary to make a final diagnosis.

Pearls

1. Erythema toxicum neonatorum is the most common newborn rash.
2. The lesions may present anywhere on the body but tend to spare the palms and soles.
3. Laboratory evaluation is unnecessary.



FIGURE 14.1 ■ Erythema Toxicum. Newborn infant with diffuse macular rash of erythema toxicum. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.2 ■ Erythema Toxicum. Close-up of lower extremity of a neonate with erythema toxicum. (Photo contributor: Robert W. Hickey, MD.)

SALMON PATCHES (NEVUS SIMPLEX)

Clinical Summary

Nevus simplex (salmon patch) is the most common vascular lesion in infancy, present in up to 80% of newborns. They are ectatic dermal capillaries that appear as a blanching, slightly pink-red macule or patch with indistinct borders most commonly on the nape of the neck, the glabella, mid-forehead, or upper eyelids. Lesions generally fade over the first 2 years of life and may become more prominent with crying or straining.

Management and Disposition

Parental education and reassurance can be helpful, but no immediate treatment is indicated. Pulsed dye laser may be considered for persistent lesions that are cosmetically undesirable.

Pearls

1. Salmon patches appear symmetrically and cross the midline in contrast to the unilateral distribution of a port-wine stain.
2. This lesion is referred to as a *stork bite* when seen on the nape of the neck or as an *angel's kiss* when appearing on the forehead.
3. About 5% of those appearing at the nape of the neck will persist or recur.
4. Obtain imaging to evaluate for spinal dysraphism in patients with a lumbosacral nevus simplex and another lumbosacral abnormality (dermal sinus or pit, patch of hypertrichosis, or deviated gluteal cleft).



FIGURE 14.3 ■ Salmon Patches. Newborn with characteristic salmon patches over his face. (Photo contributor: Anne W. Lucky, MD.)



FIGURE 14.4 ■ Salmon Patches. Child with patch over lower back consistent with salmon patches. (Photo contributor: Anne W. Lucky, MD.)



FIGURE 14.5 ■ Salmon Patches. Characteristic lesion on the nape of the neck is commonly called a stork bite. (Photo contributors: Katharine Hanlon and Kara Shah, MD, PhD.)

NEONATAL JAUNDICE

Clinical Summary

Neonatal jaundice occurs when total serum bilirubin is in excess of 5 mg/dL and progresses in a head-to-toe fashion as levels increase. Most cases of physiologic

(< 12 mg/dL) jaundice are self-limited without sequelae and appear on the 2nd or 3rd day of life, peaking between the 3rd and 5th day. Visual estimation of serum bilirubin level is not accurate enough to determine jaundice severity. Preterm infants may peak later.

Neonatal jaundice is due to increased production, decreased clearance, and increased circulation of bilirubin. The increased bilirubin production is a result of turnover of fetal red blood cells, a temporary decrease in conjugation and clearance by the immature newborn liver, and increased enterohepatic circulation. Risk factors for unconjugated (indirect) hyper-bilirubinemia include maternal diabetes, prematurity, drugs, polycythemia, traumatic delivery with cutaneous bruising or hematoma, breastfeeding, and ABO (O mother and A/B infant) or Rh(D) incompatibility [Rh(D)-negative mother and Rh(D)-positive infant]. Most infants with jaundice have no “disease” per se, but a careful history and organized approach is necessary to identify potentially pathologic causes. Kernicterus manifests in irreversible neurologic abnormalities and is the long-term result of bilirubin-induced neurologic dysfunction secondary to extreme unconjugated hyperbilirubinemia, which leads to neuronal death and pigment deposition in the basal ganglia and cerebellum.

Management and Disposition

The well-appearing jaundiced infant in day 2 or 3 of life should have a total serum bilirubin level or direct and indirect bilirubin levels sent. Additional labs for the severely jaundiced infant include blood type, Coombs test, complete blood count (CBC) with smear for red cell morphology, reticulocyte count, and indirect and direct bilirubin levels. Transcutaneous bilirubin measurement devices can underestimate total bilirubin at levels > 15 mg/dL; therefore, a serum measurement is recommended for severely jaundiced newborns. Initial management should ensure adequate hydration and phototherapy if the bilirubin level is > 95th percentile. The level of serum bilirubin at which to start phototherapy can be obtained from a standardized nomo-gram and is dependent upon the infant’s postnatal age in hours, gestational age, and an assessment of risk factors. The goal of phototherapy is to maintain the bilirubin level below 20 mg/dL. Exchange transfusion is considered if the serum level remains elevated (22-25 mg/dL) despite appropriate phototherapy.

Pearls

1. Onset of clinical jaundice in the first 24 hours of life strongly suggests the presence of a pathologic process.
2. Direct serum bilirubin concentration exceeding 10% of total serum bilirubin or 2 mg/dL suggests hepatobiliary disease, a metabolic disorder, or sepsis.
3. The Bhutani nomogram stratifies the risk of subsequent bilirubin levels being elevated without intervention, not the risk of clinically significant complications and outcomes of hyperbilirubinemia at the specified level.



FIGURE 14.6 ■ Neonatal Jaundice. Newborn with yellowish hue to skin consistent with jaundice. (Photo contributor: Kevin J. Knoop, MD, MS.)

NEONATAL MILK PRODUCTION (WITCH'S MILK)

Clinical Summary

Neonatal galactorrhea occurs in up to 6% of term newborns and is usually secondary to transplacental transfer of maternal estrogen. These hormonal effects (maternal estrogens and endogenous prolactin) lead to palpable breast buds in approximately one-third of all term newborns. Males and females are

equally affected. In most cases, the breast enlargement and galactorrhea begin to subside after the 2nd week of life in males and 2 to 6 months in females. Infants with neonatal breast hypertrophy may be predisposed to infections (mastitis or abscess) possibly incited by repetitive manipulation of the enlarged breast bud by a caregiver. The differential diagnosis includes early mastitis with purulent nipple discharge.

Management and Disposition

Treatment is not necessary unless infection is suspected. Parents can be reassured that this is a normal finding, and follow-up to resolution should occur at routine well-child care visits.

Pearls

1. Classical presentation includes the presence of clear colostrum-like secretions in newborns with hypertrophied mammary tissue without erythema or tenderness. Persistence of enlarged breast buds beyond 6 months of age should prompt follow-up with a pediatric endocrinologist.
2. In an older child, galactorrhea may be the presenting sign of hypothyroidism or pathologically elevated prolactin levels.
3. Female infants may also experience vaginal bleeding in the 1st weeks of life due to withdrawal from maternal hormones.



FIGURE 14.7 ■ Witch's Milk. Milky fluid draining from the nipple in a newborn. (Photo contributor: Michael J. Nowicki, MD.)

NEONATAL MASTITIS

Clinical Summary

Neonatal mastitis is an infection of the breast tissue that occurs in full-term neonates. The peak incidence of mastitis without abscess is in the 2nd week of life and the 4th week of life for mastitis with abscess. Females are affected more often than males in a 2:1 distribution. Clinically, it manifests as swelling,

induration, erythema, warmth, and tenderness of the affected breast. The ipsilateral axillary lymph nodes may be swollen. Approximately 50% develop an abscess. In some cases, purulent discharge may be expressed from the nipple. Fever may be present in 25% of affected patients. Other systemic symptoms (irritability, decreased appetite, and vomiting) are less common but indicate a more severe infection if present. Bacteremia is rare. *Staphylococcus aureus*, specifically methicillin-resistant strains (MRSA), is the most common pathogen, causing 75% to 85% of cases. Rarely, gram-negative organisms or group B or D streptococci are the cause. If treatment is delayed, mastitis may progress rapidly with involvement of subcutaneous tissues and subsequent toxicity. In the initial stages, neonatal mastitis may mimic mammary tissue hypertrophy owing to maternal passive hormonal stimulation. Minor trauma, cutaneous infections, and duct blockage may precede this infection.



FIGURE 14.8 ■ Neonatal Mastitis. Neonate with left-sided breast swelling, erythema, and purulent discharge. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Immediate treatment is important to avoid cellulitic spread and breast tissue damage. Well-appearing infants under 2 months of age should be admitted and treated with antistaphylococcal monotherapy. In areas of high MRSA prevalence, choose clindamycin or vancomycin. If MRSA is not a concern, nafcillin is appropriate. Adjustment of coverage can be made if results of cultures or Gram stain are available, especially in the presence of gram-negative bacilli. In cases

involving systemic signs of infection, rapid subcutaneous spread, or toxic appearance, a complete sepsis workup should be performed followed by hospitalization. If no organism is seen on Gram stain, a parenteral antistaphylococcal penicillin plus an aminoglycoside or cefotaxime alone should be used. Local MRSA prevalence should determine whether coverage is necessary. In cases of palpable fluctuance or abscess, ultrasound and prompt surgical consultation should be obtained to assess the need for incision and drainage. Conservative treatment with intravenous (IV) antibiotics often results in resolution of the fluctuance without surgical intervention. Recovery is usually within 5 to 7 days.

Pearls

1. Antibiotic choice should include coverage for *S aureus* and possibly MRSA depending on local prevalence.
2. Maintain a low threshold for initiating a sepsis workup.
3. Mastitis is typically a clinical diagnosis, but if there is uncertainty, ultrasound is useful for further characterization.



FIGURE 14.9 ■ Neonatal Mastitis. Neonate with marked swelling, erythema, and purulent discharge. (Photo contributor: Emergency Medicine Department, Naval Medical Center, Portsmouth, VA.)

UMBILICAL GRANULOMA

Clinical Summary

An umbilical granuloma is granulation tissue with incomplete epithelialization that persists following cord separation. It is the most common cause of an umbilical mass in neonates. Parents will describe a persistent discharge from the umbilicus after the cord has dried and separated. It appears soft, pink, wet, and friable. Infants with an umbilical granuloma do not have localized swelling, redness, warmth, tenderness, or fever. An umbilical polyp is a rare anomaly resulting from the persistence of the omphalomesenteric duct or the urachus and may have a similar appearance. A polyp is usually firm with a mucoid secretion. The differential diagnosis also includes omphalitis, an infection of the umbilicus and surrounding structures, which should be considered in ill-appearing neonates.

Management and Disposition

Advise parents to keep the granuloma dry and exposed to the air as often as possible. Cleaning and drying of the umbilical cord base with alcohol is unnecessary and may irritate the skin and delay healing. Cauterization of the granuloma by application of topical silver nitrate is the treatment of choice. It is important to protect the surrounding skin (apply petroleum jelly or antibiotic ointment) and remove excess silver nitrate to avoid chemical burns and skin staining. The cauterization may need to be repeated at 3-day intervals if drainage persists. Topical steroids have shown success in treating umbilical granulomas, but further studies are needed to assess safety and noninferiority.



FIGURE 14.10 ■ Umbilical Granuloma. Newborn infant with umbilical granuloma visible in umbilicus. (Photo contributor: Anne W. Lucky, MD.)

Pearls

1. An umbilical granuloma is the most common umbilical mass in neonates.
2. The only sign of granuloma formation may be the presence of nonpurulent discharge noted in the diaper area or on clothing that is in contact with the umbilicus.
3. Omphalitis presents with redness of the periumbilical area typically tracking upward in the midline and often with a purulent discharge from the umbilicus. It can progress to abdominal wall cellulitis or peritonitis and requires a complete sepsis workup and hospital admission for treatment with broad-spectrum parenteral antibiotics.



FIGURE 14.11 ■ Omphalomesenteric Duct. This red mass resembling a granuloma was found to be an omphalomesenteric duct. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.12 ■ Omphalomesenteric Duct. A fistulogram confirms continuity between the duct and intestine. (Photo contributor: Kevin J. Knoop, MD, MS.)

HYPERTROPHIC PYLORIC STENOSIS

Clinical Summary

Hypertrophic pyloric stenosis (HPS) is characterized by progressive

postprandial, nonbilious vomiting that steadily increases in frequency and amount due to hypertrophy of the pyloric musculature and edema of the pyloric canal, producing gastric outlet obstruction. It is usually diagnosed in infants from birth to 5 months, most commonly at 2 to 8 weeks of life. The vomiting may become forceful and is then described as projectile (although this pattern is not always present). There is a familial tendency, and white males (especially firstborn) are more frequently affected. During the physical examination, peristaltic waves may be observed traveling from the left upper to right upper quadrants. The hypertrophy of the antral and pyloric musculature produces the “olive” to palpation (best palpated in the epigastrium or right upper quadrant after emesis, following feeding, or after emptying the stomach with a nasogastric tube). As a result of persistent vomiting, hypochloremic, hypokalemic metabolic alkalosis with varying degrees of dehydration and failure to thrive may occur when the diagnosis is not made early in the course.

The finding of a pyloric “olive” on palpation of the abdomen is pathognomonic but has largely been supplanted by trans-abdominal ultrasound. Diagnosis is confirmed by a pyloric muscle thickness, pyloric muscle channel length, and pyloric diameter of 3 mm, 15 mm, and 10 mm, respectively. The differential diagnosis includes intestinal obstruction or atresia, malrotation with volvulus, hiatal hernia, gastroenteritis, adrenogenital syndrome, increased intracranial pressure, esophagitis, sepsis, gastroesophageal reflux, and poor feeding technique.

Management and Disposition

Treatment includes correction of electrolyte imbalances and dehydration, as well as surgical consultation for curative Ramstedt pyloromyotomy. Failure to correct metabolic alkalosis prior to surgery can increase the risk of postoperative apnea.

Pearls

1. HPS is the most common cause of metabolic alkalosis in infancy.
2. Serial examinations and observation of the child after oral fluid challenges for persistent projectile vomiting may aid in making the diagnosis.
3. Clinical manifestations of pyloric stenosis begin at a mean age of 3 weeks after birth.
4. Pyloric ultrasonography is the diagnostic study of choice.
5. Less than 2% of infants with HPS have bilious vomiting. Some may have

hematemesis.



FIGURE 14.13 ■ Gastric Wave of Hypertrophic Pyloric Stenosis. A gastric wave can be seen traversing the abdomen in this series of photographs of a patient with HPS. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.14 ■ Ultrasound Target Sign in HPS. This transverse ultrasound shows redundant, infolded mucosa (arrowheads) between muscular components (arrows), referred to as the “target sign.” (Photo contributor: Lawrence B. Stack, MD.)

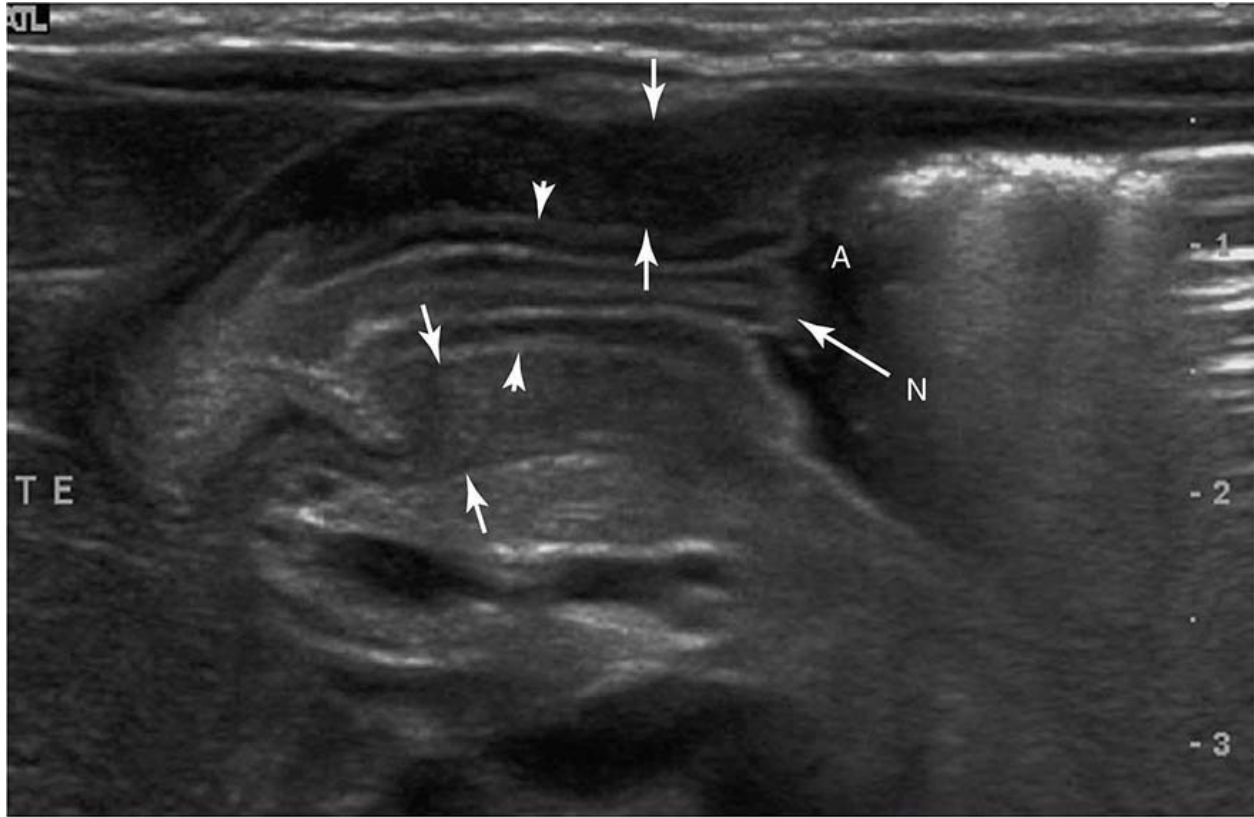


FIGURE 14.15 ■ Antral Nipple Sign in HPS. This longitudinal sonogram shows two-layered, thickened mucosa (arrowheads) surrounded by muscular components (arrows). Redundant pyloric mucosa protrudes into the fluid in the gastric antrum (A), forming the “antral nipple sign” (N) in HPS. (Photo contributor: Lawrence B. Stack, MD.)

INTESTINAL MALROTATION WITH VOLVULUS

Clinical Summary

Intestinal malrotation with volvulus is primarily a condition of infancy but can be seen in older children and adults. During week 4 of embryonic development, the primary intestinal loop bulges into the yolk sac and rotates 270 degrees counterclockwise. Between weeks 8 and 10, it returns to the enlarged abdominal cavity and is fixed via mesentery that extends from the ligament of Treitz to the ileocecal valve in the right lower quadrant. In malrotation, the duodenum, jejunum, and cecum are partially rotated with the bowel anchored by an abnormally thin band of mesentery. The bowel can twist on this thin mesentery, which contains the superior mesenteric artery, leading to acute intestinal obstruction and midgut vascular compromise known as volvulus. Also, the

abnormally positioned cecum now rests in the upper abdomen fixed to the right lateral abdominal wall by bands of peritoneum (Ladd bands) that cross and can obstruct the duodenum.

Presentation is with vomiting in 50% of cases and in 90% of newborns. The vomiting may not be bilious, but it is classically described as such. Patients are often irritable with significant abdominal tenderness. Third space fluid losses increase as gut ischemia progresses. Differential diagnosis includes pyloric stenosis, intestinal atresias, appendicitis, necrotizing enterocolitis, and intussusception.



FIGURE 14.16 ■ Intestinal Malrotation with Volvulus. Antero-posterior x-ray of the abdomen in a newborn with bilious emesis. Dilated, air-filled stomach with a relative paucity of gas in the distal bowel. (Photo contributor: Alexander Towbin, MD.)

Management and Disposition

Emergent surgical consultation is indicated. Plain x-rays are rarely diagnostic but

may show signs of small bowel obstruction. A normal ultrasound does not rule out malrotation. An upper gastrointestinal (GI) series is the most helpful study. One-quarter of cases may have an equivocal result. Preoperative management consists of resuscitation with IV fluids, placing a gastric tube for decompression, and administration of broad-spectrum antibiotics.

Pearls

1. Fifty percent of patients with malrotation present with volvulus in the 1st month of life.
2. Over 30% of cases of malrotation with volvulus present beyond early childhood, usually with an insidious onset with abdominal pain as the most common symptom.
3. The imaging test of choice is the upper GI series, but computed tomography (CT) should be considered in ill-appearing patients in whom administration of enteral contrast may be difficult.

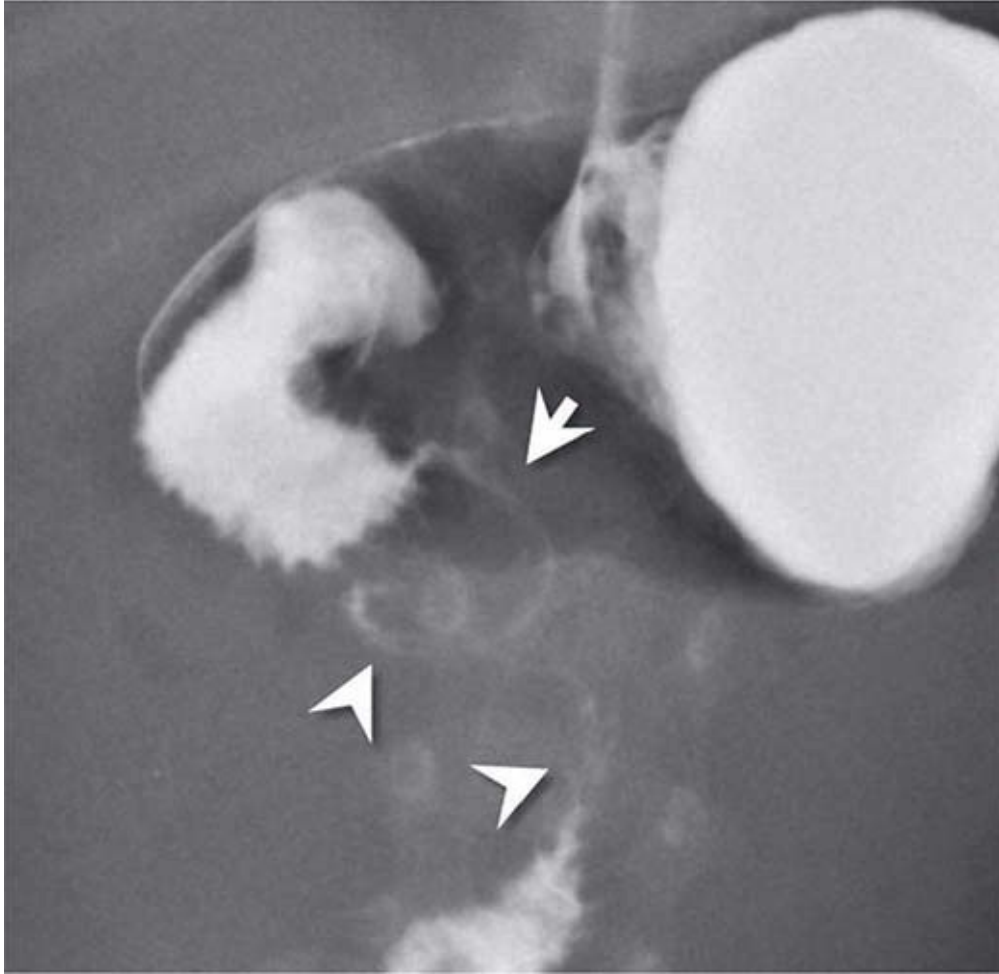


FIGURE 14.17 ■ Intestinal Malrotation with Volvulus. Antero-posterior radiograph from an upper GI series shows malrotation with midgut volvulus. The duodenum (*arrowheads*) does not cross mid-line and does not extend superiorly to the level of the pylorus. The spiraling downward appearance is consistent with volvulus. (Photo contributor: Alexander Towbin, MD.)

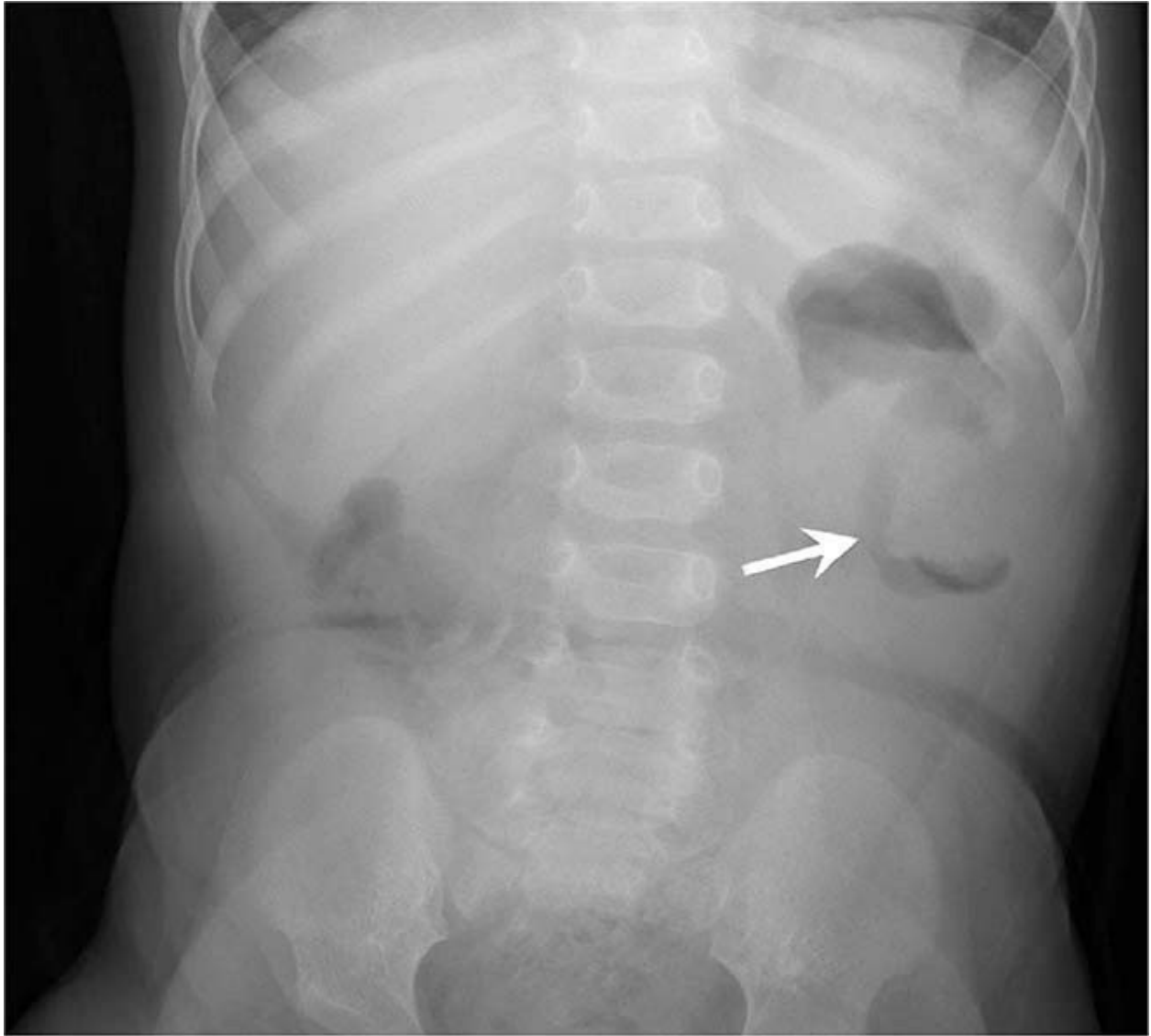


FIGURE 14.18 ■ Intestinal Malrotation with Volvulus. Soft-tissue mass (*arrow*) surrounded by air within the left upper quadrant near the hepatic flexure. (Photo contributor: Alexander Towbin, MD.)

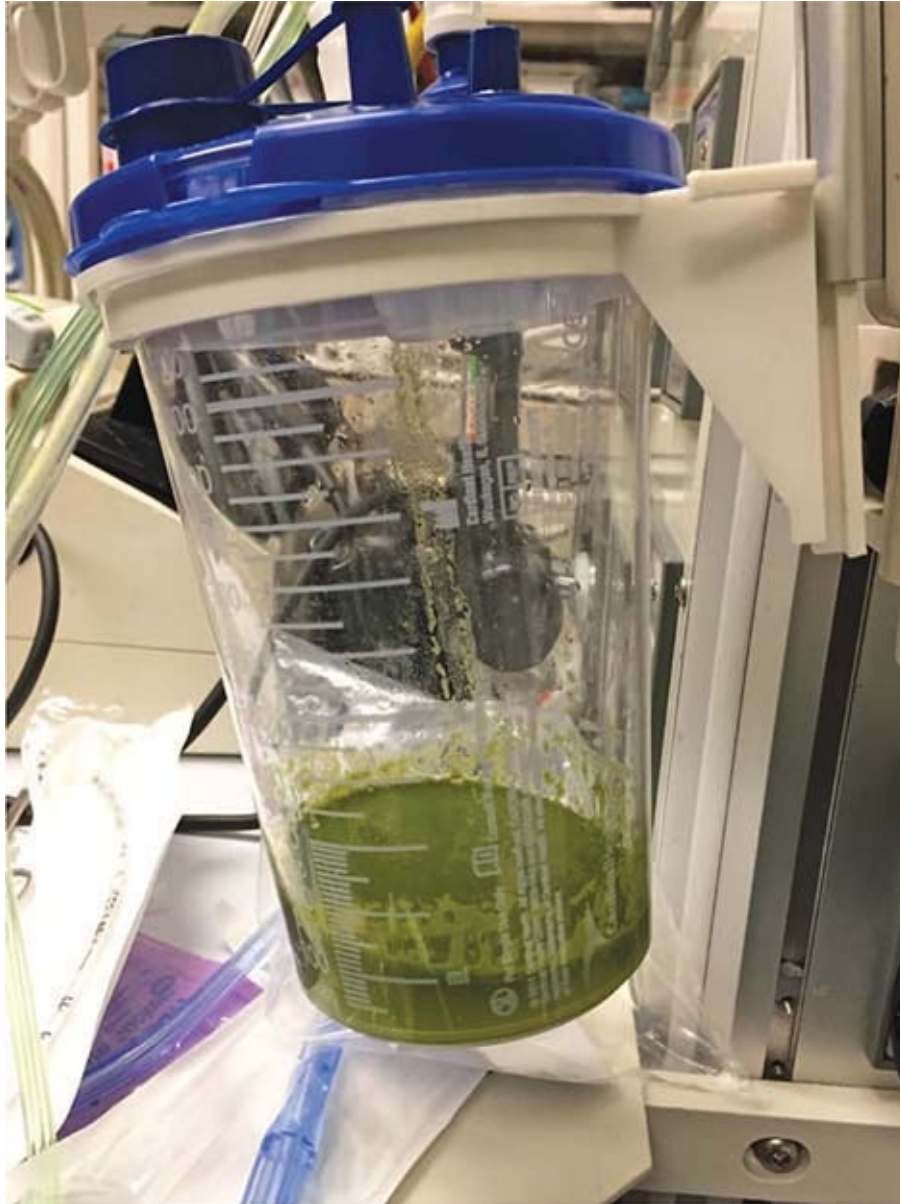


FIGURE 14.19 ■ Bilious Emesis. Green bilious emesis is seen from a pediatric patient with intestinal malrotation. (Photo contributor: Jaime Kaye Otillo, MD.)

Rashes and Lesions

ERYTHEMA INFECTIONOSUM (FIFTH DISEASE)

Clinical Summary

Erythema infectiosum is a viral infection caused by parvovirus B19, presenting

most commonly between 4 and 10 years of age. It begins with nonspecific prodromal symptoms including malaise, coryza, headache, fever, nausea, and diarrhea. In 25% of cases, the classic rash appears 2 to 5 days into the illness and is characterized initially by the “slapped cheeks” appearance of a bright red malar, macular rash that spares the nasal ridge and perioral areas. A reticulated, lacy erythematous maculopapular eruption with central clearing then appears on the extensor surfaces of extremities. The differential diagnosis includes other morbilliform eruptions such as measles, rubella, roseola, and infectious mononucleosis. Bacterial infections (eg, scarlet fever), drug reactions, and other skin conditions such as guttate psoriasis, papular urticaria, atopic dermatitis, and erythema multiforme are also included in the differential.

Management and Disposition

Treatment is aimed at symptomatic relief. Parents can be reassured that this exanthem is benign and self-limited. Once the rash appears, the patient is no longer contagious. It is important to educate the patient and family about the possible risk of parvovirus B19 as a cause of hydrops fetalis or fetal deaths early in pregnancy. It can also cause a transient and, rarely, permanent aplastic crisis in patients with hematologic conditions such as sickle cell disease, hereditary spherocytosis, and various hemolytic anemias, or in the immunocompromised.

Pearls

1. Recrudescence of the lacy, reticular rash may occur with exercise, overheating, emotional upset, or sun exposure as a result of cutaneous vasodilatation.
2. Parvovirus B19 is the most common cause of hydrops fetalis. Pregnant mothers of children diagnosed with erythema infectiosum should have their serologic status determined.
3. In young adults, parvovirus B19 can cause papular purpuric gloves and socks syndrome.



FIGURE 14.20 ■ Fifth Disease. Toddler with the classic slapped-cheek appearance of fifth disease caused by parvovirus B19. Note the lacy reticular macular rash on the shoulder and upper extremity. (Photo contributor: Anne W. Lucky, MD.)

ROSEOLA INFANTUM (EXANTHEM SUBITUM)

Clinical Summary

The typical presentation of roseola infantum involves a prodrome characterized by a 3- to 5-day history of high fever often exceeding 40°C (104°F) in a child 6 months to 3 years of age. The child may be fussy and have lymphadenopathy

and Nagayama spots (erythematous papules on the soft palate and uvula), but is often otherwise well appearing. After the child's fever abruptly abates, the typical exanthem appears, characterized by blanching erythematous macules and papules on the trunk, neck, proximal extremities, and occasionally the face. The evanescent rash fades within 2 to 4 days but may only last several hours. The causative agent in 90% of cases is human herpesvirus 6 (HHV-6). The differential diagnosis includes viruses such as measles, rubella, parvovirus B19, or infectious mononucleosis. Bacterial infections (eg, scarlet fever), drug reactions, guttate psoriasis, papular urticaria, and erythema multiforme are also included in the differential.

Management and Disposition

As with most viral infections, only supportive therapy is necessary. Special attention should be paid to maintaining fluid intake, controlling fever for the patient's comfort, and educating parents about the benign, self-limited nature of this illness.

Pearls

1. With defervescence and appearance of the rash, the patient is no longer contagious.
2. The most frequent complication of roseola is febrile seizures.



FIGURE 14.21 ■ Roseola Infantum (Exanthem Subitum). Toddler with maculopapular eruption of roseola. (Photo contributor: Raymond C. Baker, MD.)

IMPETIGO

Clinical Summary

Impetigo is a contagious bacterial infection of the superficial skin that is caused by *Streptococcus pyogenes* (group A β -hemolytic *Streptococcus*) and *S aureus*. It most commonly affects children 2 to 5 years of age and usually involves the face and extremities. It begins as small vesicles or pustules with very thin roofs that rupture easily with the release of a cloudy fluid and subsequent formation of honey-colored crusts. Variants include bullous and the ulcerative forms. The lesions may spread rapidly by autoinoculation secondary to scratching and coalesce to form larger areas of infection. The differential diagnosis includes second-degree burns, varicella, herpes simplex infections, nummular dermatitis, superinfected eczema, and scabies.

Management and Disposition

These lesions are highly contagious and spread by direct contact. Hand washing and personal hygiene should be emphasized to the patient and family. Application of topical antibacterials, such as mupirocin ointment, has proven to be as effective as oral antibiotics if there are a limited number of lesions and no bullae. Over-the-counter triple antibiotic ointments (bacitracin-neomycin-polymyxin B) may not be as effective. If lesions are extensive and/or bullous, oral antibiotic coverage is indicated.

Pearls

1. Inflicted cigarette burns may resemble the lesions of impetigo.
2. Poststreptococcal glomerulonephritis and rheumatic fever can be complications of impetigo caused by group A β -hemolytic *Streptococcus*.



FIGURE 14.22 ■ Impetigo. Infant with perioral erosions oozing discharge forming honey-colored crusts. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.23 ■ Impetigo. Chest wall and axillary erosions oozing discharge forming honey-colored crusts. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.24 ■ Bullous Impetigo. A child with impetiginous lesions on the face. Note the formation of bullae. (Photo contributor Anne W. Lucky, MD.)



FIGURE 14.25 ■ Bullous Impetigo. Bullae with honey-colored exudate in a neonate with fever. (Photo contributor: Lawrence B. Stack, MD.)

MEASLES

Clinical Summary

Measles presents as an acute febrile illness with a 3- to 4-day prodromal period characterized initially by fever, malaise, and anorexia, followed closely by conjunctivitis, coryza, and cough. Koplik spots, the pathognomonic enanthem of measles, appear as 1- to 3-mm red papules with gray-white centers on the buccal mucosa. They usually present transiently approximately 48 hours before the development of the characteristic erythematous blanching maculopapular rash. The rash appears on day 3 or 4 after the onset of fever as dark red to purple macules and papules on the forehead, around the hairline, and behind the earlobes, subsequently spreading in a cephalocaudal progression, often becoming confluent. The lesions tend to fade in the same order that they appear.



FIGURE 14.26 ■ Measles. Morbilliform rash on the face consistent with measles. (Photo contributor: Javier A. Gonzalez del Rey, MD.)

Most cases recover without complications; others may develop otitis media, croup, pneumonia, encephalitis, myocarditis/pericarditis, keratitis, and rarely subacute sclerosing panencephalitis, a very late complication. The differential diagnosis of the characteristic rash is vast and includes exanthem subitum; rubella; infections caused by echovirus, coxsackievirus, and adenoviruses; toxoplasmosis; infectious mononucleosis; scarlet fever; Kawasaki disease; drug reactions; Rocky Mountain spotted fever; and meningococemia.

Management and Disposition



FIGURE 14.27 ■ Koplik Spots. Punctate white spots are seen on buccal mucosa seen on the 3rd day of this illness. (Photo contributor: CDC Public Health Image Library.)

Supportive therapy includes bed rest, antipyretics, and adequate fluid intake. Complications should be treated accordingly. Currently available antivirals are not effective. The World Health Organization recommends oral vitamin A once per day for 2 days in vitamin A–deficient areas to reduce morbidity and mortality. Postexposure prophylaxis (PEP) includes administration of the measles-mumps-rubella (MMR) vaccine within 72 hours of exposure to a patient with active measles. Passive immunization with IV immune globulin (IVIG) is effective for prevention and attenuation of measles if given within 6 days of the initial exposure especially in pregnant women and the immunocompromised. During outbreaks, MMR vaccine can be given to infants younger than 12 months. However, such infants require an additional two doses of MMR at the recommended ages after their 1st birthday.

Pearls

1. Measles outbreaks are being seen in developing countries with decreased vaccination rates.
2. MMR vaccine is preferable to IVIG as PEP.
3. Morbilliform means measles-like.
4. Maintain airborne transmission precautions for suspected and confirmed measles cases. Healthcare providers should use appropriate respiratory protection (N95 respirator or other respirator with similar effectiveness).



FIGURE 14.28 ■ Measles. Note the maculopapular rash on the infant's face, which is one of the hallmark symptoms of this disease. (Photo contributors: James L. Goodson, MPH, courtesy of the CDC/Rebecca Martin, PhD.)



FIGURE 14.29 ■ Measles Conjunctivitis. A maculopapular facial rash and conjunctivitis are seen. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)



FIGURE 14.30 ■ Measles Rash. Morbilliform rash on the back consistent with measles. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)



FIGURE 14.31 ■ Measles. A morbilliform rash on the back consistent with measles. (Photo contributor: David Effron, MD.)

VARICELLA (CHICKENPOX)

Clinical Summary

Chickenpox results from primary infection with varicella zoster virus (VZV) and is characterized by a generalized pruritic vesicular rash, fever, and mild systemic symptoms. Fifteen days after exposure and following a prodrome of fever,

malaise, pharyngitis, and/or loss of appetite, the characteristic generalized pruritic vesicular rash develops. The lesions usually develop 24 hours after the onset of illness, appear in crops, start on the trunk and spread peripherally, and evolve from erythematous, pruritic macules to papules and vesicles (rarely bullae) that finally crust over within 48 hours. The classic lesions are tear-drop vesicles surrounded by an erythematous ring (“dewdrop on a rose petal”). The most common complication of varicella is secondary bacterial skin infection, usually with *S pyogenes* or *S aureus*. Other complications from varicella include encephalitis, glomerulonephritis, hepatitis, pneumonia, arthritis, and meningitis. Cerebellitis (manifested clinically as ataxia) may develop and is usually self-limited. Other viral infections that may manifest with vesicular rashes include herpes simplex, zoster, coxsackie, influenza, echovirus, and vaccinia. On occasion, varicella can be confused with papular urticaria.

Management and Disposition

Suspected varicella infection should lead to strict isolation in a negative air flow room early in the emergency department or office encounter. Most children have a self-limited illness and do not develop any complications. Treatment should be supportive and directed to pruritus and fever control while avoiding salicylates because of their association with Reye syndrome. Oral acyclovir initiated within 24 hours of the onset of the rash may result in a modest decrease in the duration of symptoms and in the number and duration of skin lesions. Acyclovir is not recommended routinely for treatment of uncomplicated varicella in an otherwise healthy child less than 12 years of age. In the immunocompromised host, varicella zoster immunoglobulin (VZIG), IV acyclovir, and hospital admission are indicated.



FIGURE 14.32 ■ Varicella (Chickenpox). Multiple cloudy vesicles of varicella. (Photo contributors: Katharine Hanlon and Kara Shah, MD.)

Pearls

1. Skin lesions in varicella present in successive crops so that macules, papules, vesicles, and crusted lesions may all be present at the same time.
2. Healthy children are no longer contagious when all lesions have crusted over (usually 4-5 days from the development of the initial lesions).
3. Consider oral acyclovir for those at risk for more severe infection, including anyone older than 12 years, with chronic diseases, and taking chronic aspirin or corticosteroid therapy.
4. PEP should be offered in people who do not have evidence of immunity. PEP includes VZIG within 10 days of exposure in high-risk patients (immunocompromised patients, newborns whose mothers have varicella 5 days before to 2 days after delivery, or pregnant women) or immunization with varicella vaccine within 3 to 5 days of exposure in non-high-risk patients.



FIGURE 14.33 ■ Varicella (Chickenpox). Vesicles in different stages of maturation. Note vesicles surrounded by an erythematous ring (“dewdrop on a rose petal”). (Photo contributor: Lawrence B. Stack, MD.)

HERPES ZOSTER

Clinical Summary

Zoster (shingles) represents a reactivation of latent VZV and has been noted as early as the 1st week of life in infants born to mothers who contracted varicella

during pregnancy. The lesions present as clustered vesicles or bullae in a dermatomal distribution. The pain of acute neuritis occurs in 75% of patients with herpes zoster. Prodromal pain can be constant or intermittent and burning or stabbing and can precede the lesions by days to weeks. Sometimes pain can persist beyond 1 month after the lesions have disappeared (known as postherpetic neuralgia).

The diagnosis is usually made clinically; however, tissue polymerase chain reaction, cultures, direct fluorescent antibodies, and Tzanck smears can be done from vesicle scrapings for confirmation. Impetigo and cutaneous burns may mimic the appearance of herpetic vesicles. Varicella (chickenpox) is more diffusely spread, although a small crop of lesions may mimic zoster. Zoster also may be confused with herpes simplex virus (HSV) infection, although a close examination should reveal a dermatomal distribution in zoster.



FIGURE 14.34 ■ Herpes Zoster. Vesicles in a classic thoracic dermatomal distribution are seen in this child. (Photo contributor: Frank Birinyi, MD.)

Management and Disposition

Currently, acyclovir (800 mg orally five times per day for 7-10 days initiated within 72 hours of the onset of the rash in children ≥ 12 years) is the treatment of choice for zoster infections in immunocompetent children. Pain relief and prevention of secondary infection are also important. IV antiviral therapy is recommended for immunocompromised patients.

Pearls

1. Zoster can occur in children of all ages.
2. The most common sites for the development of zoster lesions are those supplied by the trigeminal nerve and the thoracic ganglia.
3. A patient with zoster can transmit chickenpox (varicella) to a nonimmune child or adult.
4. Herpes zoster ophthalmicus is a sight-threatening condition that presents with hyperesthesia in the effected eye. Vesicular lesions on the nose (Hutchinson sign) are associated with an increased risk of eye involvement.



FIGURE 14.35 ■ Herpes Zoster. Vesicles in a trigeminal nerve distribution. (Photo contributor: Anne W. Lucky, MD.)

HAND, FOOT, AND MOUTH SYNDROME

Clinical Summary

Hand, foot, and mouth syndrome (HFMS) is a seasonal (summer-fall) viral infection caused most frequently by coxsackievirus A16, with other enterovirus serotypes implicated less frequently. Toddlers and school-aged children are affected most commonly, although adults may also be affected. It is

characterized by a prodrome of fever, malaise, sore throat, and anorexia over 1 to 2 days, followed by the appearance of the characteristic enanthem in the posterior oropharynx and tonsillar pillars consisting of small, red macules evolving into small vesicles 1 to 3 mm in diameter that rapidly ulcerate. Oral manifestations are followed by a vesicular eruption characterized by 3- to 7-mm erythematous macules with a central gray vesicle on the hands and feet involving the palmar and plantar surfaces and interdigital surfaces. A nonvesicular rash may also be present on the buttocks, face, and legs.



FIGURE 14.36 ■ Hand, Foot, and Mouth Syndrome. Erythematous vesicular rash scattered on the palms consistent with coxsackievirus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.37 ■ Hand, Foot, and Mouth Syndrome. Vesicular rash of the feet consistent with coxsackievirus. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 14.38 ■ Hand, Foot, and Mouth Syndrome. Discrete vesicular erosions on the posterior oropharynx and soft palate secondary to coxsackievirus. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 14.39 ■ Hand, Foot, and Mouth Syndrome. Nonvesicular rash on the buttocks, a common site. (Photo contributor: Binita R. Shah, MD. From Shah B, Lucchesi M, Amodio J, Silverberg M. *Atlas of Pediatric Emergency Medicine*. 2nd ed. New York, NY: McGraw Hill; 2013: Fig. 3.66D, p. 111.)

Management and Disposition

Supportive therapy (hydration maintenance with fever and pain control) is the mainstay of treatment. It is essential to discuss the duration and characteristics of the illness with the parents. In most cases, the course is self-limited, resolving in 2 to 3 days after the appearance of rash without further complication. Rare secondary complications such as myocarditis, pneumonia, pulmonary hemorrhage, and meningoencephalitis may occur.



FIGURE 14.40 ■ Hand, Foot, and Mouth Syndrome. Severe vesicular rash of the feet consistent with coxsackievirus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.41 ■ Hand, Foot, and Mouth Syndrome. Severe vesicular rash of the palms consistent with coxsackievirus. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. The child is contagious until all vesicles have resolved.
2. The oral lesions tend to involve the posterior oropharynx, as contrasted with those of herpetic gingivostomatitis, which typically involve the anterior structures of the mouth.
3. Varicella vesicles are located more centrally, are more extensive, and usually spare the palms and soles.
4. A more virulent disease caused by coxsackievirus A6 more frequently affects adults. Fever and rash are more severe, and hospitalization is more common than with typical HFMS.



FIGURE 14.42 ■ Hand, Foot, and Mouth Syndrome. Painful vesicular erosions on the posterior oropharynx in a young adult with fever, myalgias, and anorexia consistent with coxsackievirus. (Photo contributor: Timothy S. Forsythe, DO.)



FIGURE 14.43 ■ Hand, Foot, and Mouth Syndrome. Severe vesicular rash of the face seen in the patient in Figs. 14.40 and 14.41 consistent with coxsackievirus. Honey-colored crusty discharge suggests concurrent impetigo. (Photo contributor: Lawrence B. Stack, MD.)

COLD PANNICULITIS (POPSICLE PANNICULITIS)

Clinical Summary

Cold panniculitis represents acute cold injury resulting in inflammation of the subcutaneous fat. It manifests as erythematous, indurated nodules, and plaques on exposed skin, especially the perioral areas and cheeks. Lesions appear 24 to 72 hours after exposure to cold and gradually soften and return to normal over 1 to 2 weeks usually without permanent sequelae. This phenomenon is caused by subcutaneous fat solidification and necrosis following exposure to cold temperatures. It is much more common in infants. The differential diagnosis includes facial cellulitis, frostbite, trauma, pressure erythema, giant urticaria, and contact dermatitis.

Management and Disposition

Treatment is supportive. Parental education and reassurance are important.

Pearls

1. Because these lesions may also be painful, the differentiation of cold panniculitis from cellulitis may be difficult. The absence of systemic symptoms, especially fever, and the history of cold exposure are more suggestive of cold panniculitis.
2. The lesions may resolve with resulting hyperpigmentation of the affected area.



FIGURE 14.44 ■ Cold Panniculitis. Infant with cheek erythema, swelling, and discoloration consistent with popsicle panniculitis or cold injury. (Photo contributor: Anne W. Lucky, MD.)

HERPETIC GINGIVOSTOMATITIS

Clinical Summary

Herpetic gingivostomatitis is primary infection caused by HSV seen in up to 30% of children between 6 months and 5 years of age. Patients usually present with approximately 4 days of fever, malaise, decreased oral intake, cervical adenopathy, and pain in the mouth and throat. Following the prodrome, vesicular and ulcerative lesions appear throughout the oral cavity. The gingiva becomes very friable and inflamed, especially around the alveolar rim. Increased salivation, foul breath, and cervical lymphadenitis may be present. Although fever resolves in 3 to 5 days, children may have difficulty eating for 7 to 14 days. Lesions may last for up to 21 days in severe cases. Autoinoculation may produce vesicular lesions on the fingers (herpetic whitlow).

Management and Disposition

Treatment includes pain control, hydration, and consideration of oral acyclovir therapy. The pain may be significant and often requires oral narcotic pain medications. Control of the pain will allow the patient to consume fluids and remain well hydrated. Acyclovir has been shown to reduce the duration of pain, gingival swelling, oral lesions, fever, and viral shedding if initiated within the first 72 to 96 hours of illness. Avoidance of citrus juices or spicy food is recommended. Cold clear fluids, popsicles, and ice cream may be useful in small children. Not infrequently, admission for IV hydration is necessary. Topical pain control may be achieved by using mixtures of antihistamine (diphenhydramine elixir) and antacid (1:1) applied to lesions with a cotton swab. Local application of viscous lidocaine should be avoided in children, since patients may develop toxic serum levels due to altered absorption from inflamed oral mucosa leading to seizures or methemoglobinemia.



FIGURE 14.45 ■ Herpetic Gingivostomatitis. Multiple oral vesicular lesions and tongue ulcerations consistent with herpes gingivostomatitis. Vesicular lesions from autoinoculation are present on the finger (herpetic whitlow). (Photo contributor: Michael J. Nowicki, MD.)

Pearls

1. Most lesions are in the anterior two-thirds of the oral cavity. Posterior lesions sparing the gingiva are most commonly seen in coxsackievirus infections.
2. Primary HSV infection in childhood is usually asymptomatic.
3. After primary oral infection, HSV remains latent in the trigeminal ganglion until reactivation as herpes labialis.



FIGURE 14.46 ■ Herpetic Stomatitis. Multiple perioral vesicular lesions consistent with herpes gingivostomatitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.47 ■ Herpetic Gingivostomatitis. Multiple oral vesicular lesions and tongue ulcerations consistent with herpes gingivostomatitis. (Photo contributor: Scott Pangonis, MD.)

MENINGOCOCCEMIA

Clinical Summary

Meningococemia is an acute febrile illness caused by *Neisseria meningitidis* bacteremia characterized by its generally rapid onset, significant toxicity, and petechial rash involving the skin and mucous membranes. The petechiae progress to become palpable purpura and may coalesce to become purpura fulminans. It progresses rapidly to decompensated septic shock with hypotension and multisystem organ failure. In cases of fulminant disease, progressive shock is accompanied by disseminated intravascular coagulation and massive mucosal hemorrhages. Prodromal symptoms may include cough, coryza, headache, neck pain, and malaise. Children less than 5 years of age and college students who did not receive the meningococcal vaccine during high school are at greatest risk.



FIGURE 14.48 ■ Meningococemia. Diffuse petechiae and purpura in a lethal meningococcal infection. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.49 ■ Meningococcemia. Diffuse petechiae in a patient with meningococcemia. (Photo contributor: Richard Strait, MD.)



FIGURE 14.50 ■ Meningococcemia. Petechiae and purpura in an adolescent patient with meningococcemia. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.51 ■ Meningococcemia. Purpura are seen in this patient with meningococcemia. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.52 ■ Meningococcemia. Subtle petechiae and purpura on the soles of the feet in a patient with meningococcemia. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.53 ■ Meningococemia. Subtle periorbital petechiae and purpura in a patient with meningococemia. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

In stable patients in whom meningococemia is in the differential diagnosis, obtain cultures of blood and spinal fluid and from the nasopharynx, along with a CBC and coagulation studies. Consider bedside screening studies (eg, blood gas to assess acid-base status), lactate, liver function tests, and other studies as clinically indicated. These patients should be admitted for intensive monitoring to institutions capable of delivering critical care services. Broad-spectrum parenteral antibiotics should be administered initially until the organism is identified and sensitivities are available as with any patient with suspected sepsis. In the unstable septic patient, support end organ perfusion and oxygenation via early goal-directed therapy. Hemo-dynamic monitoring and blood pressure support (fluids and vasoactive drugs) are of paramount importance. Peripheral and central venous catheters and urinary and arterial catheters are usually necessary for optimal management of these patients.

Pearls

1. Skin scrapings of the purpuric lesion can be cultured and microscopically examined for the presence of gram-negative diplococci, although a negative

result does not exclude the diagnosis of meningococemia.

2. A quadrivalent meningococcal conjugate vaccine (sero-groups A, C, W, and Y) is now available and recommended for all adolescents between ages 11 and 12 and then again at age 15 years or high school entry (whichever comes first).
3. Prophylaxis for all close contacts is recommended with rifampin for 48 hours or a single dose of ceftriaxone or ciprofloxacin.
4. If meningococcal meningitis or meningococemia is likely, antibiotic therapy should not be delayed to perform a lumbar puncture.

SUMMER PENILE SYNDROME

Clinical Summary

Summer penile syndrome is a benign hypersensitivity reaction involving the skin of the penile shaft. As the name suggests, it is seen during the warm weather months. It is also known as seasonal acute hypersensitivity reaction and lion's mane penis. The history may include recent play outside in the grass or a wooded area within the last 24 hours. It is most commonly associated with insect bites, usually chiggers, but may also be caused by exposure to plants (poison ivy, sumac, and oak). The diagnosis is made clinically and is suspected when the skin of the shaft of the penis, most often just proximal to the glans, is markedly edematous. There is minimal erythema and no fluctuance. Four out of five patients have pruritus, and few have urinary symptoms. Symptoms (both swelling and pruritus) can last up to 2 to 3 weeks, but most patients have resolution in 4 to 5 days. The differential diagnosis includes trauma, nephrotic syndrome, HSP, balanitis, phimosis, paraphimosis, and priapism.

Management and Disposition

Treatment consists of an oral antihistamine if the patient has pruritus and cool compresses. Systemic corticosteroids should only be considered if the child is in significant pain or is having trouble with the voiding stream. Patients can be discharged home after education and reassurance. Urology involvement and referral is usually not necessary.

Pearls

1. Summer penile syndrome is usually painless.
2. Pruritus is the most common symptom.
3. Testing is unnecessary, but the diagnosis must be distinguished from paraphimosis.



FIGURE 14.54 ■ Summer Penile Syndrome. Edema of the distal penile shaft and left scrotum. An insect bite is seen at the base of the left mons pubis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.55 ■ Summer Penile Syndrome. Also known as “lion’s mane penis” due to the marked circumferential edema just proximal to the glans. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 14.56 ■ Summer Penile Syndrome. Swelling and ecchymosis after insect bite in an uncircumcised male with summer penile syndrome. (Photo contributor: Lawrence B. Stack, MD.)

STAPHYLOCOCCAL SCALDED SKIN SYNDROME

Clinical Summary

Staphylococcal scalded skin syndrome (Ritter disease) most commonly affects infants and children less than 5 years of age and is caused by an exfoliative exotoxin-producing strain of *S aureus*. Initial presentation includes fever, malaise, and irritability following an upper respiratory infection with pharyngitis or conjunctivitis. Patients first develop a diffuse faint blanching erythematous rash that is tender to the touch. Crusting around the mouth, eyes, and neck may be seen. Within 2 to 3 days, flaccid blisters and bullae develop, especially in flexor creases and the buttocks, hands, and feet. These bullae are sterile. In some patients, widespread desquamation occurs. The differential diagnosis includes toxic epidermal necrolysis, exfoliative erythroderma, bullous erythema multiforme, bullous pemphigoid, bullous impetigo, sunburn, enterovirus, drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome, acute mercury poisoning, toxic shock syndrome, and epidermolysis bullosa.

Management and Disposition

If staphylococcal scalded skin syndrome is suspected, obtain cultures from the blood, urine, nasopharynx, abnormal area of skin, or other suspected foci of infection. Treatment is directed to eradicate *S aureus*, thus terminating the production of toxin. Semisynthetic penicillinase-resistant penicillins or clindamycin should be used intravenously. Consider the addition of vancomycin in areas with a high prevalence of MRSA. Admission is usually necessary, especially in young infants. This age group requires careful attention to fluid and electrolyte losses and the prevention of secondary infection of the denuded skin. Management of pain associated with the rash may require opioid analgesics. Children may develop a 2nd period of desquamation during the first 10 days of the illness.

Pearls

1. The wrinkling or peeling of the upper layer of the epidermis (pressure applied with a Q-tip or gloved finger) that occurs within 2 or 3 days of the onset of this illness is known as Nikolsky sign.

2. The fluid in the bullae of staphylococcal scalded skin syndrome is sterile. The toxin is produced at a remote site and delivered to the skin via the bloodstream.
3. Infants with large body surface area involvement are at risk for hypothermia and fluid/electrolyte losses.
4. Corticosteroids are contraindicated in the treatment of staphylococcal scalded skin syndrome.



FIGURE 14.57 ■ Staphylococcal Scalded Skin Syndrome. Toddler with diffuse macular peeling eruption consistent with scalded skin syndrome from *S aureus*. (Photo contributor: Judith C. Bausher, MD.)

SCARLET FEVER (SCARLATINA)

Clinical Summary

Scarlet fever manifests as diffuse blanching “sandpaper-like” erythematous macules and papules caused by erythrogenic toxin production from group A β -hemolytic *Streptococcus* pharyngitis. Occasionally, the site of infection is skin (impetigo) or perianal. The disease usually occurs in children 2 to 10 years of age. The typical presentation of scarlet fever includes fever, headache, sore throat, nausea, vomiting, and malaise followed by the characteristic

scarlatiniform rash. The rash initially occurs on the groin and trunk, spreading to the face (often with perioral sparing) and neck, then quickly becomes generalized. Desquamation occurs after 5 to 7 days. On the tongue, a thick, white coat and swollen papillae may be seen (“strawberry tongue”). Palatal petechiae and tender anterior cervical lymph-adenopathy may be present. The gold standard for diagnosis is a positive throat culture of a swab from the tonsillar pillars, though rapid antigen testing is highly specific and can provide prompt diagnosis. The differential diagnosis includes enteroviral infections, staphylococcal scalded skin syndrome, viral hepatitis, infectious mononucleosis, toxic shock syndrome, drug eruptions, rubella, mercury poisoning, and Kawasaki disease.

Management and Disposition

Penicillin, either a single intramuscular dose of benzathine penicillin G or oral amoxicillin for 10 days, is the treatment of choice. Alternatives include erythromycin or clindamycin in penicillin-allergic patients.

Pearls

1. In scarlet fever, petechiae in a linear pattern along the major skin folds in the axillae and antecubital fossae are known as “Pastia lines.”
2. In dark-skinned individuals, the rash may be difficult to differentiate and may consist only of punctate papular elevations called “goose flesh.”
3. There has never been an isolate of group A *Streptococcus* that is resistant to penicillin.
4. Scarletina is a rash that spares the palms and soles, although desquamation can occur in those areas.



FIGURE 14.58 ■ Scarletina. Erythematous scarlatiniform rash of scarlet fever. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.59 ■ Goose Flesh. This sandpaper rash started 2 days after sore throat and fever began in this 6-year-old. The grouping of the fine papules gives the skin a “goose flesh” texture in darker skin colors. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.60 ■ Palatal Petechiae. Petechiae present on the posterior soft palate of a child with group A streptococcal infection. (Photo contributor: Hannah F. Smitherman, MD.)



FIGURE 14.61 ■ Pastia Lines. Confluent petechiae in a linear pattern in the antecubital fossa consistent with Pastia lines are seen in these patients with scarlet fever. **(A)** The forearm on the right belongs to the patient's sister and does not show Pastia lines. **(B)** Pastia lines in a Caucasian patient. A classic sandpaper rash is also evident on the arm and trunk. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 14.62 ■ Group A β -Hemolytic *Streptococcus* Toxic Shock Syndrome. Nine-year-old girl with strawberry tongue, nausea, vomiting, fever, and sandpaper rash over trunk, arms, and legs for 4 days suggestive of toxic shock syndrome. Vital signs on presentation: heart rate: 152; blood pressure: 84/55 mm Hg. (Photo contributor: Eric M. Sergienko, MD.)



FIGURE 14.63 ■ Desquamating Rash. Desquamation of the fingertips occurred as the scarlatiniform rash began to fade. Desquamation also occurred in the groin and toes. (Photo contributor: Clay. B. Smith, MD.)



FIGURE 14.64 ■ Perianal Strep Desquamation. Desquamating rash in the perineum of a child with GABHS infection. (Photo contributor: Kevin J. Knoop, MD, MS.)

BLISTERING DISTAL DACTYLITIS

Clinical Summary

Blistering distal dactylitis is a cellulitis of the fingertip caused by group A β -hemolytic *Streptococcus* or, less often, *S aureus* infection in children from infancy to teenage years. The typical lesion is a seropurulent, fluid-filled, painful, tense blister with surrounding erythema located over the palmar fat pad on the distal portion of a finger or toe. Polymorphonuclear leukocytes and gram-positive cocci can be found in the Gram stain of the purulent exudate from the lesion. The differential diagnosis includes bullous impetigo, burns, friction blisters, paronychia, felon, and herpetic whitlow.



FIGURE 14.65 ■ Blistering Distal Dactylitis. Blistering rash of the distal fingers with surrounding erythema typically caused by *Streptococcus*. Note the location of the rash over the volar finger pads. (Photo contributor: Anne W. Lucky, MD.)

Management and Disposition

There is usually a rapid response to incision and drainage of the blister and an appropriate course of oral antibiotic therapy. Consider use of agents active against MRSA (clindamycin or trimethoprim-sulfamethoxazole) if there is a high community prevalence.

Pearls

1. Nonpurulent vesicular lesions that become confluent multi-locular bullae are characteristic of herpetic whitlow, which should not be drained, and help distinguish it from blistering distal dactylitis.
2. Topical antibiotics are not recommended.



FIGURE 14.66 ■ Blistering Distal Dactylitis. Inflammation and fluid-filled vesicles in a child with blistering dactylitis. (Photo contributor: Lawrence B. Stack, MD.)

HENOCH-SCHÖNLEIN PURPURA (IGA VASCULITIS)

Clinical Summary

Henoch-Schönlein purpura (HSP) is the most common systemic vasculitis in children, with a peak incidence between ages 3 and 15 years and with 90% of patients younger than age 10. It is characterized by four main clinical manifestations. The classic **exanthem** of HSP begins with erythematous macules or urticaria that eventually coalesce and evolve into ecchymotic lesions and palpable purpura. The lesions are more often located on the buttocks and

gravity-dependent areas (lower extremities) in ambulatory children. In nonambulatory children, the lesions can be seen on the face, trunk, and upper extremities as well. Mucosal involvement is rare; however, edema of the scalp, hands, scrotum, and periorbital tissue occurs.

Migratory oligoarticular (one to four joints) **arthritis** and **arthralgia** are eventually seen in 75% of patients. **Gastrointestinal symptoms** (colicky abdominal pain, occult or gross blood in the stool, and small bowel–small bowel intussusception) usually develop within a week of the rash, but may precede it. **Renal involvement** is the most frequent serious complication and usually occurs during the 1st month. It commonly manifests as microscopic hematuria and may progress to glomerulonephritis. One percent progress to end-stage renal disease. Hypertension is uncommon.

Diagnosis is made by history and clinical examination. Most laboratory tests are usually nonspecific except for the urinalysis, which may be positive for blood or protein in 50%. The overall prognosis is excellent, with full recovery in most. The course is marked by relapses and remissions in 50% (the rash tends to recur within the first 6 weeks). Prognosis is linked to renal involvement. The rash may be confused with drug reactions, erythema multiforme, urticaria, and even physical abuse. Consider other causes of purpura, such as bleeding disorders or infection (meningococemia).

Management and Disposition

Treatment is supportive. Patients with HSP limited to the skin and joints with minimal renal involvement can be managed as outpatients. Hospitalization is warranted when patients cannot maintain oral intake; have severe abdominal pain, GI hemorrhage, and intussusception; are unable to ambulate or care for themselves because of significant joint pain; or have evidence of renal injury (renal failure, nephritis, nephrotic range proteinuria, or significant hypertension for age). Ibuprofen or naproxen can be effective for symptomatic pain relief, although providers should be cautious in patients with GI bleeding or glomerulonephritis. Corticosteroids may shorten the duration of abdominal pain in patients with severe symptoms who are unable to maintain oral intake or require admission to the hospital, but it will not prevent complications of HSP. In rare cases where ileocolic intussusception is diagnosed, air contrast enema and prompt surgical consultation are indicated. In contrast to ileocolic intussusception, small bowel–small bowel intussusception generally resolves spontaneously and rarely requires invasive intervention.

Pearls

1. Obtain a stool occult blood test and urinalysis in patients with abdominal pain and suspected HSP. The fecal occult blood test is positive in more than half.
2. Intussusception associated with HSP is seen in 2% of patients, most commonly in boys, particularly those about 6 years of age. The intussusception is often ileo-ileal.
3. The typical rash occurs in nearly 100% of patients and is the presenting feature in at least 50%. Joint symptoms may precede the rash as the presenting complaint in 25% of patients. Ankles and knees are the most commonly affected joints.
4. Scrotal swelling and pain may be the initial presenting sign in boys and can mimic testicular torsion.



FIGURE 14.67 ■ Henoch-Schönlein Purpura. These erythematous, hemorrhagic papules and petechiae in a symmetric acral distribution are classic findings in HSP. This child presented with no other symptoms.

(Photo contributor: Lawrence B. Stack, MD.)

SERUM SICKNESS–LIKE REACTION

Clinical Summary

Serum sickness–like reaction is characterized by rash, fever, angioedema of the face, hands, and feet, and polyarthralgias/polyarthritis, with onset 1 to 2 weeks following exposure to an offending agent, and resolves within 1 to 2 weeks after exposure is discontinued. It is felt to occur via an immune complex–mediated mechanism and can be precipitated by exposure to a number of drugs, with trimethoprim-sulfamethoxazole, cephalosporins, and penicillins being most common. Almost all patients develop a polymorphous pruritic rash that starts in the trunk, groin, and axillae, eventually spreading to the limbs. The urticarial lesions are longer lasting than typical hives. Some patients can exhibit palpable purpura, maculopapular lesions, or target lesions reminiscent of erythema multiforme but without evidence of blister formation. In all patients, the mucous membranes are spared. Almost all patients develop remittent fever without temporal spikes. Other symptoms include arthralgias, angioedema, and rarely, frank arthritis. The differential diagnosis includes viral exanthems, hypersensitivity vasculitis, scarlet fever, acute rheumatic fever, meningococemia, disseminated gonococemia, reactive arthritis, Lyme disease, Still disease, and Stevens-Johnson syndrome.

Management and Disposition

The diagnosis is clinical and based on history of exposure to a potential offending agent. Treatment consists of discontinuing the causative agent and supportive care. Scheduled antihistamines and nonsteroidal anti-inflammatory drugs (NSAIDs) may bring relief. Addition of H₂ blockers may provide additional relief from angioedema. Glucocorticoids are sometimes used for patients with severe symptoms. Patients who are ill-appearing or do not have a history of a potential offending agent should have a CBC with differential, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), urinalysis, blood urea nitrogen (BUN), creatinine, serum electrolytes, urinalysis, and blood culture obtained to exclude other inflammatory or infectious causes.

Pearls

1. Serum sickness–like reaction is a self-limited syndrome that does not progress to mucous membrane involvement. Treatment is supportive and includes discontinuing suspected medications and administering antihistamines and NSAIDs.
2. The etiology of serum sickness–like reactions includes medications, and it frequently occurs at the end or after completion of the course of medications.
3. Due to the delayed nature of the syndrome, a causative drug may have been discontinued 1 to 3 days prior to the onset of symptoms.
4. Future consultation with an allergy specialist is recommended.



FIGURE 14.68 ■ Serum Sickness–Like Reaction. Fever, joint pain, and erythematous plaques to face (**A**) and legs (**B**) 14 days after receiving amoxicillin in a 7-year-old girl. These clinical findings suggest serum sickness. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.69 ■ Serum Sickness–Like Reaction. Fever, joint pain, ankle swelling, and erythematous plaques seen here in an infant 11 days after taking Ceclor, suggesting serum sickness. (Photo contributor: Lawrence B. Stack, MD.)

SUPERFICIAL (STRAWBERRY) HEMANGIOMA

Clinical Summary

Hemangiomas are benign vascular tumors characterized by a rapid proliferative phase followed by a spontaneous involutional phase. They are the most common soft-tissue tumors of infancy. The appearance of hemangiomas is defined by the lesion's depth, location, and stage of evolution. A superficial hemangioma lies in the upper dermis and often originates as an erythematous macular patch, a pale macule, or a localized telangiectasia with a pale halo. The lesion grows and becomes vascularized during the first 2 months of life. The classic presentation is a bright red, slightly raised, noncompressible plaque. It commonly regresses by 2 to 3 years of age but may persist throughout life. Hemangiomas can also affect the airway, eyes, and liver or cause high-output cardiac failure if sufficiently large. The most important local complication is ulceration, which can be exquisitely painful. The differential diagnosis includes vascular malformations, malignant vascular neoplasms, pyogenic granulomas, and giant melanocytic birthmarks.



FIGURE 14.70 ■ Strawberry Hemangioma. Raised umbilicated vascular lesion on the left upper chest consistent with strawberry hemangioma. (Photo contributors: Kara Shah, MD, PhD, and Katharine Hanlon.)

Management and Disposition

Most cases require serial observation as superficial hemangiomas usually regress without residual problems. Treatment of hemangiomas is indicated when there is an obstruction of a vital orifice (ie, airway, mouth, or nares), lesion in the peri-orbital region, or lesion in the GI tract or airway, or if hematologic or cardiovascular complications are present. Education and parental reassurance are

important for small hemangiomas because there is great pressure to treat for cosmetic reasons. There is growing evidence supporting the use of topical β -blockers for superficial, uncomplicated hemangiomas to expedite resolution. For more complicated hemangiomas, first-line therapy is propranolol as it inhibits the growth and induces regression. Infants should undergo a pretreatment consultation with a pediatric dermatologist or vascular tumor specialist to determine eligibility and risk of starting β -blocker therapy. Due to the risk of hypoglycemia and hemodynamic changes, many higher risk infants are admitted for close monitoring when initiating propranolol therapy. Topical and systemic corticosteroids were the mainstay of therapy prior to the use of β -blockers and are still beneficial in children in whom β -blockers are contraindicated.

Pearls

1. Kasabach-Merritt phenomena (hemolytic anemia, thrombocytopenia, and coagulopathy) is associated with Kaposi-form hemangioendothelioma or tufted angiomas and not with common hemangiomas as was previously thought.
2. As many as 20% of affected infants have multiple lesions.
3. In contrast to hemangiomas, vascular malformations, such as port-wine stains, do not proliferate or involute. They persist throughout life and grow in proportion with the child.



FIGURE 14.71 ■ Strawberry Hemangioma. Vascular lesion on the nipple. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.72 ■ Strawberry Hemangioma. Vascular lesion on the upper lip consistent with strawberry hemangioma. (Photo contributors: Kara Shah, MD, PhD, and Katharine Hanlon.)



FIGURE 14.73 ■ Hemangioma. Large hemangioma beginning to show involution as a result of treatment. (Photo contributor: Lawrence B. Stack, MD.)

4. Children with a segmental distribution hemangioma of the scalp, trunk, upper extremity, or most commonly the face are at risk for PHACE syndrome (**p**osterior fossa anomalies, **h**emangioma, **a**rterial anomalies, **c**ardiac anomalies, and **e**ye anomalies) and require further screening through a multi-disciplinary team.

ORBITAL AND PRESEPTAL (PERIORBITAL) CELLULITIS

Clinical Summary

Orbital cellulitis is a serious bacterial infection involving the fat and muscle within the orbit characterized by fever, painful purple-red eyelid swelling, ophthalmoplegia, pain with extra-ocular movements, proptosis, and variable decreased visual acuity. It may begin with eye pain and fever. In general, it is caused by *Streptococcus anginosus*, *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*, and *S aureus*. It usually arises as a complication of ethmoid or maxillary sinusitis. If not treated promptly, it can lead to blindness, cavernous venous sinus thrombosis, meningitis, subdural empyema, or brain abscess.

Preseptal (periorbital) cellulitis is much more common and involves the structures anterior to the orbital septum. It usually presents with edema and circumferential erythema of the eyelids and periorbital skin, fever, and minimal pain. Proptosis and ophthalmoplegia are not characteristic as it does not involve the orbit or other ocular structures. Preseptal cellulitis usually results from sinusitis or contiguous infection due to local skin trauma, insect bite, or hordeolum. Common organisms are *S aureus* and group A *Streptococcus*.



FIGURE 14.74 ■ Orbital Cellulitis. Eye redness, swelling, purulent drainage, and mild entrapment are seen in this patient with painful orbital cellulitis. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.75 ■ Orbital Cellulitis. Right ethmoid sinus with subperiosteal abscess and extension into the orbital space seen in the patient in Figure 14.74. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.76 ■ Orbital Cellulitis. CT confirmed orbital cellulitis in this 2-month-old. (Photo contributor: Lawrence B. Stack, MD.)

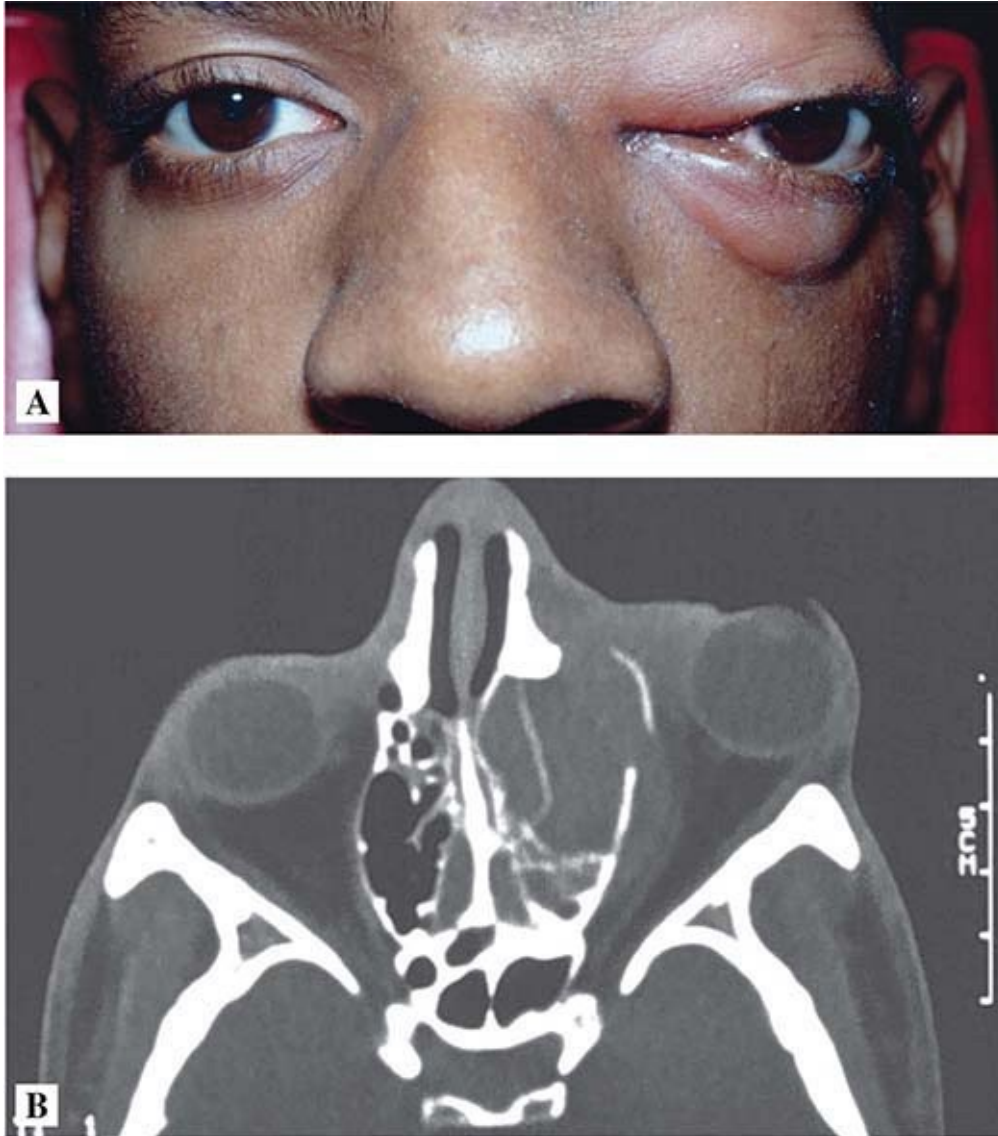


FIGURE 14.77 ■ Orbital Cellulitis. Left ethmoid sinusitis with extension into the orbital space, periosteal abscess formation, and proptosis is seen in this patient (**A**) and on CT (**B**). (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.78 ■ Preseptal Cellulitis. Periorbital cellulitis with abscess formation seen in this patient (**A**) and on CT (**B**). (Photo contributor: Eftitan Akam, MD.)



FIGURE 14.79 ■ Preseptal Cellulitis. Cellulitis originating from local skin trauma in the left eyebrow.
(Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.80 ■ Preseptal Cellulitis. Preseptal cellulitis extending from a hordeolum. (Photo contributor: David Effron, MD.)

Management and Disposition

Broad-spectrum IV antibiotics with methicillin-resistant staphylococcal coverage, ophthalmologic consultation, and admission are indicated in cases of orbital cellulitis. Orbital CT with contrast is needed to assess for abscess requiring surgical drainage and confirms orbital cellulitis. In febrile or ill-appearing patients with preseptal cellulitis, admission with broad-spectrum IV antibiotic therapy is also indicated. Adults and children older than 1 year with mild cases (especially those with a history of trauma such as abrasion or insect bite or sting) can be treated with outpatient oral antibiotic therapy with close follow-up. Consider the use of antimicrobials with MRSA coverage as the prevalence of community-acquired MRSA is high.

Pearl

1. Obtain orbital and sinus CT if the diagnosis is uncertain and in patients who have pain with eye movement or vision changes or if unable to assess vision

(usually age < 1 year).



FIGURE 14.81 ■ Preseptal Cellulitis. Left periorbital cellulitis with edema and erythema of the eyelids in a nontoxic cooperative toddler with a normal ocular exam. (Photo contributor: Kevin J. Knoop, MD, MS.)

BRANCHIAL CLEFT CYST

Clinical Summary

A branchial cleft cyst arises from the incomplete obliteration of one of the four branchial clefts during embryogenesis. As obliteration of the clefts occurs, a portion may remain, forming a cystic space with an epithelial lining and no connection to the skin or pharynx. The anatomic location of a branchial cleft cyst depends on the specific arch/cleft involved. Involvement of the 1st cleft may result in a cyst in the region of the parotid gland, the preauricular or postauricular area, or inferior to the angle of the mandible. Second cleft anomalies represent 70% to 90% of cysts and may be found along the anterior border of or deep to the sternocleidomastoid muscle, in the vicinity of the carotid arteries. Third and 4th arch/cleft anomalies are rare. A cyst usually presents clinically as a lateral, tender neck mass due to acute infection and enlargement usually in association with an upper respiratory infection.

Management and Disposition

Diagnosis is suggested by history and location of physical examination findings. A CT scan or magnetic resonance imaging may help to define the extent of the lesion. Definitive diagnosis comes from pathologic examination after surgical excision. Treatment includes antibiotics if there is an associated infection followed by complete surgical excision by a specialist in order to prevent recurrence and because these lesions may be intimately involved with major vessels and nerves.

Pearl

1. Branchial cleft anomalies are second only to thyroglossal duct cysts in frequency of congenital head and neck lesions in children.



FIGURE 14.82 ■ First Branchial Cleft Cyst. Periauricular mass from a first brachial cleft cyst. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.83 ■ Second Branchial Cleft Cyst. Right neck mass seen in second branchial cleft cyst.
(Photo contributor: Scott Manning, MD.)

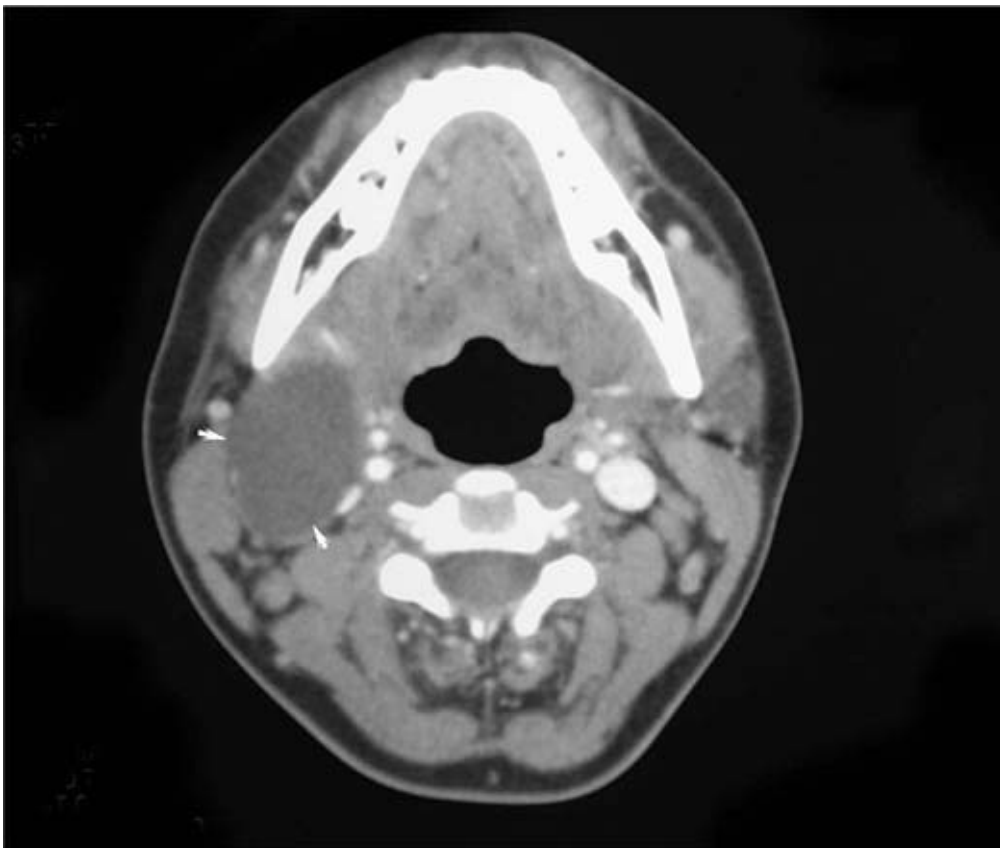


FIGURE 14.84 ■ Second Branchial Cleft Cyst. Neck CT showing second branchial cleft cyst. (Photo contributor: Scott Manning, MD.)

THYROGLOSSAL DUCT CYST

Clinical Summary

A thyroglossal duct cyst arises from the incomplete obliteration of the thyroglossal duct during fetal development. It usually presents as a painless, midline, anterior neck mass that moves with swallowing and protrusion of the tongue. It may occur anywhere from the base of the tongue to the sternal notch but is usually located at or below the hyoid bone adjacent to the thyrohyoid membrane. These cysts may rapidly enlarge if infected, which often occurs in association with upper respiratory symptoms.

Management and Disposition

The diagnosis of this lesion is suggested by history and physical examination. Antibiotics are indicated if the lesion has rapidly enlarged due to infection. Common pathogens include *H influenzae*, *S aureus*, and *Staphylococcus epidermidis*. Treatment involves complete surgical excision of the cyst and tract (Sistrunk procedure) following resolution of any associated infection. Referral to an otolaryngologist is appropriate. The differential diagnosis includes a dermoid cyst.



FIGURE 14.85 ■ Thyroglossal Duct Cyst. A midline mass is seen in thyroglossal duct cyst. (Photo contributor: Lawrence B. Stack, MD.)

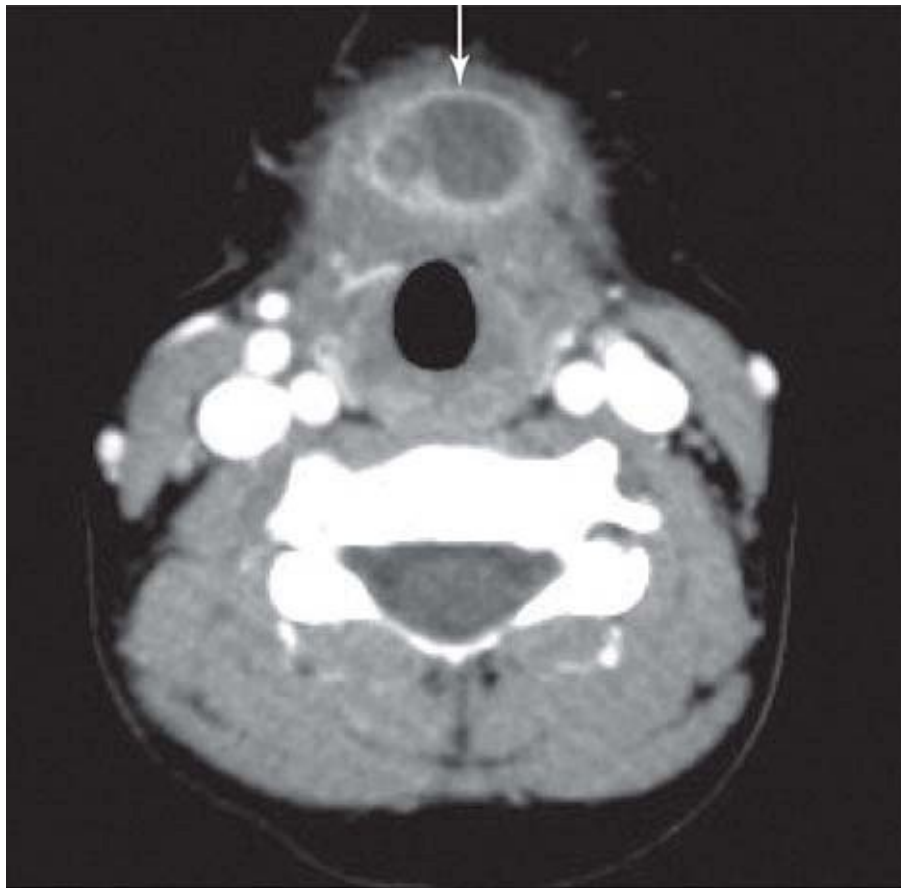


FIGURE 14.86 ■ Thyroglossal Duct Cyst. CT of the neck showing anterior thyroglossal duct cyst. (Photo

contributor: Lawrence B. Stack, MD.)

Pearls

1. A thyroglossal duct cyst is the most frequent congenital head and neck lesion in children.
2. In approximately 1% of patients with this lesion, the only functional thyroid tissue is located within the cyst. Therefore, patients should be screened by history for symptoms of hypothyroidism. If symptoms are present, a serum thyroid-stimulating hormone should be sent, and ultrasound of the midline neck should be considered prior to surgical removal.
3. The cyst typically moves up with protrusion of the tongue or with swallowing due to its relationship with the hyoid bone and larynx.



FIGURE 14.87 ■ Thyroglossal Duct Cyst. Lateral view of thyroglossal duct cyst. (Photo contributor: Lawrence B. Stack, MD.)

CYSTIC HYGROMA

Clinical Summary

Cystic hygromas are congenital lymphatic malformations found most commonly

in the neck in infants and children less than 2 years of age. They present as nontender, compressible, unilocular, or multilocular masses with thin, transparent walls filled with straw-colored fluid. Unlike hemangiomas, these lesions rarely undergo spontaneous involution. The vast majority tend to grow and infiltrate adjacent structures. In cases where the tongue is involved, they may lead to upper airway obstruction. The differential diagnosis includes branchial arch remnants, thyroglossal duct cysts, cystic teratomas, cervical lymphadenopathy, and other primary neoplastic diseases.

Management and Disposition

Elective surgical removal is the treatment of choice in the vast majority of cases and especially in emergent situations, since these lesions do not regress and may compress local tissues. Extent of the lesion should be evaluated through further imaging prior to its removal. The earlier these lesions can be removed, the better is the cosmetic result. Aspiration of the lesion, with or without injection of a sclerosing agent, is another therapeutic option.



FIGURE 14.88 ■ Cystic Hygroma. A bright, supraclavicular, soft, boggy, compressible mass consistent

with cystic hygroma. (Photo contributor: Richard M. Ruddy, MD.)

Pearls

1. Rapid enlargement of a cystic hygroma is most likely due to bleeding or infection. This can result in airway compromise.
2. Chromosomal abnormalities are found in a significant number of infants with cystic hygromas. These lesions are frequently associated with Noonan, Turner, and Down syndromes.

CAT-SCRATCH DISEASE

Clinical Summary

Cat-scratch disease is a generally benign, self-limited condition caused primarily by *Bartonella henselae* that usually manifests with regional lymphadenopathy, although visceral organ, neurologic, or ocular involvement can occur. A history of contact with saliva or scratch from a cat (especially kittens with fleas) is usually present. Cat-scratch disease typically starts with an inoculation lesion, which sequentially will appear vesicular, erythematous, and then papular. Lymphadenopathy near the site of inoculation usually appears within 2 weeks of skin inoculation and may persist for months. In rare cases, patients may develop complications such as ocular disease (neuroretinitis and Parinaud oculoglandular syndrome), encephalitis, osteolytic lesions, hepatosplenic lesions, weight loss, prolonged fever, and fatigue. The differential diagnosis includes lymphogranuloma venereum, bacterial adenitis, sarcoidosis, infectious mononucleosis, tumors (benign or malignant), tuberculosis, tularemia, brucellosis, and histoplasmosis.

Management and Disposition

The disease is usually self-limited, and management is primarily symptomatic. Parents and patients should be reassured that the nodes are benign and frequently resolve within 2 to 4 months. Although self-resolving, treatment with antimicrobials has been shown to decrease the duration of symptoms and is recommended. The antibiotic of choice is azithromycin. Other options include

clarithromycin, trimethoprim-sulfamethoxazole, rifampin, or ciprofloxacin. Patients with hepatosplenic, neurologic, or neuroretinal disease should be treated with parenteral antibiotics (rifampin plus gentamicin or azithromycin). If the diagnosis is in doubt, serologic assays for *Bartonella* species can be sent. Surgical excision of the affected nodes is generally unnecessary.

Pearls

1. Cat-scratch disease is the most common cause of regional adenopathy and should be considered in all children or adolescents with persistent lymphadenopathy.



FIGURE 14.89 ■ Cat-scratch Disease. An erythematous, tender, suppurative node is seen in a young febrile patient with a history of cat scratch on the extremity. The node required drainage 2 days later. (Photo contributor: Kevin J. Knoop, MD, MS.)

2. Parinaud oculoglandular syndrome is characterized by a unilateral conjunctivitis and preauricular lymphadenopathy caused by *B henselae*.
3. Even in the presence of severe and multiple hepatic lesions, liver transaminase levels are normal and hepatomegaly is rare.
4. *B henselae* is a rare cause of unilateral neuroretinitis and vision loss.
5. In cases treated with antibiotics, a Jarisch-Herxheimer reaction may occur, which manifests as fever, tachycardia, hyperventilation, hypotension,

peripheral vasodilation, diffuse myalgias, and exacerbation of skin lesions.



FIGURE 14.90 ■ Cat-scratch Disease. The precipitating wound that caused the suppurative node in [Fig. 14.89](#). (Photo contributor: Kevin J. Knoop, MD, MS.)

General Conditions

EPIGLOTTITIS

Clinical Summary

Epiglottitis (also known as supraglottitis) is a life-threatening condition characterized by sudden onset of fever, irritability, sore throat, moderate to severe respiratory distress with stridor, and variable degrees of drooling. It results from a cellulitis of the epiglottis, aryepiglottic folds, and adjacent tissues. The patient generally appears toxic and prefers a sitting position, leaning forward with the neck extended in a sniffing position with an open mouth. With the addition of the *H influenzae* type B vaccine to the routine immunization schedule, there has been a dramatic decrease in the incidence of epiglottitis as well as a shift in the bacterial etiology. Although *H influenzae* type B is still the most common cause, many cases are now caused by nontypeable *H influenzae*, streptococci, staphylococci (especially MRSA), and *Candida albicans*. Adults

typically have a more indolent course characterized by severe sore throat and odynophagia. Direct thermal injury has been reported as a noninfectious cause. On soft-tissue lateral neck x-ray, the epiglottis is seen as rounded and blurred (thumbprint sign). Epiglottitis may progress to complete upper airway obstruction if not treated. Differential diagnosis includes acute infectious laryngitis, acute laryngotracheobronchitis (croup), acute spasmodic laryngitis, membranous (bacterial) tracheitis, anaphylactic reaction, foreign-body aspiration, retropharyngeal abscess, and extrinsic or intrinsic compression of the airway (tumors, trauma, cysts).



FIGURE 14.91 ■ Epiglottitis. Lateral soft-tissue x-ray of the neck demonstrating thickening of aryepiglottic folds and thumbprint sign of epiglottis. (Photo contributor: Richard M. Ruddy, MD.)

Management and Disposition

Immediate intervention is required. If epiglottitis is suspected, the child should be allowed to remain in a position of comfort if they are maintaining an adequate airway. If impending respiratory failure is present, an airway must be established. If possible, this should be done in the operating room or designated area where advanced airway management with sedation is available. An experienced anesthesiologist and surgeon should be readily available in case a surgical airway is necessary. Once the airway has been controlled, the patient should be sedated to avoid unplanned extubation. Parenteral antibiotic therapy should be initiated with ceftriaxone or cefotaxime. Antistaphylococcal coverage against MRSA with clindamycin or vancomycin is also indicated, and therapy can be adjusted once culture results are available.

Pearls

1. Definitive diagnosis of epiglottitis requires direct visualization of a red, swollen epiglottis, preferably in an operating room with advanced airway measures readily available.
2. Allow the child to remain undisturbed in a position of comfort while preparing for airway management. An agitated child is at increased risk for sudden, complete upper airway obstruction.
3. *H influenzae* type B can still cause epiglottitis even in immunized children.
4. The mean age of epiglottitis is increasing since the introduction of the *H influenzae* type B vaccine.

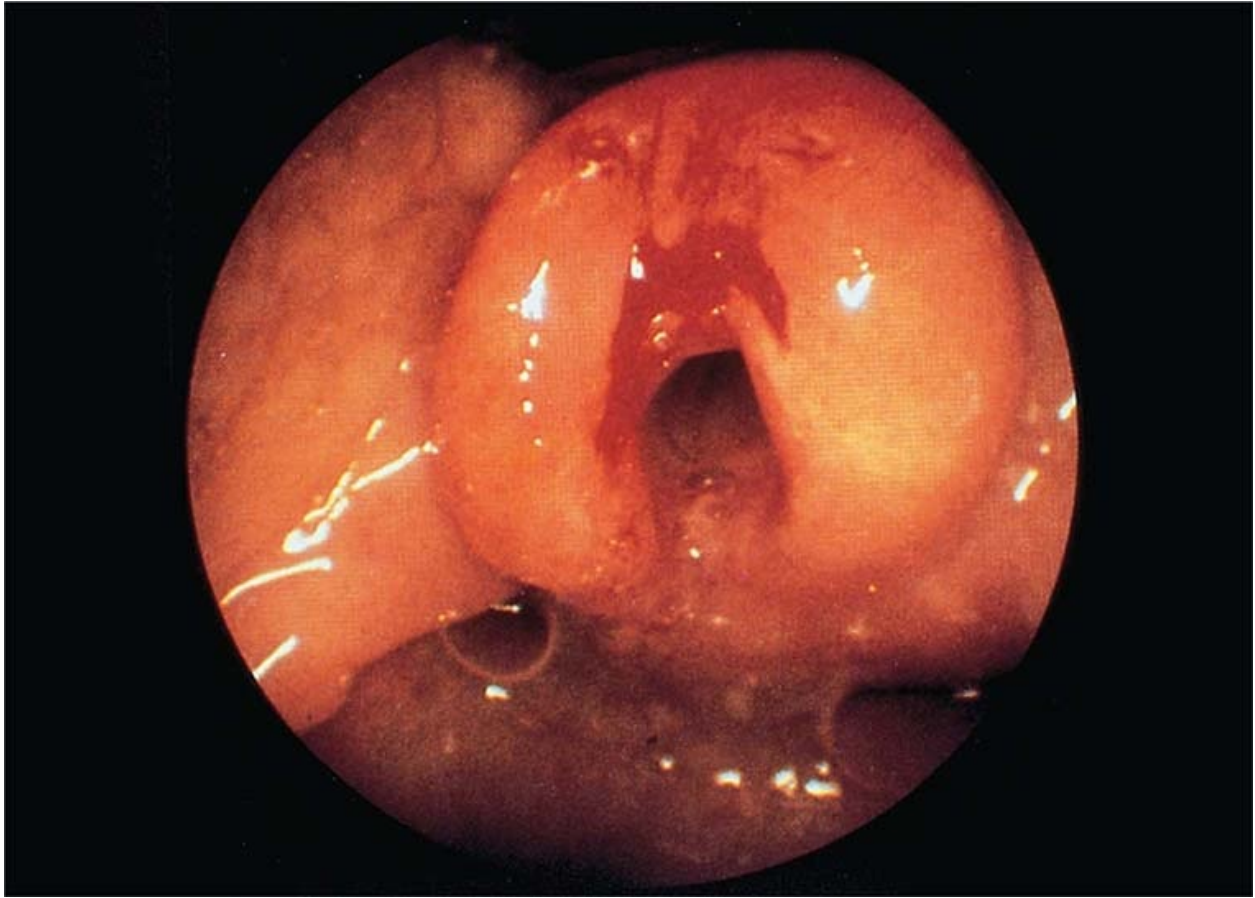


FIGURE 14.92 ■ Epiglottitis. The same patient immediately after extubation. Although erythema and some edema persist, the airway is widely patent. (Photo contributor: Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.)

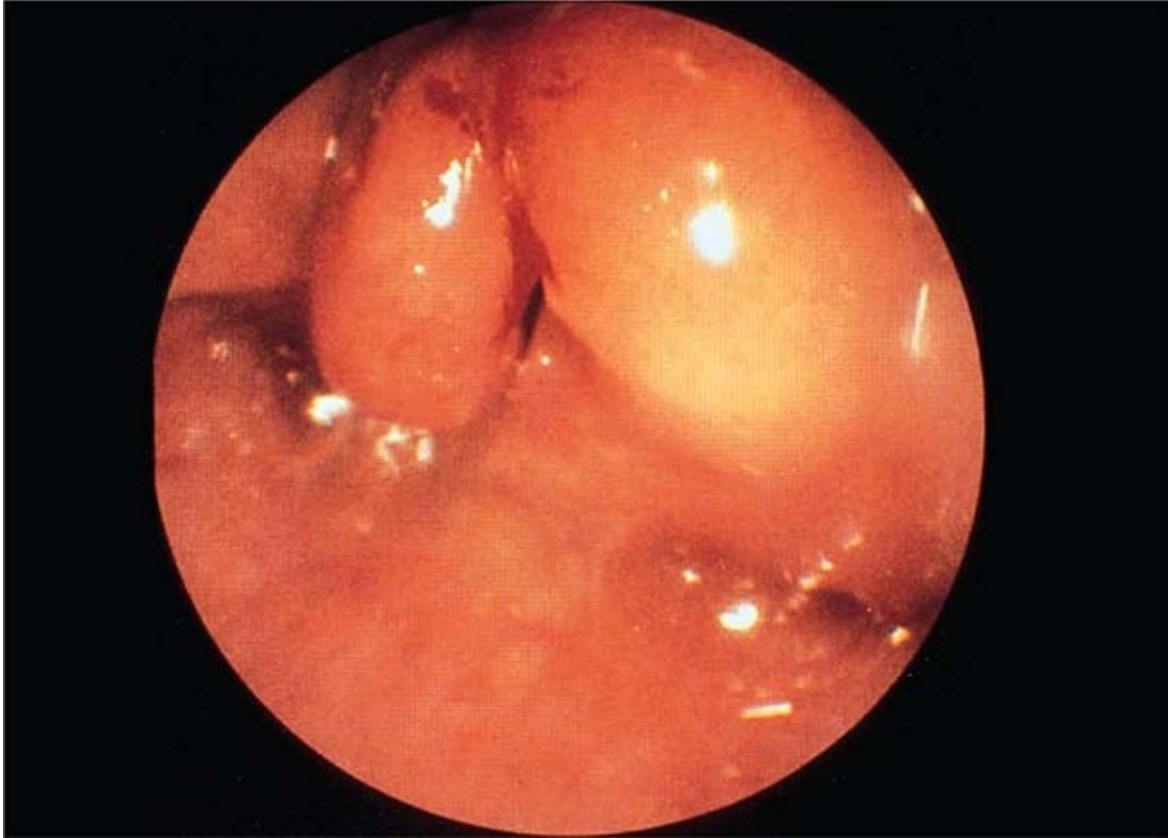


FIGURE 14.93 ■ Epiglottitis. Endoscopic view of almost complete airway obstruction secondary to epiglottitis. Note the slit-like opening of the airway. (Photo contributor: Department of Otolaryngology, Children's Hospital Medical Center, Cincinnati, OH.)

RETROPHARYNGEAL ABSCESS

Clinical Summary

Retropharyngeal abscess (RPA) usually presents with fever, difficulty swallowing, excessive drooling, sore throat, changes in voice, or neck stiffness. Limitation of neck movement on examination, especially with hyperextension, or torticollis will often be seen. The stiff neck may mimic meningitis. The characteristic retropharyngeal edema is a result of cellulitis and suppurative adenitis of the lymph nodes located in the retropharyngeal space between the buccopharyngeal fascia and the alar fascia. It is seen on a soft-tissue lateral x-ray of the neck as prevertebral soft-tissue thickening. The RPA may be preceded by an upper respiratory infection, pharyngitis, otitis media, or a wound infection following a penetrating injury to the posterior pharynx. The differential

diagnosis includes pharyngitis, acute laryngotracheobronchitis, epiglottitis, membranous (bacterial) tracheitis, cervical adenitis, infectious mononucleosis, peritonsillar abscess, foreign-body aspiration, and diphtheria.

Management and Disposition

RPA requires immediate assessment of the airway with establishment of a definitive airway if physical exam indicates progressive upper airway obstruction. The most common pathogens that cause RPA are group A *Streptococcus*, *S aureus*, MRSA, and respiratory anaerobes. Antibiotic coverage should be initiated immediately (clindamycin or a β -lactamase-resistant penicillin in areas where *S aureus* remains susceptible to methicillin). Analgesia should be administered as needed. Radiologic evaluation includes soft-tissue lateral neck x-ray and neck CT with contrast to define the extent of infection. In the absence of airway obstruction, medical treatment with IV antibiotics for 24 to 48 hours is first-line therapy. If impending obstruction is present or the infection is unresponsive to IV antibiotic therapy, needle aspiration or incision and drainage should be performed in the operating room. These patients require hospitalization and immediate otolaryngologic or surgical consultation.

Pearls

1. On lateral soft-tissue x-ray of the neck, the prevertebral soft tissue can measure up to 7 mm in width at the level of C2. At C6, it can measure up to 14 mm in width. This represents approximately one-half the width of the corresponding vertebral body.
2. The prevertebral soft tissue may appear falsely enlarged during neck flexion or crying.
3. The peak incidence occurs in 3- to 5-year-olds. It is rare beyond 6 years of age as the retropharyngeal lymph nodes involute.
4. RPAs in older patients most commonly arise as a complication of trauma or an immunocompromised state.
5. Children with severe RPA should be treated as an impending airway emergency and remain undisturbed while preparing for airway control, preferably in the operating room.



FIGURE 14.94 ■ Retropharyngeal Abscess. This ill-appearing 6-year-old child presented with a several-day history of fever, neck pain, sore throat, cough, and headache. Soft-tissue lateral radiography of the neck showed thickened prevertebral tissues opposite C2 to C4. CT showed the airway narrowed to a width of 5 mm within the oropharynx. (Photo contributor: Mark Ralston, MD.)

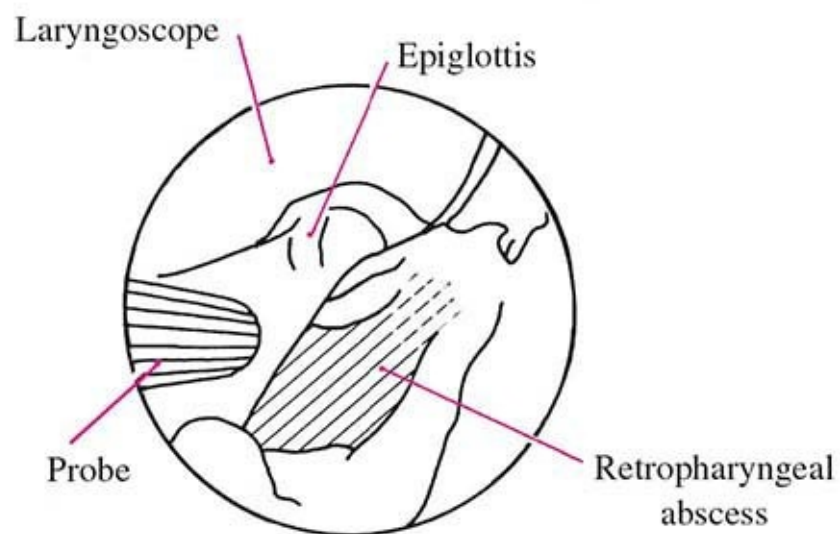
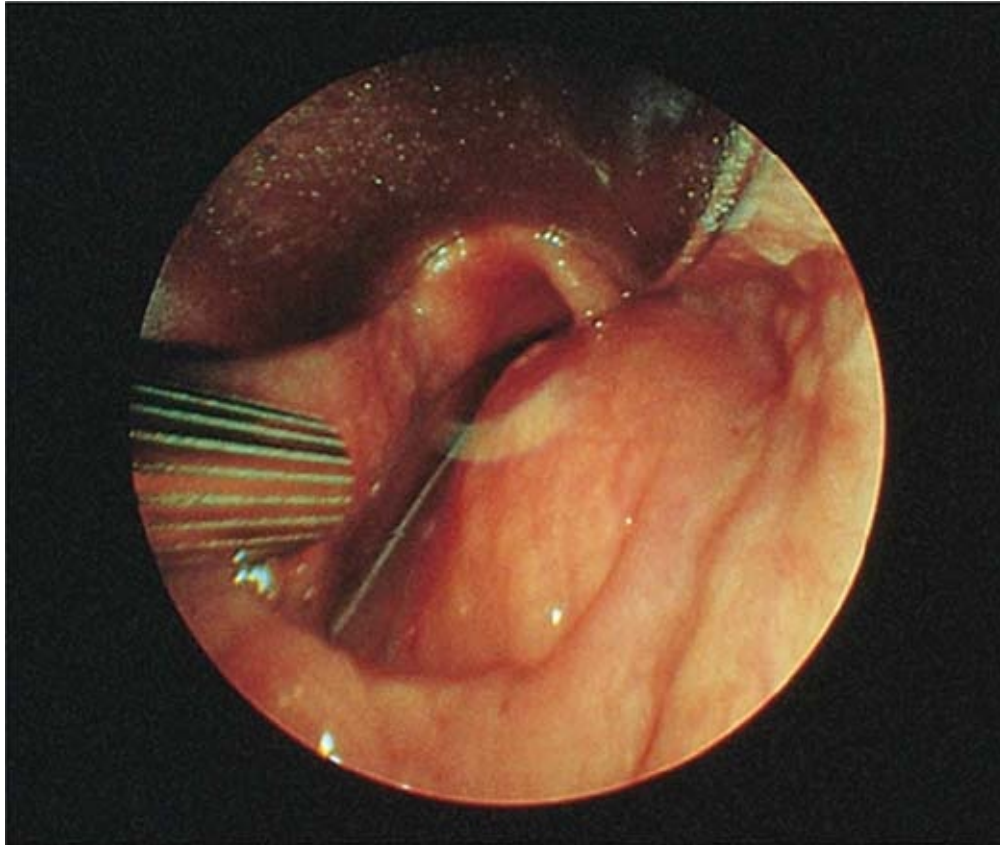


FIGURE 14.95 ■ Retropharyngeal Abscess. Endoscopic view of a retropharyngeal abscess. Note the massive swelling posteriorly. (Photo contributor: Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.)



FIGURE 14.96 ■ Retropharyngeal Abscess. Lateral soft-tissue neck x-ray demonstrating prevertebral soft-tissue density consistent with retropharyngeal abscess. (Photo contributor: Richard M. Ruddy, MD.)

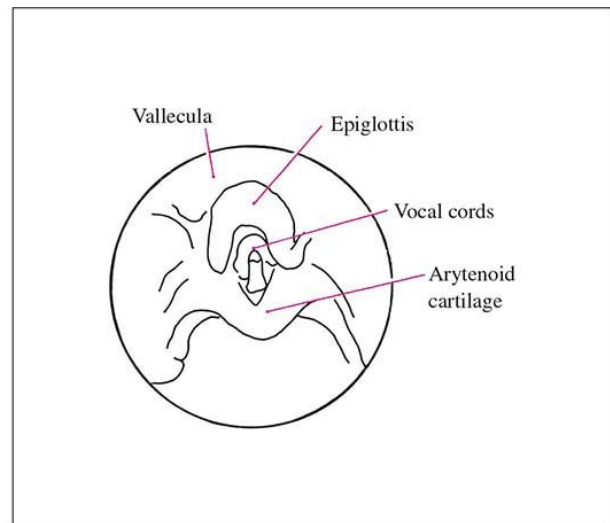
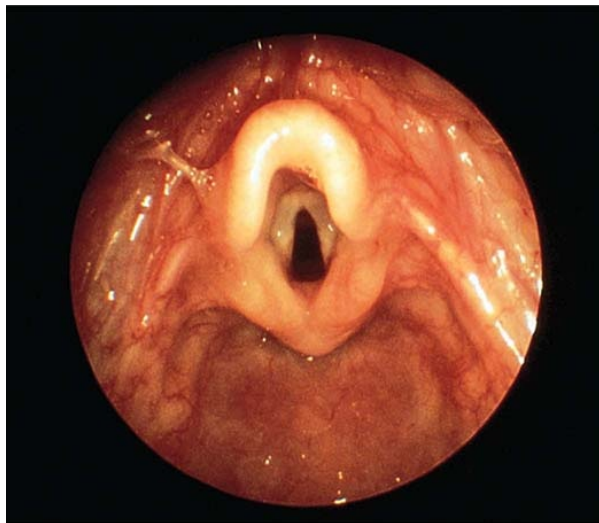


FIGURE 14.97 ■ Normal Laryngeal Structures. Endoscopic view of a normal epiglottis and surrounding structures. (Photo contributor: Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.)

BUTTON (DISK) BATTERY INGESTION

Clinical Summary

The majority of button battery ingestions occur in children less than 6 years of age, peaking between age 1 and 2 years. The most important factors in determining symptoms at presentation, as well as management, are location within the GI tract and duration of contact with the mucosal surface. Batteries that are lodged in the esophagus may be asymptomatic initially or can present with pain, drooling, dysphagia, poor oral intake, cough, vomiting, or fever. Mechanisms of injury associated with button battery ingestion include liquefactive necrosis resulting from alkali exposure due to battery leakage or the de novo synthesis of alkali at the surface of the battery, electrical current-induced soft-tissue injury, and tissue pressure necrosis.

Management and Disposition

Anteroposterior and lateral plain radiographs should be obtained to locate the battery within the GI tract. Button batteries can be distinguished from coins on plain x-ray by demonstration of a double contour. Batteries that are lodged in the esophagus can lead to potentially fatal complications and require immediate removal in consultation with a gastroenterologist or surgeon. This is best accomplished with direct visualization via endoscopy. Batteries that are in the stomach should also be removed promptly. Once the battery is past the pylorus, x-rays can be repeated at weekly intervals in asymptomatic patients until passage is documented. Instructions detailing concerning symptoms (abdominal pain, abdominal distention, hematemesis, or blood in the stools) should be provided to the parents at the time of discharge.

Pearls

1. Do not induce vomiting or attempt blind esophageal battery removal techniques.

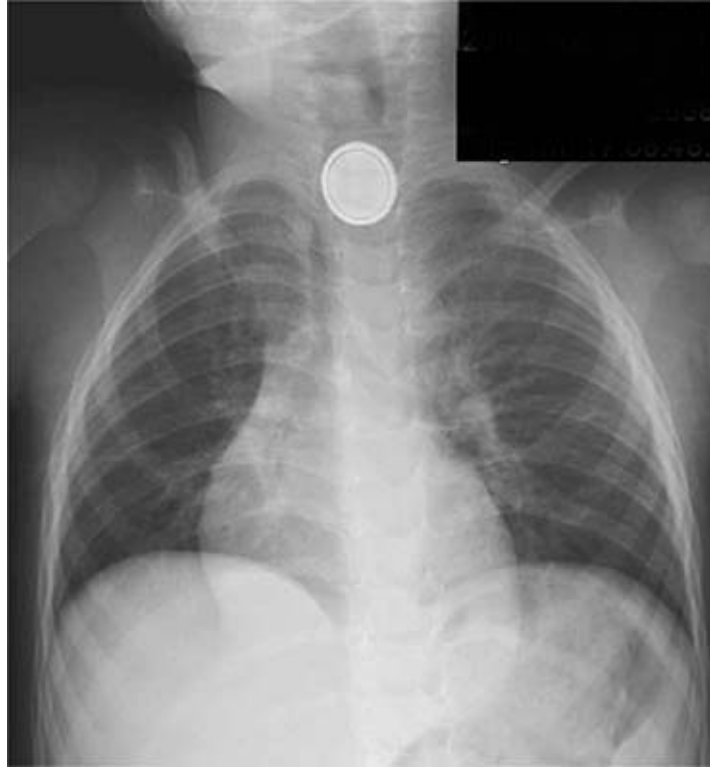


FIGURE 14.98 ■ Disk Battery Ingestion. Chest x-ray showing circular “coin-like” appearance with a second concentric ring from the plastic insulating grommet. This identifies the foreign body as a disk battery. (Photo contributor: Scott Manning, MD.)

2. Serious esophageal burns can occur within 2 hours, and impaction for more than 12 hours increases the risk of perforation.
3. Button batteries lodged in the nasal cavity or external auditory canal also require emergent removal to prevent complications such as perforation or stenosis.



FIGURE 14.99 ■ Disk Battery. X-ray showing “step-off sign” and showing the narrow end of a button battery in the nasal cavity. (Photo contributor: Craig Folsom, MD.)



FIGURE 14.100 ■ Disk Battery Ingestion. This endoscopic view shows esophageal necrosis after disk battery lodged in the esophagus. (Photo contributor: Scott Manning, MD.)

NASAL FOREIGN BODY

Clinical Summary

The nose is the most common site of aerodigestive tract foreign bodies in children. Most children present with a history of witnessed or suspected foreign-body insertion and are symptom free. However, one-quarter are discovered incidentally, with no preceding history by the caregiver. Common symptoms include mucopurulent discharge, foul odor, epistaxis, pain, and nasal obstruction. Objects most often include inorganic material (beads or small toys) and food. Unilateral foul-smelling discharge suggests organic or porous material (paper, sponge, foam rubber) or the long-standing presence of a foreign body leading to a localized inflammatory reaction.



FIGURE 14.101 ■ Nasal Foreign Body. Purulent drainage from the left nostril due to a candy wrapper which had been in place for 2 weeks. (Photo contributor: Larry B. Mellick, MD, MS, FAAP, FACEP.)

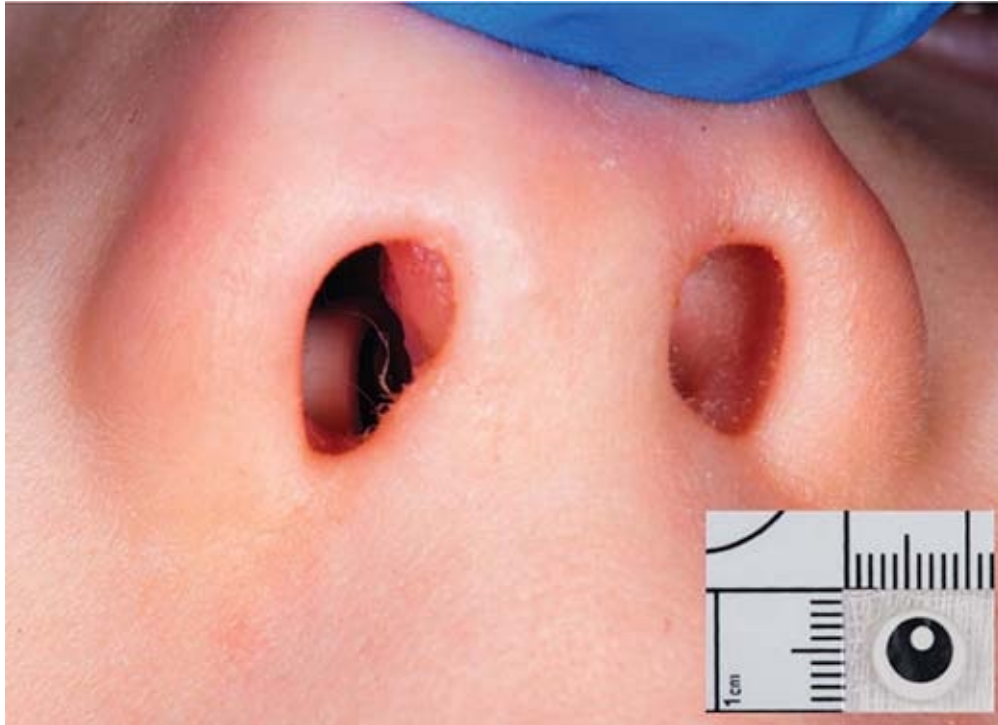


FIGURE 14.102 ■ Nasal Foreign Body. A round circular bead (insert) is embedded in the right nostril. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.103 ■ Button Battery. A button battery is seen in the right nasal passage, surrounded by necrotic tissue. (Photo contributor: Craig Folsom, MD.)

Foreign bodies are typically lodged anteriorly on the floor of the nasal

passage under the inferior turbinate or superiorly in front of the middle turbinate. Diagnosis is through direct visualization, although high foreign bodies may preclude visual diagnosis even with a head lamp or otoscope. Most are radiolucent, and unless you suspect a concomitant airway or GI foreign body, plain films are unnecessary. Button batteries and paired magnets can be especially dangerous due to the risk of local tissue compromise and should be removed promptly. Otherwise, removal can be an elective procedure. Techniques for removal include the use of positive pressure through the mouth with simultaneous occlusion of the unaffected nares (“mother’s kiss maneuver”) and removal with alligator forceps, a day hook (right-angle hook), or via a balloon catheter device threaded past the foreign body with subsequent balloon inflation and device retraction (Katz extractor). Children should be adequately prepared and properly immobilized. The value of an adept holder cannot be underestimated. The differential diagnosis includes allergic rhinitis, upper respiratory infection, acute sinusitis, cerebrospinal fluid leak, and nasopharyngeal polyp or mass.

Management and Disposition

Most foreign bodies can be successfully removed in the emergency department. After removal, always inspect for trauma and the presence of additional foreign bodies. Otolaryngology consultation or referral is warranted for poorly visualized posterior foreign bodies, chronic or impacted objects with significant inflammation, button batteries, penetrating or hooked foreign bodies, or any foreign body that cannot be removed due to poor cooperation or limited resources. After successful removal, patients can be safely discharged with instructions to return with any persistent, unilateral nasal discharge, recalcitrant epistaxis, or new fevers. Patients with minor bleeding after removal can be treated with a vasoconstricting nasal spray like oxymetazoline for no longer than 3 days.

Pearls

1. Organic materials that may expand with exposure to moisture (eg, dried beans) require urgent removal.
2. A button battery can lead to septal perforation in as little as 4 hours!
3. There is little concern for migration of inert foreign bodies into the airway in children with intact protective reflexes.



FIGURE 14.104 ■ Button Battery. A “double ring” or “halo” sign is seen in the aerodigestive tract of this child, identifying the object as a disk battery. (Photo contributor: Craig Folsom, MD.)

MEMBRANOUS (BACTERIAL) TRACHEITIS

Clinical Summary

Membranous tracheitis is an acute exudative bacterial infection (*S aureus*, *H influenzae*, *M catarrhalis*, streptococci, and pneumococci) of the upper airway capable of causing life-threatening airway obstruction. It may present as a primary infection or occur as a secondary bacterial complication of a viral infection of the upper respiratory tract. This locally invasive infection of the tracheal mucosa below the vocal cords produces copious purulent secretions. The exudate can form a thick plug that may ultimately lead to an acute tracheal obstruction. Patients appear toxic, with high fever and a croup-like syndrome that can progress rapidly. The characteristic “membranes” may be seen on x-rays

of the airway as edema with an irregular border of the subglottic tracheal mucosa. On direct laryngoscopy, profuse purulent secretions can be found in the presence of a normal epiglottis. The differential diagnosis includes acute laryngotracheobronchitis, RPA, epiglottitis, peritonsillar abscess, foreign-body aspiration, and acute diphtheric laryngitis.

Management and Disposition

Otolaryngologic consultation should be obtained as soon as the diagnosis is considered. Direct visualization of the trachea is more important than pursuing a radiologic diagnosis. Aggressive airway management, including endotracheal intubation, may be needed to protect the airway and allow for repeated suctioning to prevent acute airway obstruction. The patient should be admitted to the intensive care unit for close monitoring. Parenteral antibiotic coverage against suspected organisms (*S aureus*, *S pneumoniae*, group A *Streptococcus* (*S pyogenes*), α -hemolytic streptococci, *H influenzae* strains, and *M catarrhalis*) should be instituted immediately.



FIGURE 14.105 ■ Membranous Tracheitis. Lateral soft-tissue x-ray of the neck in a 13-year-old girl with the acute onset of stridor after 3 days of sore throat. Membranes (arrows) are visible in the subglottic region. (Photo contributor: Matthew R. Mittiga, MD.)

Pearls

1. Bacterial tracheitis often presents with acute, severe airway obstruction after a short prodrome. It should be suspected in all patients with an atypical croup-like presentation: unusual age group, toxic appearance, not improving with routine croup therapy, and unusual appearance of the tracheal lumen on plain radiographs.
2. In bacterial tracheitis, up to 50% of soft-tissue films may delineate a

subglottic membrane.

DACTYLITIS (HAND-FOOT SYNDROME)

Clinical Summary

This painful vaso-occlusive condition is commonly the 1st clinical manifestation of sickle cell disease in infants. It usually presents in children younger than 5 years of age, with nearly 45% of first cases presenting prior to the age of 2. Patients are acutely ill with fever, leukocytosis, and swollen hands and/or feet that are exquisitely painful due to bone infarction. Some children may present with fussiness and swelling with minimal erythema or fever. X-rays are often normal, and it is not until 1 to 2 weeks later that subperiosteal new bone, cortical thickening, and even complete bone destruction can be seen. The differential diagnosis includes osteomyelitis, trauma, cold injuries, acute rheumatic fever, juvenile rheumatoid arthritis, and leukemia.

Management and Disposition

Treatment of vaso-occlusive crises in sickle cell disease centers around providing adequate fluid balance, oxygenation, and analgesia. Therapy should be individualized. Fentanyl, hydromorphone, morphine, and ketorolac are analgesic agents commonly used in the treatment of children with painful sickle crises. If fever is present, bacterial infection should be assumed until proven otherwise. CBC, reticulocyte count, and blood cultures should be obtained from all febrile sickle cell patients. If the patient is afebrile, you can omit the blood culture. Empiric broad-spectrum antibiotic coverage should be instituted immediately (third-generation cephalosporin). In cases of dactylitis, very close follow-up is necessary not only for the management of sickle cell disease, but also to reevaluate the radiologic changes in the small bones of the hands and feet. In most instances, the previously described changes disappear; however, rarely, shortening of the fingers and toes may occur due to significant infarct.

Pearls

1. Most clinical manifestations of sickle cell disease occur after the first 5 to 6

months of life. Hemolytic anemia gradually develops over the first 2 to 4 months (changes that follow the replacement of fetal hemoglobin by hemoglobin S) and leads to the clinical syndromes associated with an increased percentage of abnormal SS hemoglobin.

2. Sick cell patients are at high risk for infection from encapsulated organisms due to their functional asplenia. The most common organisms causing osteomyelitis in sickle cell patients are *Salmonella* species, *S aureus*, and *S pneumoniae*.
3. Dactylitis can often be differentiated from osteomyelitis based on the symmetrical involvement of multiple bones and a negative blood culture.
4. Dactylitis, severe anemia, and leukocytosis within the first 2 years of life increase the risk for adverse sickle cell–related outcomes by age 10 years (death, stroke, frequent pain crises, or acute chest syndrome).
5. Hydroxyurea therapy has been shown to significantly reduce the rate of dactylitis in young children with sickle cell disease.



FIGURE 14.106 ■ Acute Sickle Dactylitis. Bilateral cylindrical swelling of soft tissue of the hands in sickle cell disease consistent with vaso-occlusive crisis or dactylitis. (Photo contributor: Donald L. Rucknagel, MD, PhD.)

HAIR TOURNIQUET

Clinical Summary

A single strand of hair or thread may encircle a finger, toe, or the penis, leading to circumferential strangulation of the appendage. Children in their 1st year of life are particularly at risk from inadvertent encirclement by a parent's hair or loose thread especially within confining clothing. The affected digit appears edematous, erythematous, and painful. If not corrected, vascular compromise or infection can ensue. The differential diagnosis includes insect bites, trauma, cellulitis of the digit, osteomyelitis, or ainhum (dactylosis spontanea), a painful constriction in the base of the 5th toe of unknown etiology followed by spontaneous autoamputation months to years later.

Management and Disposition

Visualization of the constricting material may be difficult. Edema, erythema, and periarticular skin folds may hide the hair or thread. It is imperative to carefully retract the skin around the proximal aspect of the edematous appendage. A magnifying lens may be helpful in identifying the band. Since the removal can be painful, consider a digital or penile block prior to removal. A topical anesthetic may also be applied to assist in reducing local pain. If the tourniquet is deeply embedded, a higher level of sedation may be required to appropriately localize and remove it. For superficial tourniquets that are visible with the naked eye and without significant skin breakdown, chemical hair removers are an appropriate first choice. If the tourniquet is synthetic, chemical removal fails, or there is significant skin breakdown, mechanical removal is indicated. Using a small hemostat, grasp a portion of the material, cut it with a surgical blade, and unwind it. Elevation of the involved digit after removal of the constricting agent provides resolution of the edema and erythema within 2 to 3 days. In some cases, the digit's blood supply may have been irreversibly compromised. Subspecialty consultation should be considered whenever neurovascular integrity is in question or if the constricting band cannot be visualized and removed. The patient should be reevaluated in 24 hours to assure improvement and need for further surgical consultation or intervention.

Pearls

1. In the vast majority of cases, a clear line of demarcation can be identified between the normal tissue and the affected area.
2. Fussiness or irritability may be the only presenting symptom. Examination for hair tourniquets should be included in the evaluation of any inconsolable infant.
3. There may be an insidious onset with reepithelialization over the hair tourniquet, leaving it difficult to visualize.



FIGURE 14.107 ■ Hair Tourniquet. A strand of hair has encircled the middle toe in two places, causing erythema and swelling. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.108 ■ Hair Tourniquet. Erythema distal to the hair tourniquet of the affected toes due to strangulation by the tourniquet. (Photo contributor: Robert W. Hickey, MD.)



FIGURE 14.109 ■ Hair Tourniquet. Erythema and edema from ischemia due to a hair tourniquet. (Photo contributor: David Effron, MD.)

FAILURE TO THRIVE

Clinical Summary

Failure to thrive (FTT) is the inability to maintain a normal growth pattern in weight, stature, and occasionally in head growth. Definitions are varied and

include a fall in weight below the 2nd percentile relative to corrected gestational age and sex or growth deceleration that crosses two major percentiles on a standardized growth chart. It is most common in infancy, and the condition is nonorganic (50%), organic (25%), or mixed (25%) in etiology. The diagnosis is made after complete history and physical examination with comparison of the measurements of length (supine in children < 3 years of age), weight, and head circumference (maximal occipital-frontal circumference) to standard measurements. In cases of deficient caloric intake or malabsorption, the patient's head circumference is normal and the weight is reduced out of proportion to length/height. In general, FTT is due to decreased intake, increased output, increased caloric demand, or some combination of all three. The differential diagnosis of FTT is lengthy. Nonorganic disorders are more common and include poor feeding technique, disturbed maternal-child interaction, emotional deprivation, inadequate caloric intake, and child neglect. Organic causes are numerous.



FIGURE 14.110 ■ Failure to Thrive. This infant has not been able to maintain a normal growth pattern. Note skin folds in upper extremities due to loss of subcutaneous fat. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Depending on history, physical findings, the social situation, and ability to ensure close monitoring by a primary care physician, most cases can be managed as outpatients. The primary care provider can assist in determining whether outpatient management is indicated. If the diagnosis of FTT is made in the

emergency department, admission is suggested to complete the evaluation. That the child presented to the emergency department could be the only indicator of a poor social environment or inadequate access to medical care. Simple initial testing includes CBC, urinalysis, electrolytes, BUN, creatinine, ESR, and CRP. If the history and physical exam suggest it, liver function tests, screening thyroid studies, prealbumin, and lead testing are appropriate. For very young infants, it is important to document a normal newborn screen. Early involvement of social services may facilitate the evaluation and follow-up. Treatment will vary according to the underlying disorder and often involves a team approach.

Pearls

1. FTT in neglected children is accompanied by signs of developmental delays, emotional deprivation, apathy, poor hygiene, withdrawn behavior, and poor eye contact.
2. The major contributor to FTT is caloric inadequacy. Dietary history should include details of formula preparation, volume consumed, and, in toddlers, the volume of juice consumed.



FIGURE 14.111 ■ Failure to Thrive. Accentuation of the gluteal folds secondary to loss of subcutaneous fat in an infant with FTT. (Photo contributor: Andrew H. Urbach, MD.)

NURSING BOTTLE CARIES

Clinical Summary

The etiology of dental caries is multifactorial with an interplay between microflora (plaque colonized with *Streptococcus mutans*), substrate (fermentable carbohydrates from breast milk, formula, or juice), environmental factors (poor parental education, low socioeconomic status), and host (saliva and teeth). Nursing or milk bottle caries result from prolonged and frequent night time breastfeeding or sleeping with a bottle containing milk or sugar-containing juices. The sugars are fermented by the bacteria in plaque, lowering the pH in the mouth and resulting in demineralization of the tooth enamel. The condition generally occurs before 18 months of age and is more prevalent in medically underserved children. Upper central incisors are most commonly involved. Outpatient dental referral is indicated.

Management and Disposition

Parental education and timely referral to a dentist are necessary to prevent complications. If untreated, the caries may destroy the teeth and spread to contiguous tissues. These patients have a high risk for microbial invasion of the pulp and alveolar bone with the subsequent development of a dental abscess and facial cellulitis. In these cases, aggressive treatment with antibiotics (amoxicillin) and pain control, with prompt dental referral for definitive care, is necessary.

Pearls

1. The role of the emergency department physician is to recognize this pattern of dental decay (upper incisors most commonly) and initiate dental referral and parental education.
2. Nursing or milk bottle caries tends to spare the lower front teeth because of the shielding of the lip and tongue and the increased exposure to saliva from the sublingual glands that washes away cariogenic substrates.
3. The newborn mouth is generally devoid of microorganisms. Newborns and infants are colonized with *S mutans* from parents and family members. Education on avoidance of sharing utensils and cups may help delay

colonization of infants.



FIGURE 14.112 ■ Nursing Bottle Caries. Extensive tooth decay from sleeping with bottle containing milk or sugar-containing juices. (Photo contributor: Lawrence B. Stack, MD.)

NURSEMAID'S ELBOW (RADIAL HEAD SUBLUXATION)

Clinical Summary

Nursemaid's elbow is a condition that occurs commonly in children younger than 6 years of age who are usually picked up or pulled by the extended, pronated arm. The peak incidence is 2 to 3 years of age. These children present unwilling to supinate or pronate the hand on the affected side. Generally they hold the affected arm close to their side in a passive pronation with partial flexion at the elbow. Radiographic studies should be considered only in patients

with an unusual mechanism of injury, with significant bony tenderness, or who do not become rapidly asymptomatic after the reduction maneuver. The differential diagnosis includes radial head fracture or complete dislocation, posterior elbow dislocation, condylar and supracondylar fractures of the distal humerus, or buckle fracture of the radius or ulna.

Management and Disposition

Carefully palpate all points of the affected arm for tenderness. There should be minimal to no pain with palpation. Orthopedic consultation is generally not indicated unless an underlying fracture is diagnosed. Reduction is usually achieved by one of two maneuvers:

1. Hyperpronation. While holding the elbow in extension, hyperpronation of the forearm is maintained until reduction is achieved.
2. Flexion/supination. Beginning with the elbow flexed at 90 degrees, the wrist of the affected arm is grasped with one hand, while the other stabilizes the elbow. The forearm is then gently supinated while the arm is flexed until the palm approaches the anterior shoulder.

In either maneuver, a palpable click over the radial head is evident upon successful reduction. Studies have indicated that hyperpronation is more likely to be successful when used as the initial reduction maneuver. The patient usually begins using the arm normally within minutes. When the injury has been present for several hours, reduction may be difficult, and it may take several hours to recover full function of the elbow.

Pearls

1. Radiographs of radial head subluxation typically appear normal.
2. Immobilization after reduction is not necessary.
3. If the patient remains symptomatic after reduction attempts, obtain x-rays to assess for fractures.
4. Up to one-third of children will have recurrence, but rarely beyond age 4 to 5 years.



FIGURE 14.113 ■ Nursemaid's Elbow Reduction. Simultaneous supination with elbow flexion technique for reduction of nursemaid's elbow. (Photo contributor: R. Jason Thurman, MD.)

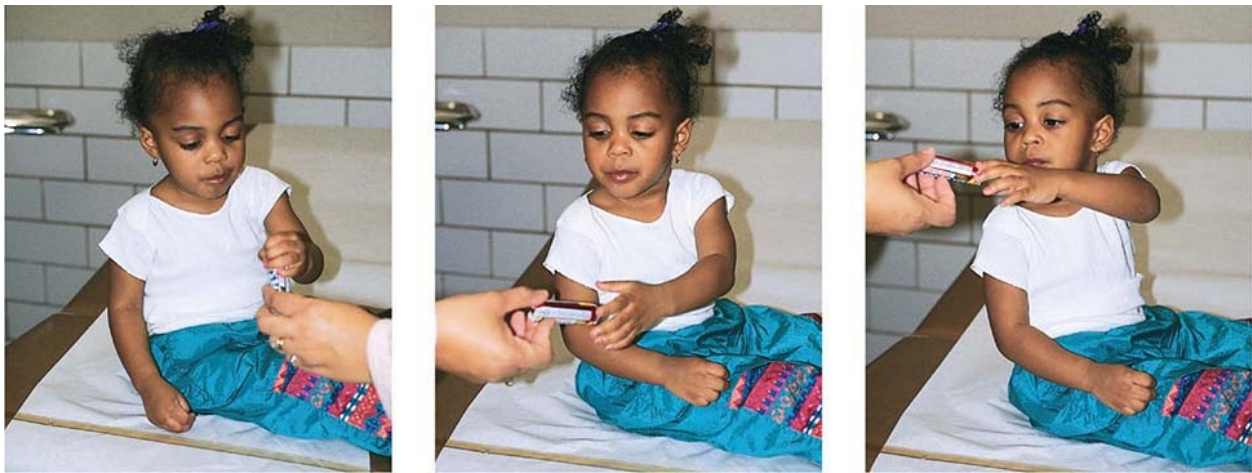


FIGURE 14.114 ■ Nursemaid's Elbow. This child presents with pseudoparalysis of the right arm after a pulling injury. Note how she avoids use of the affected arm and preferentially uses the other arm. (Photo contributor: Kevin J. Knoop, MD, MS.)

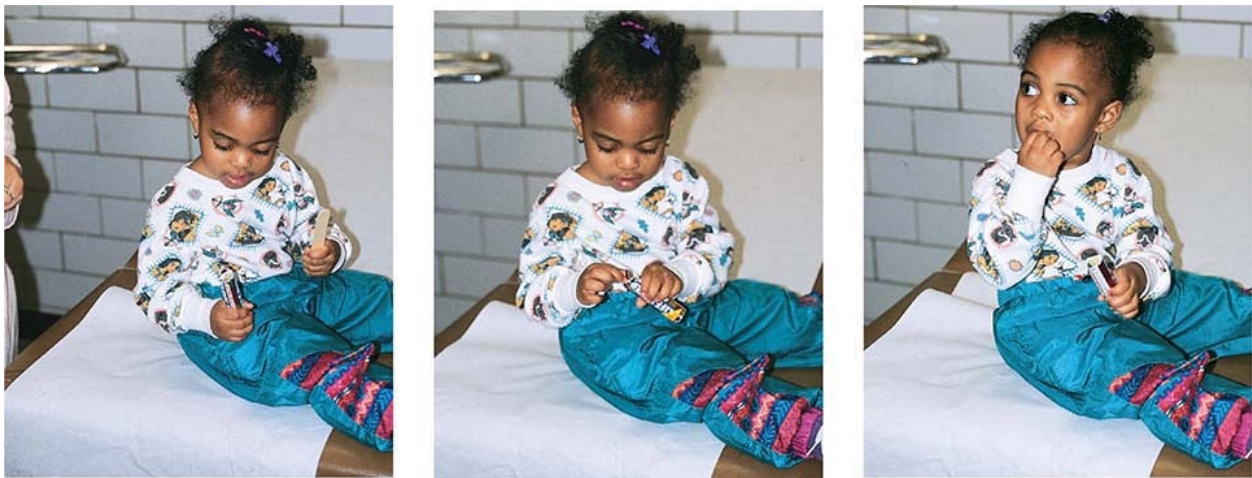


FIGURE 14.115 ■ Nursemaid's Elbow—Reduced. After reduction, there is initial reluctance to use the injured arm. With distraction and encouragement, the patient demonstrates successful use of the extremity. (Photo contributor: Kevin J. Knoop, MD, MS.)

ILEOCOLIC INTUSSUSCEPTION

Clinical Summary

Intussusception occurs when one segment of bowel invaginates into itself, most

commonly at the ileocecal junction. It is the most common abdominal emergency and cause of intestinal obstruction in children less than 2 years of age. The precise cause is unknown in 75% of cases, although the incidence increases during seasonal viral gastroenteritis outbreaks. Adenovirus, bacterial enteric infections, Meckel diverticula, tumors, hematomas, and vascular malformations are thought to be potential lead points.

Patients typically present with sudden onset of intermittent severe episodes of cramping abdominal pain with inconsolable crying and drawing the legs up to the abdomen. Episodes usually occur at 15- to 20-minute intervals, but become more frequent as the illness progresses. Vomiting may follow painful episodes and can eventually become bilious. Initially, children appear normal between episodes, but progressive lethargy eventually develops. Bloody stools (occult or gross), a sign of intestinal ischemia, are seen in 50% of patients. A sausage-shaped mass may be palpated in the right upper quadrant. The differential diagnosis includes viral gastroenteritis, constipation, HPS, intestinal malrotation with volvulus, appendicitis, and meningoencephalitis.

Management and Disposition

If the diagnosis is uncertain, ultrasound is the imaging test of choice and often demonstrates the classic “coiled spring” or “bull’s eye” lesion. Plain x-rays obtained early in undifferentiated cases of abdominal pain may show a target or crescent sign, along with a paucity of cecal gas. Treatment begins with nonoperative reduction at an experienced institution with an air enema under fluoroscopic or sonographic guidance using pneumatic pressure to reduce the intussusception. The success rate is 80% to 85%. The risk of perforation is < 1%. As 24-hour recurrence rates are low (< 5%), admission to the hospital should be a case-by-case decision. Many patients can be discharged with strict return precautions after a 4- to 6-hour observation period if they are well appearing and tolerating a diet. Seek surgical consultation, as laparoscopy is indicated when nonoperative reduction is unsuccessful or incomplete.

Pearls

1. Currant jelly stools are seen in only 20% of patients and are a combination of blood and mucus indicative of sloughed intestinal villi.
2. The classic triad of pain, palpable mass, and bloody stools is seen in less than 15%.

3. Infants can present with profound lethargy alone, with one-third having no apparent abdominal pain. Consider intussusception in any infant with altered mental status.



FIGURE 14.116 ■ Ileocolic Intussusception. Bloody “currant jelly” stools in a hypersomnolent 3-month-old female infant with transverse colon intussusception reduced by air enema. (Photo contributor: Donald H. Arnold, MD, MPH.)



FIGURE 14.117 ■ Intussusception. The apex of the intussusception may extend through the anus mimicking rectal prolapse. It is distinguished from rectal prolapse by the separation between the protruding intestine and the rectal wall. (Photo contributor: Binita R. Shah, MD. From Shah B, Lucchesi M, Amodio J, Silverberg M. *Atlas of Pediatric Emergency Medicine*. 2nd ed. New York, NY: McGraw Hill; 2013: Fig. 10-3, p. 379.)



FIGURE 14.118 ■ Rectal Prolapse. Protrusion of the sigmoid colon is seen in this infant. (Photo contributor: Lawrence B. Stack, MD.)

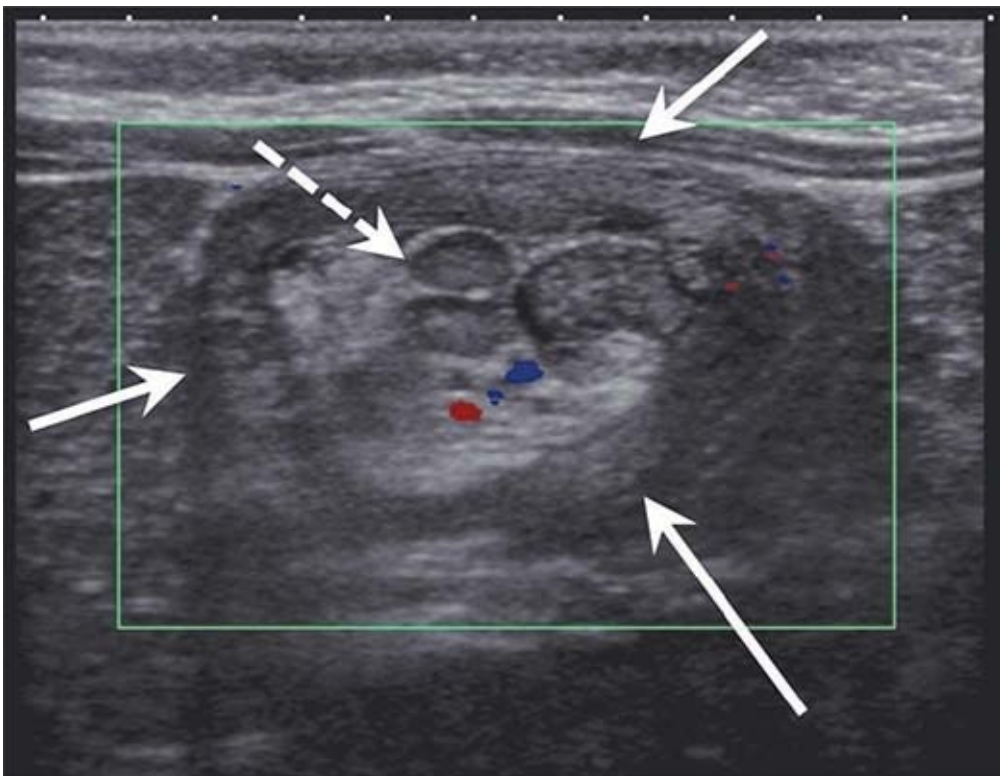


FIGURE 14.119 ■ Ileocolic Intussusception. Ultrasound showing intussusciens (*arrows*) containing echogenic fat and multiple lymph nodes (*dashed arrow*). (Photo contributor: Alexander Towbin, MD.)



FIGURE 14.120 ■ Intussusception Reduction via Air Contrast Enema Part 1. Air distending the rectum and colon just before reduction. The intussusception mass (*arrow*) is in the right upper quadrant near the hepatic flexure. (Photo contributor: Alexander Towbin, MD.)

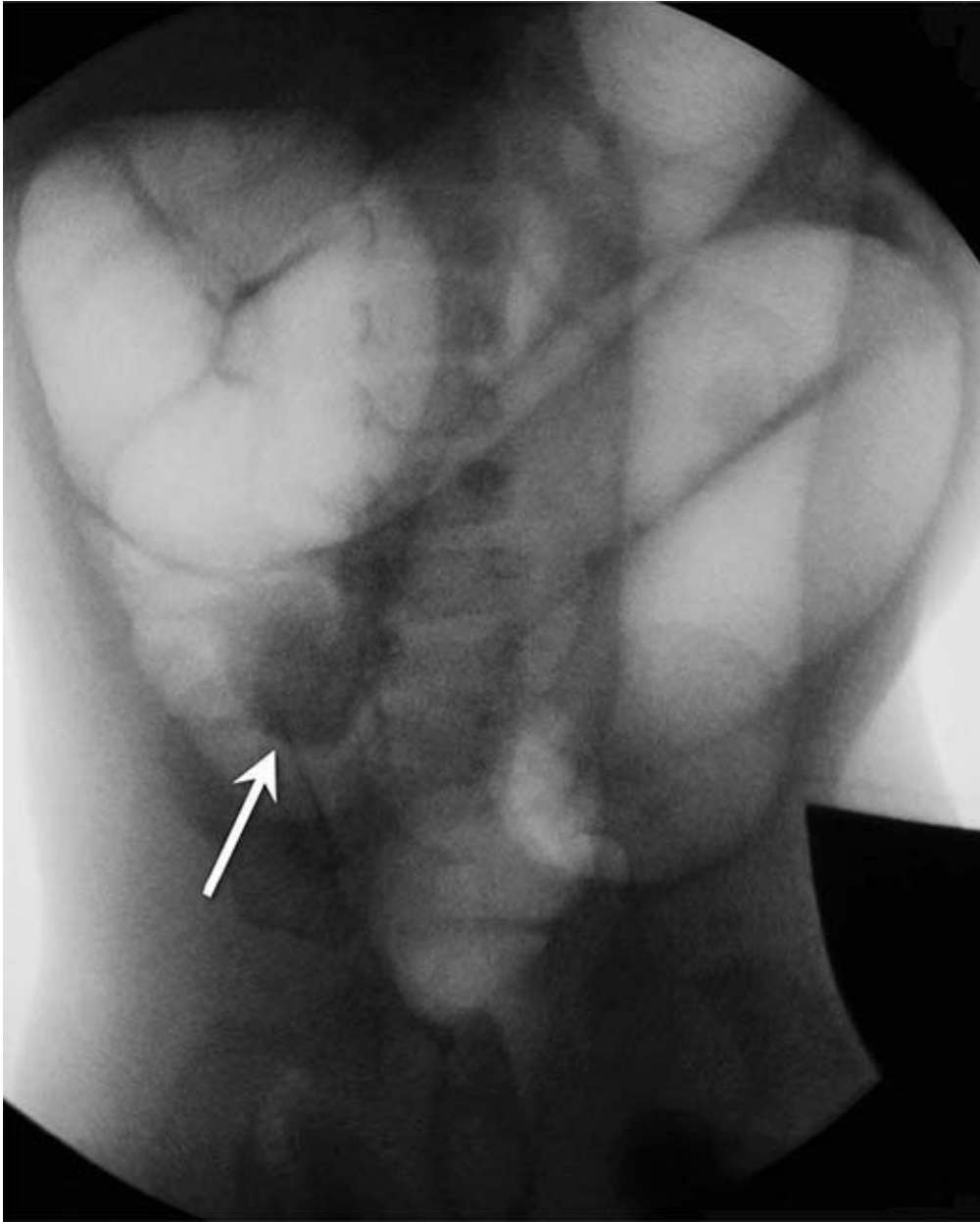


FIGURE 14.121 ■ Intussusception Reduction via Air Contrast Enema Part 2. Reduction of the intussusception (*arrow*) to the level of the ileocecal valve. (Photo contributor: Alexander Towbin, MD.)



FIGURE 14.122 ■ Intussusception Reduction via Air Contrast Enema Part 3. The intussusception has been reduced. There is free reflux of air into the small bowel. (Photo contributor: Alexander Towbin, MD.)

HYDROCELE OF THE TESTIS

Clinical Summary

A hydrocele of the testis refers to a collection of fluid surrounding the testicle. It is caused by fluid collecting between the parietal and visceral layers of the tunica vaginalis. There are two common types of hydroceles: communicating and noncommunicating. Communicating hydroceles lead to a collection of peritoneal fluid around the testes from a failure of the processus vaginalis closing during development. Noncommunicating is from the direct secretion of fluid from the

tunica vaginalis. Infants and adolescents generally present with painless scrotal swelling.

Management and Disposition

Diagnosis can often be made clinically through a physical exam and transillumination of the scrotum. If the diagnosis is unclear, a scrotal ultrasound with Doppler should be obtained to differentiate the hydrocele from more concerning pathology. In infants and young children, the management is largely supportive if the hydrocele is asymptomatic. Communicating or noncommunicating hydroceles in patients older than 2 rarely spontaneously resolve and should be referred to a subspecialist for an elective closure.

Pearls

1. Hydroceles in the less than 1 age group do not often require intervention and resolve spontaneously.
2. Communicating hydroceles increase in size with the Valsalva maneuver (crying in an infant) or during the day due to gravity, unlike noncommunicating hydroceles.
3. Reactive hydroceles can be painful and erythematous and are likely a secondary finding to a different pathology (epididymitis, testicular torsion, testicular rupture).

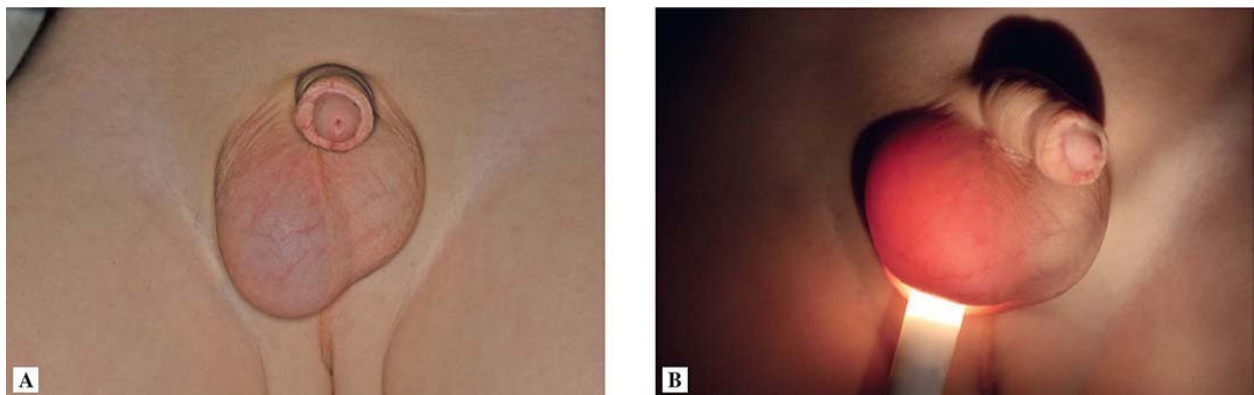


FIGURE 14.123 ■ Hydrocele. Painless scrotal swelling (A) which transilluminates (B). This patient had an inguinal hernia that was repaired electively. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Inguinal hernias are common in childhood with an incidence as high as 5%. Premature infants have an even higher incidence, and boys are approximately 10 times more likely than girls to develop an inguinal hernia. There are two types of inguinal hernias: indirect (common) and direct (rare). Indirect inguinal hernias result from failure of the processus vaginalis to obliterate toward the end of fetal development. With a patent processus vaginalis, the intra-abdominal viscera can protrude through the internal inguinal ring. Indirect inguinal hernias are more common on the right and present as a bulge in the groin by parental history or on physical examination. Maneuvers that increase intra-abdominal pressure, such as crying in an infant or blowing bubbles in an older child, may make the hernia easier to visualize. Associated symptoms such as vomiting, abdominal distention, constipation, blood in the stool, lethargy, or irritability suggest incarceration or strangulation of the hernia. Incarceration is most common in the 1st year of life. The differential diagnosis includes hydrocele, inguinal lymphadenopathy, testicular torsion, torsion of the appendix testis/epididymis, epididymitis/orchitis, and a retractile testicle.

Management and Disposition

With the history of scrotal swelling but a normal physical examination, refer to a pediatric surgeon for timely evaluation and repair. If a hernia is palpable, ensure that it can be easily reduced manually. Reduction of an incarcerated inguinal hernia may be facilitated by adequate pain control and sedation (80% successful). The reduction technique consists of gentle traction inferiorly on the hernia sac with pressure from above to straighten the inguinal canal. There is a high rate of early recurrence of incarceration. Admission to the hospital with repair in 24 to 48 hours after the associated edema has subsided is advisable. Immediate surgical consultation for operative repair is indicated for any incarcerated hernia that cannot be manually reduced.

Pearls

1. Transilluminate the scrotum. Hydroceles will transilluminate, whereas hernias will not.
2. The Trendelenburg position may aid in hernia reduction.

3. Palpate both testicles in the scrotum prior to diagnosing an inguinal hernia.
4. Plain abdominal radiographs are unlikely to be helpful in cases of scrotal swelling where the diagnosis is not apparent. Scrotal ultrasound has a diagnostic accuracy of over 90%.

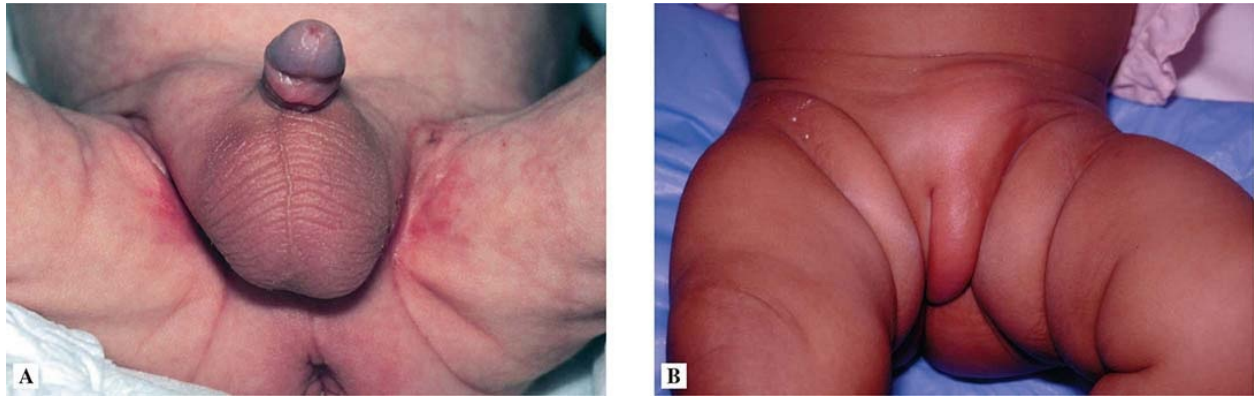


FIGURE 14.124 ■ Inguinal Hernia. (A) Right inguinal hernia in a male infant (B) Left inguinal hernia in a female infant. (Photo contributor: Lawrence B. Stack, MD.)

PINWORM INFECTION (ENTEROBIASIS)

Clinical Summary

Enterobius vermicularis is a threadlike white worm that infects the colon and causes intense nocturnal perianal pruritus when the gravid adult female migrates to deposit eggs at night. Female worms measure 8 to 13 mm in length and can be observed moving about the perianal area at night. On rare occasions, this nematode can lead to vulvovaginitis. The diagnosis can be made by direct visualization of the nematode by the parents or by using a piece of transparent adhesive tape and touching it to the perianal area upon awakening in the morning. This tape is then applied to a glass slide for microscopic examination under low power to look for “bean-shaped” eggs. The differential diagnosis includes perianal irritation, perianal group A *Streptococcus* infection, cellulitis, fissures, hemorrhoids, and contact dermatitis.

Management and Disposition

Stool studies are not warranted as the eggs are not generally passed in the stool. The treatment of choice is the anthelmintic agents, albendazole or mebendazole.

The initial single dose is repeated in 2 weeks to treat secondary *Enterobius* hatchlings. Because of the high frequency of reinfection, families should be treated as a group, and household bedding and clothes should be washed.

Pearls

1. Reinfection from other infected individuals (daycare cohorts) or autoinfection is necessary to maintain enterobiasis in the individual, since these nematodes usually die after depositing their eggs in the perianal region. Frequent hand washing may reduce chances of infection as transmission occurs by the fecal-oral route.
2. *E. vermicularis* is the most common intestinal nematode in the United States, affecting 5% to 15% of the population. Many infections are asymptomatic.
3. Suspect pinworm infection in children who present with nocturnal restlessness. These patients are often evaluated for urinary tract infection because the scratching of the perineal area is misinterpreted by the parents and treating clinicians as painful urination.



FIGURE 14.125 ■ Pinworms. Multiple tiny pearly white worms are seen at the anus. (Photo contributor:

LICE

Clinical Summary

There are three varieties of lice specifically parasitic to humans: *Pediculus humanus capitis* (head louse), which infests the hair and scalp; *Pthirus pubis* (crab louse), which infests the pubic hair; and *Pediculus humanus corporis* (body louse). Affected patients may be asymptomatic or may have pruritus. The diagnosis is made via visual examination whereby crawling lice (nymphs and adults) and eggs (nits) are found on hair shafts. The treatment is topical pediculicides (including permethrin, malathion, benzyl alcohol, spinosad, and topical ivermectin) and wet combing to remove lice and nits. Lindane has an association with neurotoxicity, and its use should be avoided in children. Of note, nits may persist for several weeks following therapy. The differential diagnosis includes pseudonits (2- to 7-mm hair casts), psoriasis, cutaneous fungal infections, scabies, seborrheic dermatitis, and dandruff.

Management and Disposition

The diagnosis is clinical. Patients should be treated with topical pediculicides and educated on signs of resolution. Treatment failure is associated with noncompliance, reinfestation, and drug resistance. There is growing resistance to permethrin within the United States and Canada so other therapies should be considered if there is treatment failure with good compliance. Close contacts and those who share bedding should be treated prophylactically. Head lice generally do not survive for more than 2 days when off of a person. The nits die within 1 week if the temperature close to the scalp is not maintained. Clothing and items (eg, pillows, bedding, or stuffed animals) that cannot be washed should be dry-cleaned or sealed for 2 weeks in a plastic bag.

Pearls

1. Children with head lice do not necessarily need to be excluded from school as many have had lice for several weeks and been attending school prior to

diagnosis.

2. Combs and brushes can be disinfested by soaking in hot water (130°F or greater) for 5 to 10 minutes.
3. Although shaving hair is anecdotally an effective treatment, it can have adverse psychological effects.



FIGURE 14.126 ■ **Nits.** Several small white cylindrical nits (louse eggs) are seen clinging to dark hair. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 14.127 ■ Lice Infestation. Multiple lice are seen in the hair of this patient. (Photo contributor: David Effron, MD.)

ORAL FRENULUM TEAR

Clinical Summary

A frenulum is a mucosal fold that connects to mobile tissue and secures it in place. The oral frenula include the *frenulum linguae* (connects underside of the

tongue to base on mouth), *frenulum labii superioris* (connects upper lip to the gingiva), and the *frenulum labii inferioris* (connects lower lip to the gingiva). Tearing of the frenula can be caused by direct trauma to the area. Like most oral injuries, there can be a significant amount of bleeding, but it generally stops spontaneously or with direct pressure. Frenulum tears can be a result of but are not pathognomonic for nonaccidental trauma. Mechanisms of injury include forceful feeding (“bottle jamming”) or pacifier insertion, gripping or stretching lip, or direct blow.

Management and Disposition

Management is generally reassurance, and tears rarely require primary closure. If there is continued bleeding, hold pressure to the outside of the lip until bleeding stops. Advise parents to avoid pulling the lip open to check for healing as it can lead to further trauma. Rarely, primary closure is required. If the tear extends further into the gingiva or is a portion of a larger oral injury, consider closure with absorbable sutures. Frenulum tears and other intraoral injuries are seen in a significant number of nonaccidental trauma cases. If the development of the infant and the history are not consistent with the injury, be sure to complete a full skin assessment to look for other injuries and have a high index of suspicion for nonaccidental trauma.

Pearls

1. A frenulum tear can be caused by “bottle jamming” from force feeding and can be a sign of nonaccidental trauma. Be sure to have a high index of suspicion in nonambulatory infants.
2. An isolated frenulum tear can be managed conservatively and rarely needs primary closure.



FIGURE 14.128 ■ Frenulum Tear. This ambulatory child fell, causing the frenulum to tear. An associated chin abrasion is present. Feasible history in an ambulatory child makes nonaccidental trauma unlikely. (Photo contributor: Kevin J. Knoop, MD, MS.)

OCULOGYRIC CRISIS

Clinical Summary

Oculogyric crisis (OGC) is the most common of the ocular dystonic reactions. It includes blepharospasm, periorbital twitches, and protracted staring episodes. It usually occurs as a side effect of neuroleptic drug treatment. OGC represents approximately 5% of dystonic reactions. The onset of a crisis may be paroxysmal or stuttering over several hours. Initial symptoms include restlessness, agitation, malaise, or a fixed stare followed by the more characteristically described maximal sustained upward deviation of both eyes. The eyes may also converge, deviate upward and laterally, or deviate downward.

The most frequently reported associated findings are backward and lateral flexion of the neck, widely opened mouth, tongue protrusion, and ocular pain. Episodes generally last minutes but can range from seconds to even hours. A wave of exhaustion follows some episodes. Other features noted during attacks include eye blinking, lacrimation, pupil dilation, drooling, facial flushing, vertigo, anxiety, and agitation. Several medications have been associated with the occurrence of OGC: cetirizine, neuroleptics, amantadine, benzodiazepines, carbamazepine, chloroquine, levodopa, lithium, metoclopramide, and nifedipine. Careful history and physical examination should exclude the possibility of focal seizures, meningitis, encephalitis, head injury, conversion reaction, Parinaud syndrome, and other types of movement disorders.



FIGURE 14.129 ■ Oculogyric Crisis. This 6-year-old boy developed extrapyramidal symptoms, including opisthotonos and oculogyric crisis, after his dosage of risperidone was increased. Benadryl 12.5 mg orally given at home resolved the opisthotonos. Persistent oculogyric crisis (upward gaze deviation) and hypertonia resolved completely after Benadryl 25 mg intramuscularly. (Photo contributor: Mark Ralston, MD.)

Management and Disposition

Treatment in the acute phase in children involves reassurance, discontinuation of the causative agent, and diphenhydramine at a dosage of 1.25 mg/kg initially; this may be repeated if there is no effect. For moderate to severe cases, give the initial dose parenterally. Occasionally, doses up to 5 mg/kg are required.

Treatment with diphenhydramine should be continued every 6 hours for 4 to 7 days. Benztropine can also be used; however, it is not approved for children below 3 years of age. Close monitoring is important during treatment as dystonic reactions are occasionally accompanied by fluctuations in blood pressure and arrhythmias.

Pearls

1. The abrupt termination of the symptoms at the conclusion of the crisis or after the use of diphenhydramine is diagnostic and most striking.
2. In infants presenting with “seizures,” unusual behavior, eye deviation, and a history of reflux treated with metoclopramide, the possibility of OGC should be considered. Although the overall incidence of extrapyramidal effects associated with metoclopramide is 0.2%, pediatric and geriatric patients are affected more commonly, with an incidence as high as 10%. These side effects usually occur within a few days of initiation of the medication and are more common at higher doses.

SETTING-SUN PHENOMENON (SUNDOWNING)

Clinical Summary

The setting-sun phenomenon, also known as “sundowning,” is a concerning sign that may represent a pathologic increase in intracranial pressure in the young infant. The clinical presentation consists of an upward-gaze paresis with eyes that appear to be driven downward. There is usually sclera showing between the upper eyelid and the iris. Retraction of the upper eyelids, sometimes accompanied by raising of the brow, may be seen. Although this sign is commonly seen in children with obstructive hydrocephalus, it may also be seen as a result of intracranial hypertension of other causes (trauma or

ventriculoperitoneal shunt dysfunction) and occasionally in normal infants. It is thought to result from compression of the periaqueductal structures.

The phenomena could be an important sign in early detection of elevated intracranial pressure, appearing sooner than enlarged head circumference, full fontanelle, separation of sutures, irritability, or vomiting.



FIGURE 14.130 ■ Setting-Sun Phenomenon (Sundowning). The eyes appear driven downward in this infant with hydrocephalus. (Photo contributor: Stephen W. Corbett, MD.)

Management and Disposition

Suspect increased intracranial pressure and obtain neuroimaging urgently. Consult neurosurgery for guidance in initial management and for definitive treatment in the operating room.

Pearls

1. This sign is a valuable cue for obtaining prompt neuroimaging and urgent surgical intervention.
2. When persistent, this sign is a frequent marker of elevated intracranial

pressure, appearing in 40% of children with hydrocephalus (of any cause) and in 13% of patients with shunt dysfunction.

3. Ask parents to share photographs of their child taken since birth to assess for changes to head size and for the presence of sundowning.

KAWASAKI DISEASE

Clinical Summary

Kawasaki disease (KD), also known as mucocutaneous lymph node syndrome, is an acute self-limited medium- to small-vessel vasculitis of unknown etiology occurring most commonly in young children with a peak incidence between 1 and 2 years of age. The classic diagnosis is made clinically and is based on the presence of ≥ 5 days of fever and at least four of the following five principal clinical features without an alternative explanation:

1. Generalized polymorphous rash usually occurring within 5 days of the onset of fever and most commonly appearing as a nonspecific, diffuse maculopapular eruption. Early desquamation may occur in the perineal region, especially in infants.
2. Bilateral bulbar conjunctivitis sparing the limbus and most often nonexudative.
3. Changes to the lips and oral mucosa including dry, red, cracked lips, strawberry tongue, and diffuse erythema of the oropharyngeal mucosa.
4. Peripheral extremity changes: acute erythema of the palms and soles and/or swelling of the dorsal aspects of the hands and feet followed in 2 to 3 weeks by desquamation of the fingers and toes beginning in the periungual region. One to 2 months after the onset of the fever, deep transverse grooves across the nails (Beau lines) may appear.
5. Cervical lymphadenopathy that is usually unilateral, confined to the anterior cervical triangle, and with at least one node ≥ 1.5 cm in diameter.

The fever of KD is typically spiking and unremitting with peak temperatures often greater than 40°C (104°F). Incomplete (atypical) KD, which is more common in infants, does not meet all of the classic criteria but should be suspected in any child with unexplained fever for ≥ 5 days with two to three KD-associated clinical criteria or in infants (especially those ≤ 6 months of age) with

≥ 7 days of unexplained fever alone. Laboratory evaluation is necessary to support the diagnosis of incomplete KD and includes elevated ESR (≥ 40 mm/h) or CRP (≥ 3 mg/dL), and ≥ 3 supplemental laboratory criteria (albumin ≤ 3 g/dL, anemia for age, elevation of alanine aminotransferase, platelet count after 7 days $\geq 450,000/\text{mm}^3$, white blood cell count $\geq 15,000/\text{mm}^3$, and urine ≥ 10 white blood cells/high-power field). An abnormal echocardiogram may also aid in the diagnosis of incomplete KD.



FIGURE 14.131 ■ Kawasaki Disease. Irritability in an infant with Kawasaki disease. Note also the conjunctivitis and red, cracked lips. (Photo contributor: Tomisaku Kawasaki, MD.)

Coronary artery aneurysms or ectasias are the most important complication of KD and develop in 20% to 25% of untreated children and may lead to ischemic heart disease, myocardial infarction, or sudden death. KD may also cause abdominal pain, aseptic meningitis, uveitis, myocarditis, arthritis, liver dysfunction, and gallbladder hydrops. Severe complications include macrophage activation syndrome and “Kawasaki shock syndrome” due to myocardial dysfunction. The differential diagnosis for KD includes scarlet fever, staphylococcal scalded skin syndrome, toxic shock syndrome, viral infections (measles, adenovirus, enterovirus, Epstein-Barr virus), bacterial cervical lymphadenitis, drug hypersensitivity reaction, Stevens-Johnson syndrome, and acrodynea.

Management and Disposition

If suspected, consultation with a cardiologist or other specialist in KD is highly recommended for confirmation of diagnosis as well as treatment. A baseline echocardiogram should be obtained to document the status of the coronary arteries. Treatment includes hospitalization, administration of IVIG 2 g/kg infused over 10 to 12 hours, and initiation of high-dose aspirin 80 to 100 mg/kg/day in four divided doses. Approximately 80% to 90% of patients will defervesce with this regimen. Failure to respond is usually defined as persistent or recurrent fever \geq 36 hours after completion of the initial IVIG infusion. Retreatment with IVIG 2 g/kg is recommended. With IVIG treatment in the acute phase of the disease, the risk of coronary artery abnormalities is decreased to less than 5%. Consultation with a pediatric infectious disease or rheumatology specialist would be warranted in cases of refractory KD regarding treatment options (corticosteroids, tumor necrosis factor- α inhibitors, interleukin-1 inhibitors).



FIGURE 14.132 ■ Conjunctivitis. Note the intense redness of the bulbar conjunctiva seen in this patient with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)



FIGURE 14.133 ■ Lymphadenopathy. Visible cervical lymphadenopathy is seen in this child with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)



FIGURE 14.134 ■ Extremity Findings. Red swollen hands are present in this patient with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)



FIGURE 14.135 ■ Periungual Desquamation. This finding typically begins 2 to 3 weeks after the onset of Kawasaki disease, in contrast to perineal desquamation that occurs during the early course of the disease in infants. (Photo contributor: Tomisaku Kawasaki, MD.)



FIGURE 14.136 ■ Beau Lines. Deep transverse grooves across the nails may appear 1 to 2 months after the onset of fever. (Photo contributor: Tomisaku Kawasaki, MD.)

Pearls

1. The diagnosis of typical KD is clinical, but laboratory studies may be helpful in cases of suspected incomplete KD.
2. Children with KD are commonly irritable and difficult to console.
3. KD is the leading cause of acquired heart disease in children in the United States. Echocardiography should be considered in any infant less than 6 months of age with fever ≥ 7 days duration, laboratory evidence of inflammation, and no other explanation for the febrile illness.
4. Even when treated with high-dose IVIG regimens within the first 10 days of illness, 20% of children will develop at least transient coronary artery dilation, with 5% developing aneurysms.
5. Vaccination against influenza should be provided to all patients receiving aspirin therapy to avoid Reye syndrome.

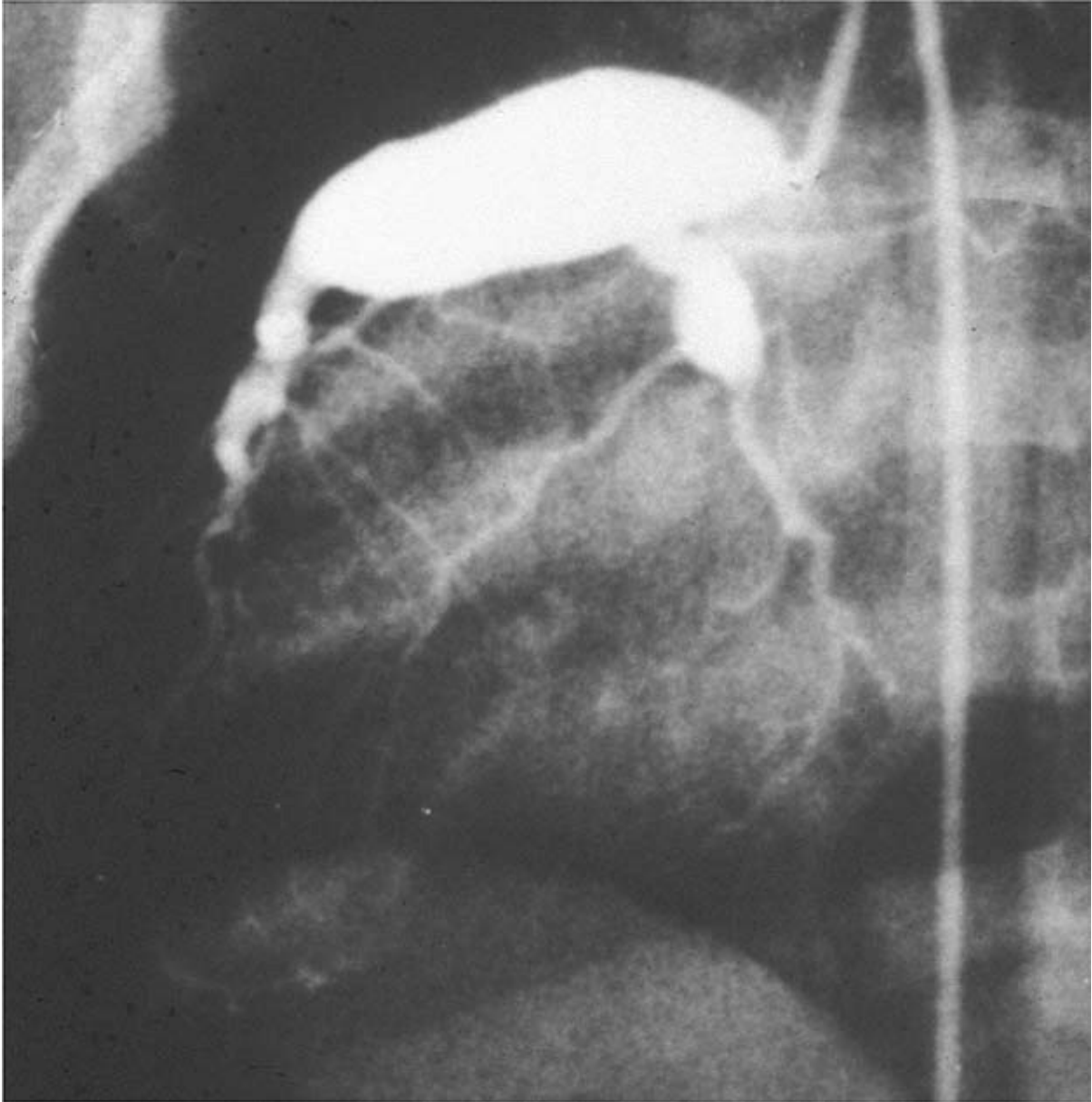


FIGURE 14.137 ■ Coronary Artery Aneurysms. A large coronary artery aneurysm is seen in this patient with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)

The authors acknowledge the special contributions of Javier A. Gonzalez del Rey, MD to the previous four editions.

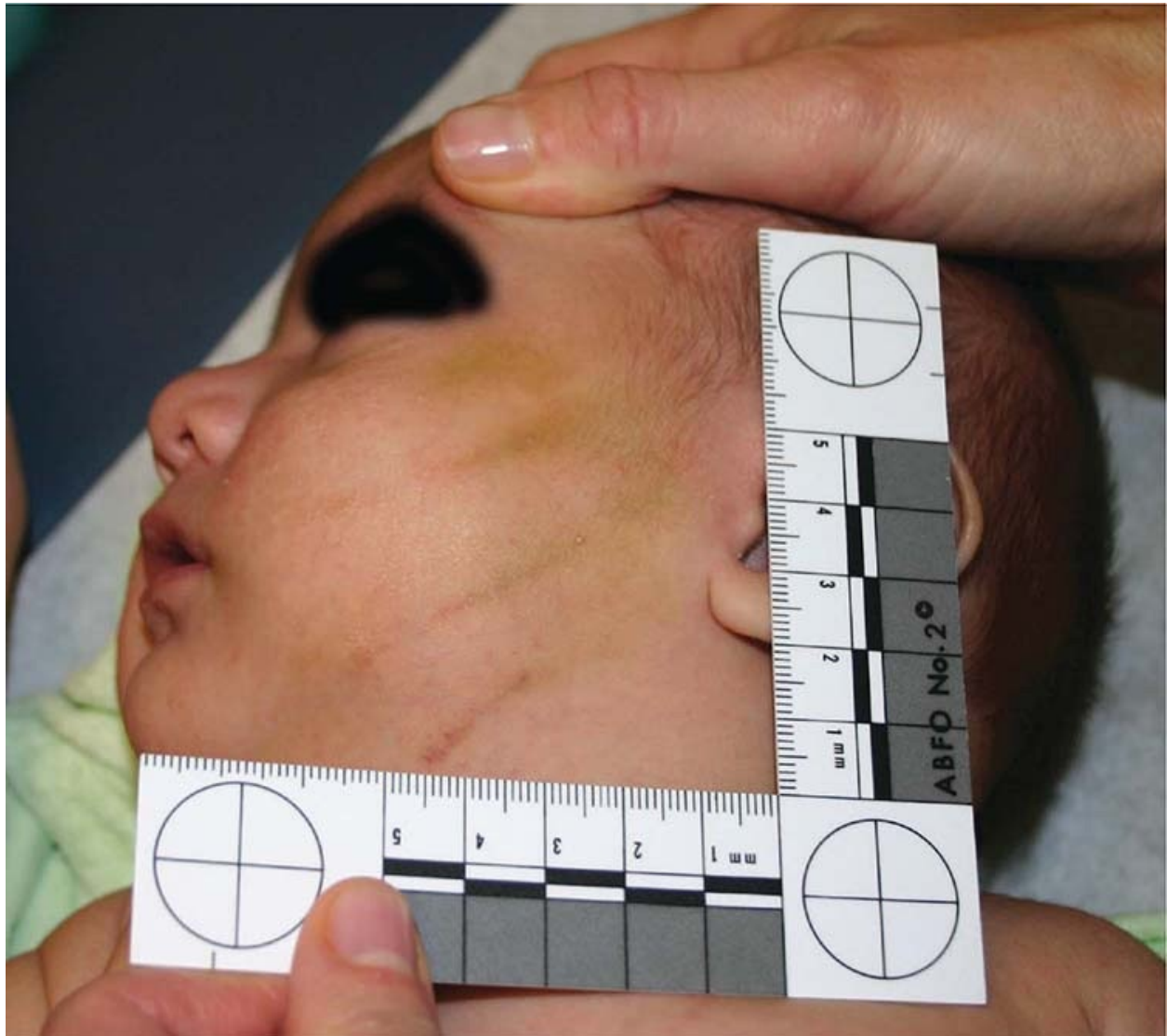
Chapter 15

CHILD ABUSE

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Physical Abuse

EXTERNAL FINDINGS

Clinical Summary

Until we can prevent all physical abuse, the goal of the emergency clinician is *early recognition*. In most cases, this will rely on recognizing “sentinel injuries” (relatively minor injuries that should prompt an evaluation for abuse) since the history is frequently incomplete or incorrect. External cutaneous or facial injuries are the most common abusive injuries and can be easy to dismiss because they may seem minor or self-limiting.

Serious physical abuse is much more common in children < 3 years old, who are generally unable to give their own history. Rates are especially high in young infants < 6 months old, perhaps because this is the peak incidence of colic or because of the difficulties faced by new parents. In this age group, almost any injury should prompt consideration of abuse and at least a thorough physical examination, including ears, oropharynx, genitals, entire skin, fontanel, and growth chart.

While bruises are common in toddlers and older children, bruising in children who are not yet pulling to stand (cruising) and in those < 6 months old is highly concerning. In children up to 4 years old, bruises are most concerning when they occur on the torso, ears, neck, cheek, or eyelid, or when they are patterned in the shape of an object.

In a child with burns, abuse should be considered with patterned injury, when the history of injury is inconsistent with burn severity or the child’s developmental abilities, or when the burn pattern suggests immersion. Immersion burns can include the extremities (stocking or glove distribution) or the torso, with the latter commonly occurring with unrealistic toileting expectations.



FIGURE 15.1 ■ Loop Marks. Subtle loop and linear marks are seen on the thigh and buttock of this child.
(Photo contributor: Robert A. Shapiro, MD.)



FIGURE 15.2 ■ Loop Marks. Loop and linear marks signify use of a cord or other similar object. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 15.3 ■ Pinch Marks. Fusiform marks formed by pinching. (Photo contributor: Daniel M. Lindberg, MD.)

Perhaps because the mouth and face are the site of so many stressful infant behaviors (crying, not eating), unexplained facial injuries (to the frenula, lips, palate, or sclera) should prompt thorough testing for abuse.



FIGURE 15.4 ■ Belt Pattern. This patterned bruise was inflicted by a woven belt. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.5 ■ Buttocks Bruising. Forceful spanking can cause buttocks bruising and longitudinal bruises parallel to the gluteal fold. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.6 ■ Adult Bite Marks. This adult bite mark demonstrates evidence of suction (hickey) within the bite mark itself. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.7 ■ Adult Bite Marks. This bite mark on a child's arm is consistent with an adult bite. While marks from individual teeth are not visible, facing arches of appropriate size are indicative of a bite mark. (Photo contributor: Megan McGraw, MD.)



FIGURE 15.8 ■ Child Bites. Multiple bite marks were inflicted on this child's face by another child. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

While certain findings (bite marks, patterned bruises) are very specific for abuse, cutaneous findings are not sensitive for predicting other injuries such as fractures, brain injury, or abdominal injury. The absence of bruising should not preclude screening for other injuries.

Management and Disposition

With the exception of significant burns, abusive external injuries rarely require significant treatment. Rather, children with these sentinel injuries should be screened for other abusive injuries, and reasonable concerns for abuse should be reported. In the United States and many other countries, medical professionals are mandated to report cases concerning for child abuse. When available, consult a child abuse team.



FIGURE 15.9 ■ Child Bite. Distinct impressions of teeth are seen in this injury with the outlines of the upper and lower oral arches. Note the size of the mother's mouth in relation to the size of the bite on the neck, making the adult an unlikely source. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 15.10 ■ Ligature Bruise. This child survived beating and attempted strangulation that resulted in circumferential, linear neck abrasions and occipital ecchymosis. (Photo contributor: Barbara R. Craig, MD.)



FIGURE 15.11 ■ Ligature Bruise. These bruises around the neck of this child represent ligature marks. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.12 ■ Gag Ligature. This child had a sock stuffed into his mouth and tied around his head. The bruises in the corners of the child's mouth are indicative of gagging. (Photo contributor: Robert A. Shapiro, MD.)



FIGURE 15.13 ■ Slap Mark. Note that the bruises run between and around the fingers, creating parallel lines of bruising. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.14 ■ Slap Mark. While this child does not show distinct outlines of fingers, nonetheless these parallel lines of bruising on the cheek suggest a slap injury. The perpetrator confessed to slapping this child. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.15 ■ Pinna Bruise. Bruising to the ear is concerning for abuse and should trigger a thorough evaluation for other abusive injuries. It is important to examine the entire ear. In this case, there is bruising to the posterior part of the pinna. (Photo contributor: Robert A. Shapiro, MD.)



FIGURE 15.16 ■ Pinna Bruise. Bruising to the inner auricle concerning for abuse. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.17 ■ Subconjunctival Hemorrhage. This 2-month-old presented after a reported fall from bed and was found to have facial bruising and this subconjunctival hemorrhage. Additional injuries identified included rib and arm fractures. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.18 ■ Hair Pulling Subgaleal Injury. This child has a large posterior subgaleal hemorrhage without associated skull fracture. It resulted from abusive hair yanking. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.19 ■ Forehead Hematoma with Eye Bruising. This child with hemophilia demonstrates the tendency of blood from a forehead hematoma to track downward with gravity and become visible in the periorbital region. In some cases, evidence of the forehead injury may be subtle. (Photo contributor: Ralph A. Gruppo, MD.)



FIGURE 15.20 ■ Oropharyngeal Injury. Fingers, utensils, or other objects can perforate the oropharynx when shoved into the mouth of young children in the course of feeding or in an attempt to pacify an infant. This should trigger a thorough evaluation for other abusive injuries. This x-ray demonstrates retropharyngeal air (arrows) indicating oropharyngeal perforation. (Photo contributor: Marguerite Caré, MD.)



FIGURE 15.21 ■ Palate Abrasions. This child presented with multiple pinch marks (see [Fig. 15.3](#)) and was found to have bilateral palate abrasions. Lesions like these can be seen with force-feeding, sexual abuse, or infectious etiologies. (Photo contributor: Daniel M. Lindberg, MD.)

To identify other occult injuries or medical mimics, order neuroimaging (computed tomography [CT] or magnetic resonance imaging [MRI]) for children < 6 months of age, skeletal survey for children < 2 years old, and hepatic transaminases (alanine transferase [AST]/aspartate aminotransferase [ALT]) for children < 5 years old. Order prothrombin time/partial thromboplastin time (PT/PTT) and complete blood count (CBC) and consider hematology consultation for children with concerning bruises or other findings that could indicate a coagulopathy. If a human bite is suspected, photo document and swab for potential DNA analysis.



FIGURE 15.22 ■ Palate Injury. Bruising to the soft palate is identified in this young adolescent girl who presented with concern for sexual assault. Bruising is consistent with penile-oral contact. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.23 ■ Labial Frenulum Tear and Lip Injury. Injuries of the labial or lingular frenula are concerning for abuse, especially in nonmobile children. The frenula should be specifically examined when there are other concerns for abuse, especially in infants. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.24 ■ Dental Injury. This 2-year-old child had been seen for an unexplained traumatic tooth loss before ultimately presenting with multiple abusive injuries. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.25 ■ Folk Remedies: Coining. This child has linear petechiae along her spine from the Southeast Asian practice of coining (Cheut Sah or Cao Gio) where a coin is rubbed along the skin to heal illness. This practice is neither painful nor dangerous and is not abusive. (Photo contributor: Charles Schubert, MD.)



FIGURE 15.26 ■ Folk Remedies: Cupping. Circular ecchymoses from the practice of “cupping” result when warm cups are placed on the skin to draw out illness. (Reproduced with permission from American Academy of Pediatrics. *Visual Diagnosis of Child Abuse*. 3rd ed. Elk Grove Village, IL: American Academy of Pediatrics; 1994.)



FIGURE 15.27 ■ Congenital Syphilis. X-rays raised concern for metaphyseal lesions (**A**) when this 1-month-old child presented with decreased movement of the arm. The child was in the midst of a workup of a rash that involved the trunk (**B**), extremities (**C**), and palms and soles (**D**). (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.28 ■ Henoch-Schönlein Purpura (HSP). Note palpable purpura on the extensor surfaces of the legs. HSP should be considered if there is symmetric ecchymosis along the extensor surfaces of the extremities and buttocks. Migratory arthritis and abdominal pain may be present. (Photo contributor: Ralph A. Gruppo, MD.)



FIGURE 15.29 ■ Dermal Melanosis (Mongolian Spots). These blue-gray congenital marks are more common in darker-skinned children, frequently found on the buttocks or low back, can be seen anywhere, and are distinguished from bruising, which resolves in days or weeks. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.30 ■ Pattern Burn. Multiple contact burns on the chest and abdomen from a curling iron. Accidental curling iron burns occur, but this number of injuries is very concerning for abuse. (Photo contributor: Robert A. Shapiro, MD.)



FIGURE 15.31 ■ Pattern Burn. This child was held against a heater grate. The pattern indicates that the child's leg was flexed at the time of injury. (Photo contributor: David W. Munter, MD.)

Pearls

1. Do not use the color or appearance of a bruise to estimate its age; bruise color is affected by injury type and patient complexion, as well as time from injury, and timing by color is unreliable.
2. If you suspect abuse, your minimal physical examination should include the entire skin, genitalia, ears, oral cavity, fontanel (if present), and growth chart. All infants (< 12 months old) need to be completely undressed for the exam.
3. Cigarettes can burn as hot as 400°C to 700°C. Inflicted cigarette burns are therefore usually ulcerated and full-thickness and rarely superficial.



FIGURE 15.32 ■ Immersion Burn. Immersion burns are often associated with toilet-training accidents. This girl was plunged into hot water after soiling herself. She shows sparing of the buttocks, which contacted the surface of the bathtub and avoided being burned. (Reproduced with permission from American Academy of Pediatrics. *Visual Diagnosis of Child Abuse*. 3rd ed. Elk Grove Village, IL: American Academy of Pediatrics; 1994.)



FIGURE 15.33 ■ Immersion Burn. A child's extremities may also be immersed. Burns in a "stocking" or "glove" pattern are concerning for an immersion mechanism. (Photo contributor: Cincinnati Children's

Hospital Medical Center.)



FIGURE 15.34 ■ Splash Burn. This toddler pulled a cup of hot liquid on to her. Note the “flowing” pattern that starts on her face, extends down her arm with burn depth decreasing as the liquid flows and cools. Note characteristic “skip” areas of unaffected skin. (Photo contributor: Cincinnati Children’s Hospital Medical Center.)



FIGURE 15.35 ■ Laxative “Burn.” Laxatives can cause skin irritation or sloughing, and often results in a diamond-shaped pattern. Blistering can also occur. (Photo contributor: Kathi L. Makoroff, MD.)

4. Burn mimics to consider include impetigo, phytophotodermatitis, and contact dermatitis. Bruise mimics to consider include hemophilia, von Willebrand disease, Henoch-Schönlein purpura (HSP), and idiopathic thrombocytopenic purpura (ITP).
5. Using the characteristics of a bite to identify the biter is controversial. Instead, swab human bites to identify the biter’s DNA. You can use the evidence collection tools in your standard “rape kit.”
6. In children with concerning injuries, do not rely on social factors or demeanor to identify abuse. When serious abuse is missed, it is commonly missed in affluent families with no identified social risks.
7. Focus on raising the concern of abuse, not on making a final conclusion about whether abuse occurred. The mandate to report concerns for abuse is triggered by “reasonable concern” not “absolute certainty.” The final determination of abuse often takes days, weeks, or months and may require data that are

unavailable in the emergency department.



FIGURE 15.36 ■ Scald Burn with Sparing. Superficial and partial-thickness burns were noted on the patient's anterior surface only. The areas of abdominal sparing indicate that the victim was flexed and curled at the time of injury. The child's caretaker, the mother's boyfriend, admitted to holding the child under a running hot-water tap. Partial-thickness burns on the penis and medial thighs are indicative of pooling of the liquid in those areas, resulting in a time-dependent injury. (Photo contributor: William S. Smock, MD.)



FIGURE 15.37 ■ Cigarette Burn—Inflicted. Cigarette burns are circular injuries with a diameter of about 8 mm. Children who accidentally run into a lit cigarette often have burns to the face or distal extremities. Accidental burns may be less distinct or deep compared with inflicted burns. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.38 ■ Cigarette Burn—Inflicted. This child suffered an inflicted cigarette burn to the ear. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.39 ■ Impetigo. These circular lesions of impetigo may be confused for healing cigarette burns. (Photo contributor: Michael J. Nowicki, MD.)

ABUSIVE HEAD TRAUMA

Clinical Summary

Abusive head trauma (AHT) is the most deadly form of physical abuse but can be easy to miss because the neurologic examination can be difficult in young infants, who are at highest risk. A low threshold for neuroimaging (CT or MRI) should be used for infants who present with bruising, vomiting without fever or diarrhea, fussiness or lethargy, or transient loss of consciousness (apparent life-threatening event [ALTE] and brief, resolved, unexplained event [BRUE]), especially those with increased head circumference or anemia.

Subdural hematoma is the most commonly recognized injury in AHT, but other injuries can include skull fractures, subarachnoid hemorrhage, or parenchymal injury. None of these injuries is seen exclusively with abuse; isolated, linear parietal skull fractures are most commonly accidental and can occur from relatively minor trauma. Abuse should be suspected when significant injury occurs after relatively minor trauma, such as a short fall, or when other abusive injuries are identified.

Be careful estimating the age of an injury using radiographic findings alone. The dogma that hyperdense “bright” blood is new and that hypodense “dark” blood is old is not reliable. The skull is made up of membranous bones and, unlike most other fractures, skull fractures do not display the normal healing patterns (periosteal reaction and callus formation) typical of endochondral bones.

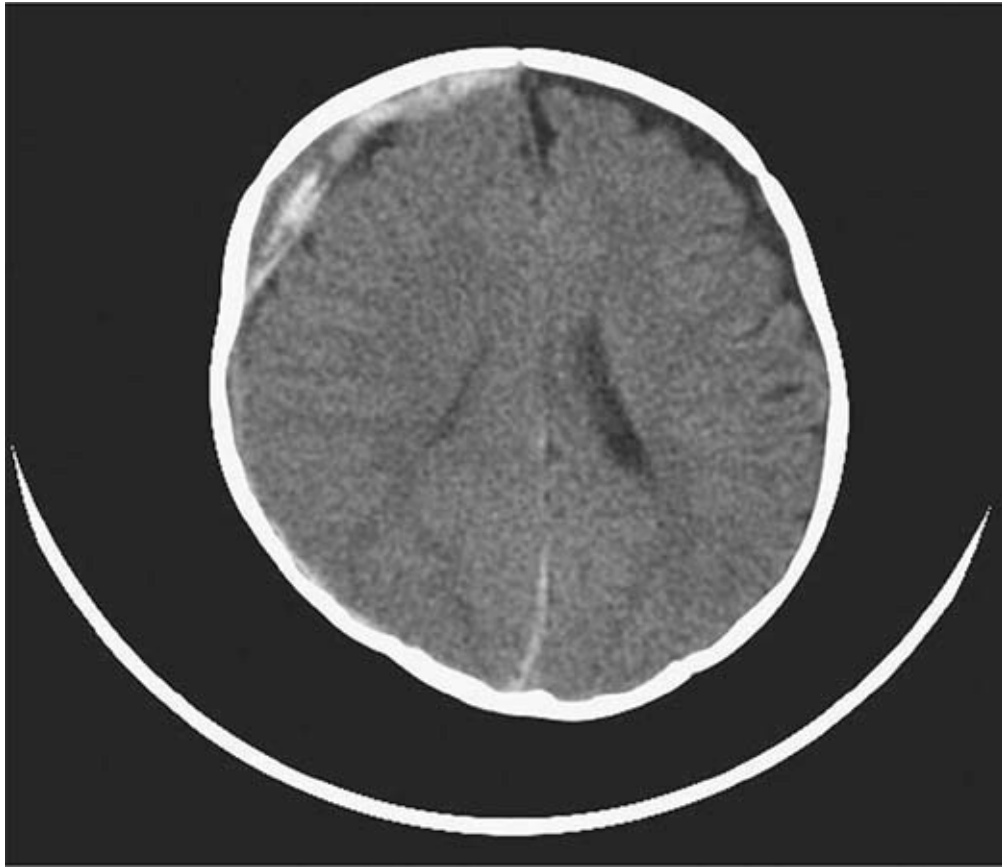


FIGURE 15.40 ■ Subdural Hematoma. There is a crescent-shaped, hyperdense collection, indicating a subdural hematoma over the right cerebral hemisphere. In addition, the right side of the brain demonstrates mass effect. (Photo contributor: Cincinnati Children’s Hospital Medical Center.)

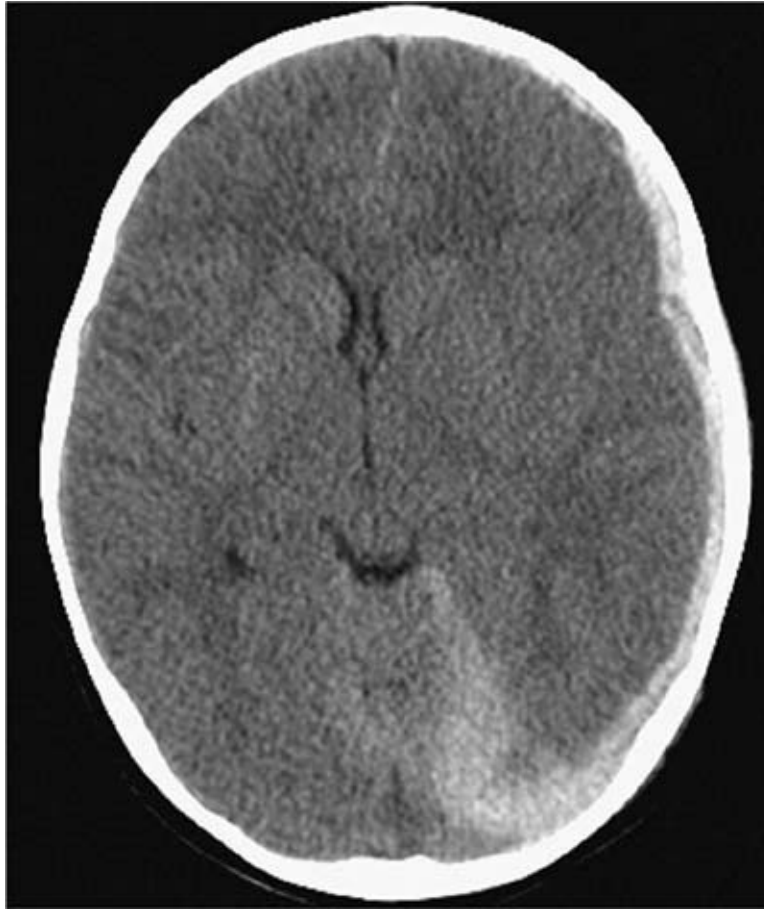


FIGURE 15.41 ■ Left Subdural Hematoma. There is a thin hyper-dense collection that extends along the entire left hemisphere as well as along the tentorium indicating a subdural hematoma. (Photo contributor: Marguerite Caré, MD.)



FIGURE 15.42 ■ Abusive Head Trauma. This CT shows right parietal skull fracture, subdural hematoma, and subarachnoid hemorrhage with effacement of the right-sided ventricles. (Photo contributor: Angie L. Miller, MD.)

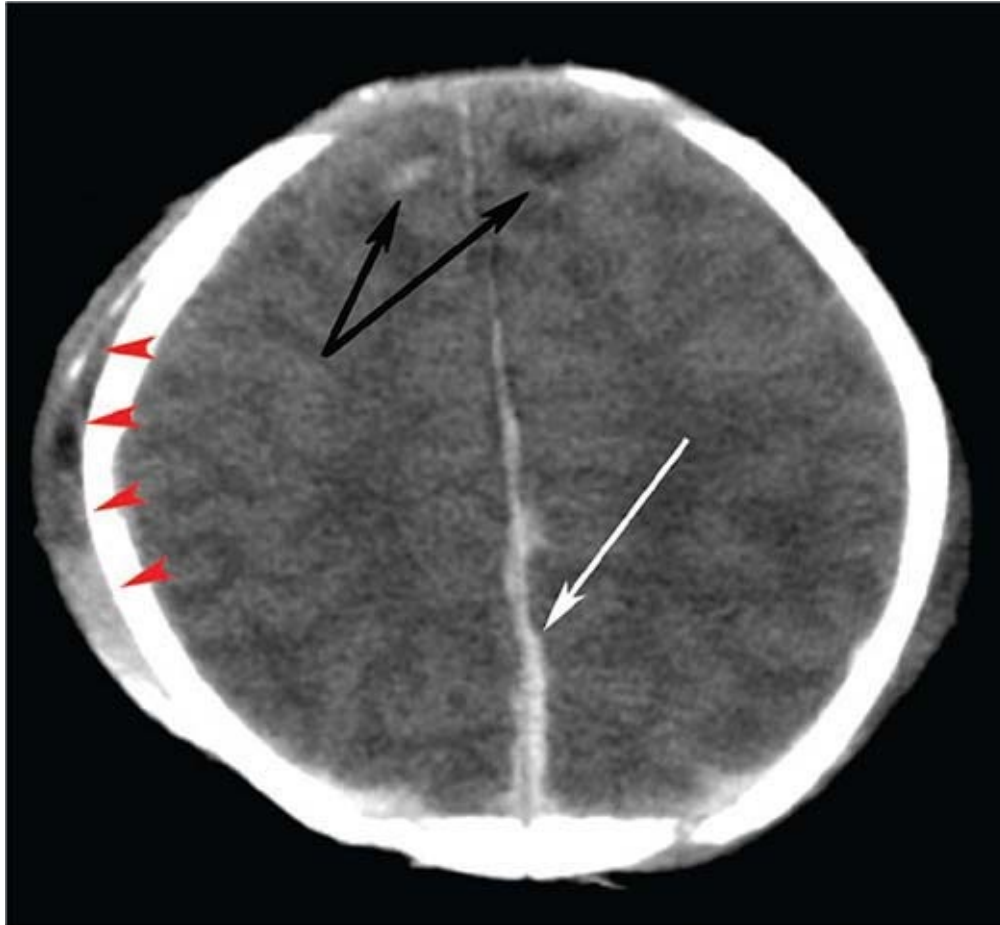


FIGURE 15.43 ■ Subdural Hematoma and Extra-Axial Swelling. This image shows interhemispheric subdural blood (white arrow) and subtle contusions (black arrows) of the frontal lobes. Extracranial soft-tissue swelling (arrowheads) is also present; sometimes this is visible on CT but not on physical examination. (Photo contributor: Marguerite Caré, MD.)



FIGURE 15.44 ■ Mixed Density Subdural Hematoma. Mixed density collections can result from multiple episodes of trauma, hyperacute bleeding, the mixing of blood and cerebrospinal fluid, or new bleeding into an established subdural collection. It is not possible in most cases to estimate the age of a subdural collection using the density of the collection on CT alone. (Photo contributor: Angie L. Miller, MD.)

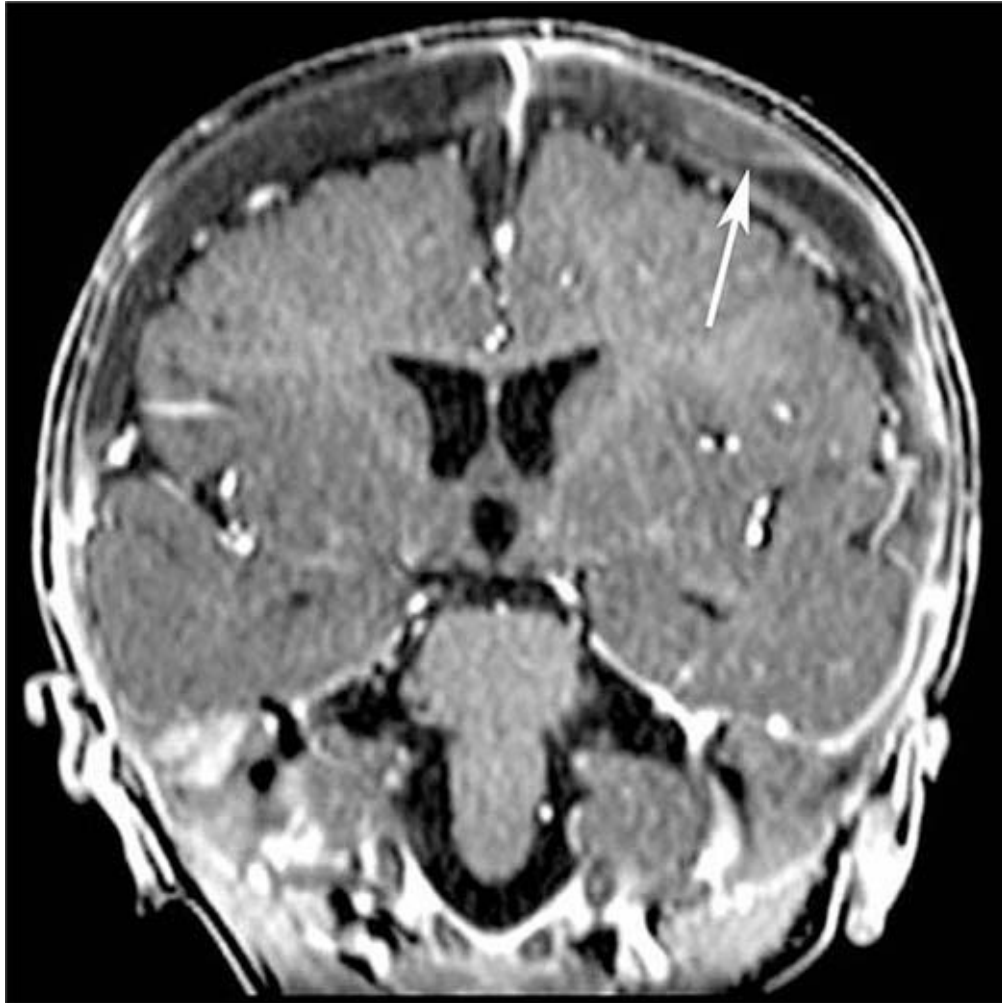


FIGURE 15.45 ■ Chronic Subdural Hematoma. This MRI demonstrates membrane formation (arrow) within the left-sided subdural hematoma. This is one of the most reliable indications that the subdural collection is at least several days old. (Photo contributor: Angie L. Miller, MD.)



FIGURE 15.46 ■ Epidural Hematoma. The left-sided, lens-shaped, hyperdense collection is an epidural hematoma, which is *less* commonly associated with abuse. (Photo contributor: Angie L. Miller, MD.)

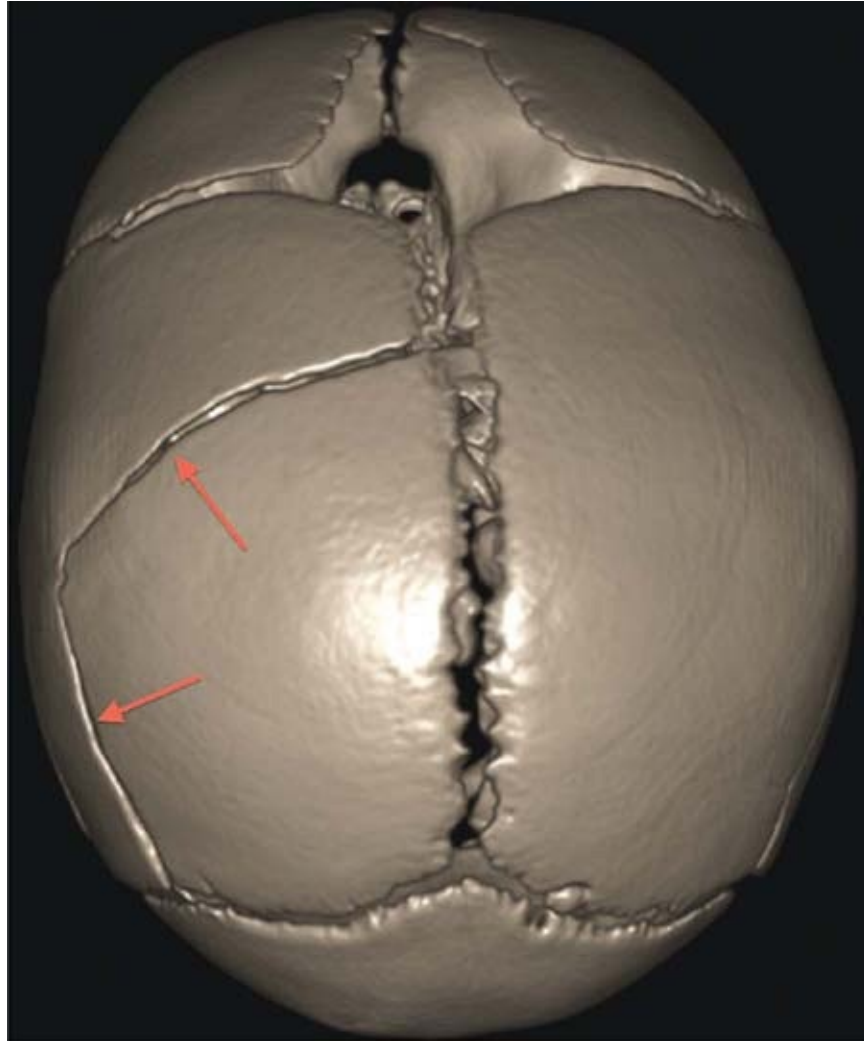


FIGURE 15.47 ■ Skull Fracture. This three-dimensional reconstructed image shows a left parietal skull fracture (arrows). Skull fracture can result from inflicted or accidental mechanisms. (Photo contributor: Marguerite Caré, MD.)

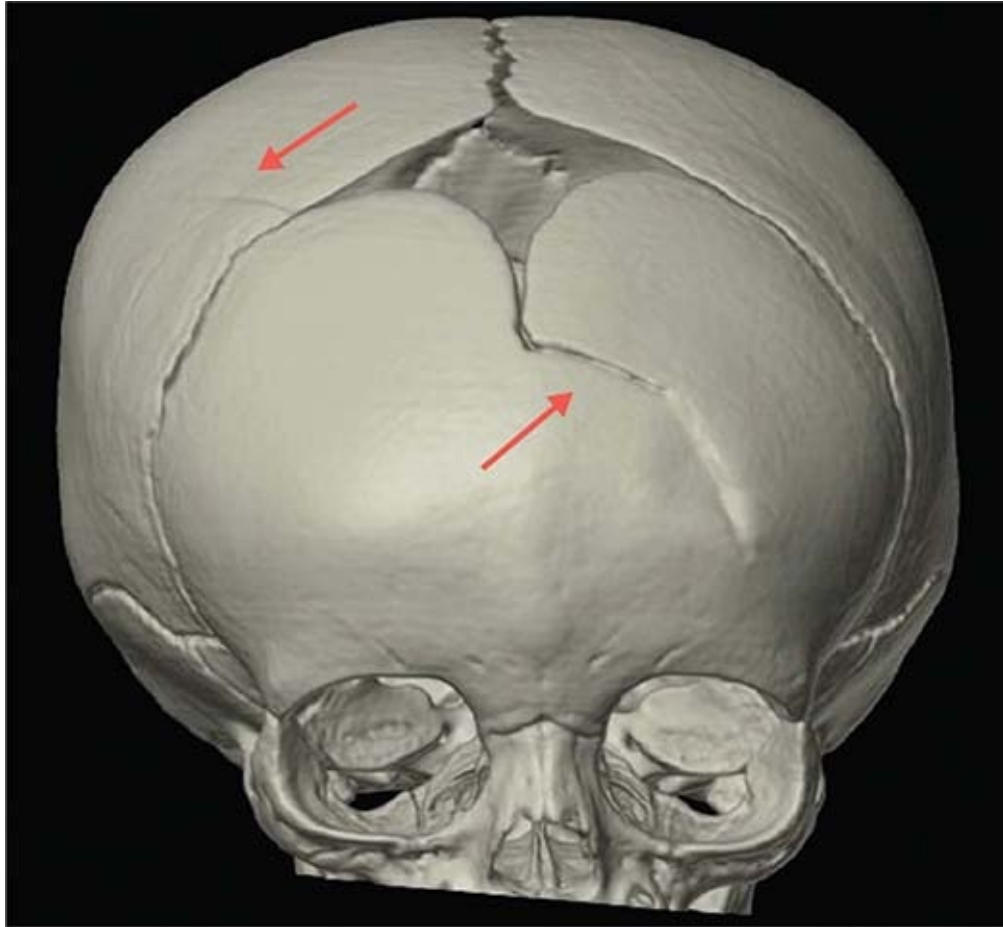


FIGURE 15.48 ■ Vertex Skull Fracture. This infant was accidentally dropped on her head. The three-dimensional reconstructed image shows two fractures that originated from the anterior fontanelle. It is possible that two distinct fractures resulted from a single impact to the vertex of the head. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.49 ■ Complex Skull Fracture. (A and B) These plain films demonstrate multiple diastatic fracture lines, suggesting high-energy trauma, crushing, or multiple impacts. (Photo contributor: Angie L. Miller, MD.)

Management and Disposition

The large majority of children with concern for AHT require hospital admission. Most will require retinal examination and subacute MRI scanning, and many centers have adopted routine electroencephalogram (EEG) because seizures are very common and can be difficult to appreciate clinically. As with all forms of physical abuse, children may require additional testing and investigation to determine the likelihood of abuse and to identify a safe outpatient environment.

Retinal hemorrhages that are numerous, diffuse, and in multiple retinal layers are strongly associated with AHT but have also rarely been seen with significant nonabusive trauma, such as crush injuries. Children with intracranial injuries and concern for abuse should have a dilated retinal examination by an experienced ophthalmologist, ideally within 24 to 48 hours, because some retinal hemorrhages can quickly resolve. Conversely, significant retinal hemorrhages are rare in children without intracranial injuries, and the retinal examination can often be omitted in these children.

In the absence of major trauma, subdural hematomas are very concerning for abuse and should trigger a thorough search for other abusive injuries, coagulopathy, or a history of abuse. A report of suspected child abuse should be made to children's services if there is a concern for AHT.



FIGURE 15.50 ■ Cervical Spine Epidural Hematoma (arrow). Soft-tissue cervical spine injuries are identified in a significant fraction of children with abusive head trauma, leading many centers to perform cervical spine MRI routinely in children who undergo inpatient MRI with concerns for abusive head trauma. (Photo contributor: Angie L. Miller, MD.)



FIGURE 15.51 ■ Retinal Hemorrhages. Multiple retinal hemorrhages are present in this image. A pediatric or general ophthalmologist can obtain a more complete view of the retina with dilated direct ophthalmoscopy and should be consulted to evaluate for retinal hemorrhages. (Photo contributor: Rees W. Sheppard, MD.)

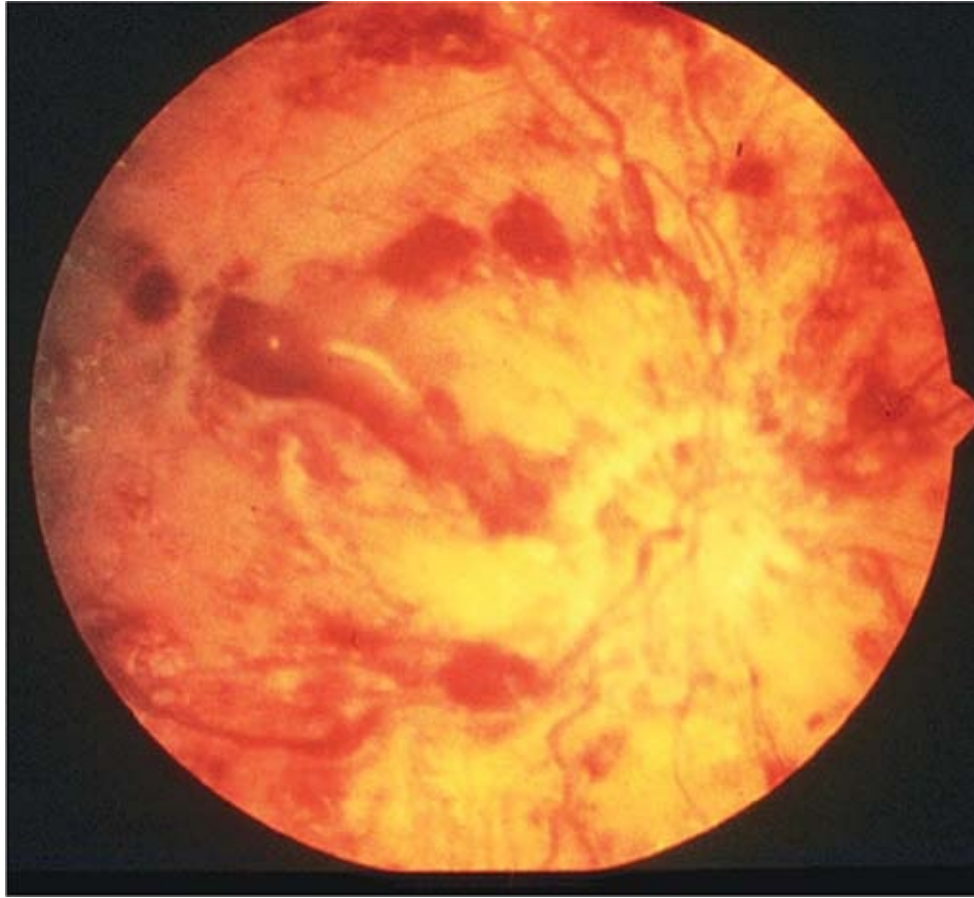


FIGURE 15.52 ■ Retinal Hemorrhages. Multiple discrete subhyaloid hemorrhages are seen. (Photo contributor: John D. Baker, MD, and Massie Research Laboratories, Inc.)

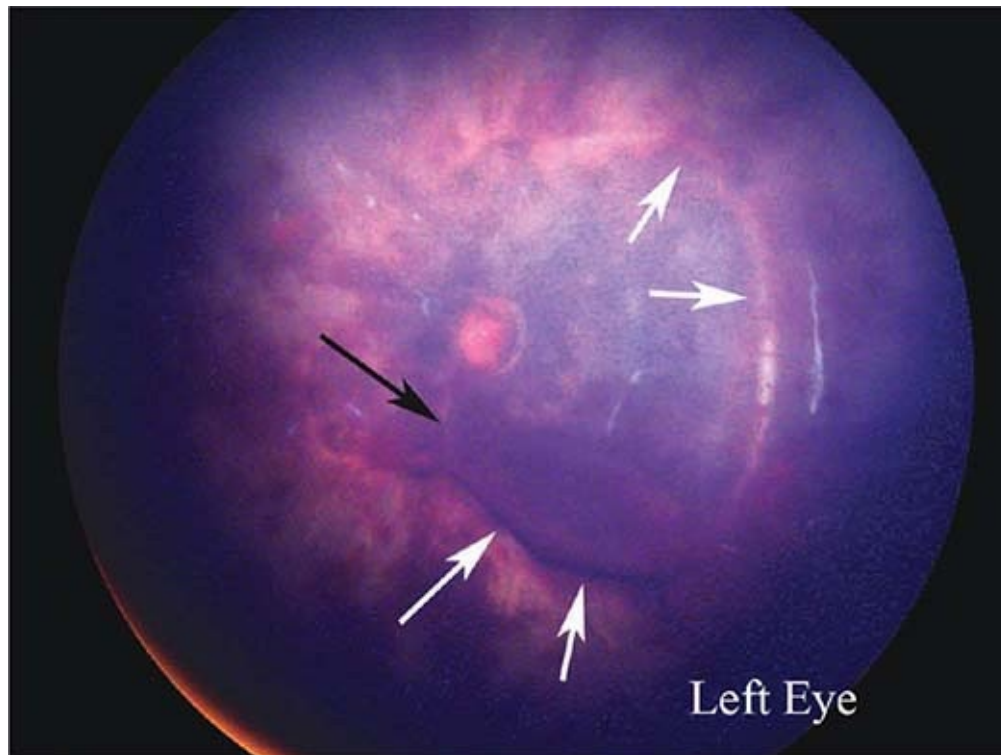


FIGURE 15.53 ■ Retinoschisis. This is the retina of a child with multiple, ultimately fatal abusive injuries. In addition to severe, characteristic retinal hemorrhages and large areas of preretinal bleeding (black arrow), there is a perimacular fold (white arrows; fold is obscured inferiorly by blood), which demonstrates retinoschisis, or separation of retinal layers. This finding has only been described in cases of abusive head trauma and rare cases of severe accidental crush injuries. (Photo contributor: Daniel M. Lindberg, MD.)

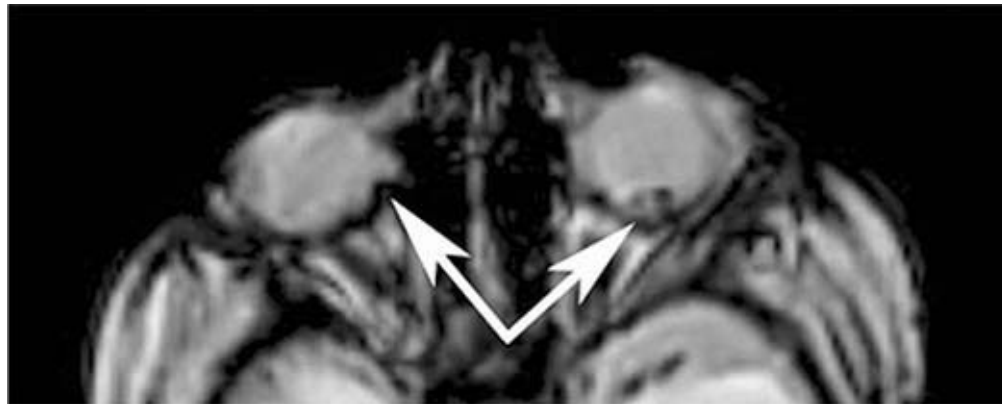


FIGURE 15.54 ■ Retinal Hemorrhages. Severe retinal hemorrhages can sometimes be appreciated on MRI (arrows), as in this child, using susceptibility-weighted sequences. MRI is not as sensitive as dedicated retinal exam and should not be used to screen. (Photo contributor: Angie L. Miller, MD.)

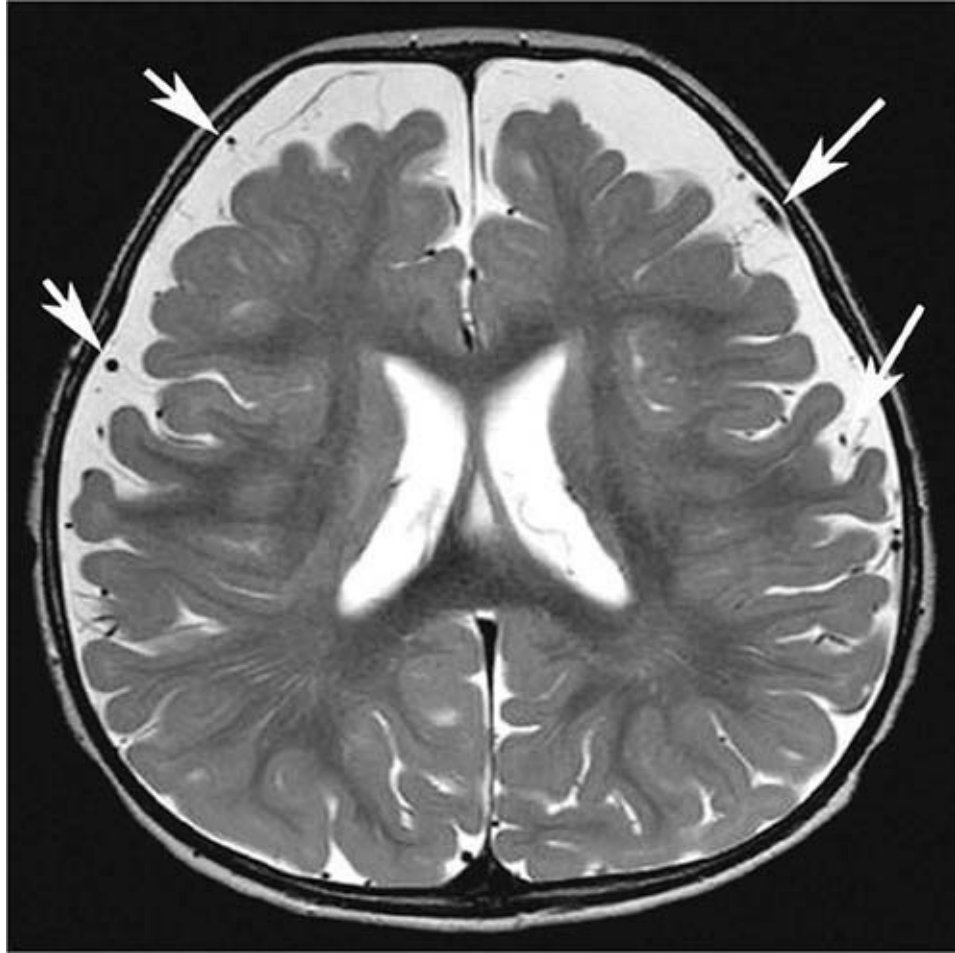


FIGURE 15.55 ■ Benign Extra-Axial Fluid. This axial T2-weighted MRI shows enlarged subarachnoid spaces. This nontraumatic condition is not uncommon in children with macrocrania and can be distinguished from hypodense subdural hematoma by the identification of blood vessels running through the collections (arrows). This is more easily accomplished using MRI than CT. (Photo contributor: Angie L. Miller, MD.)

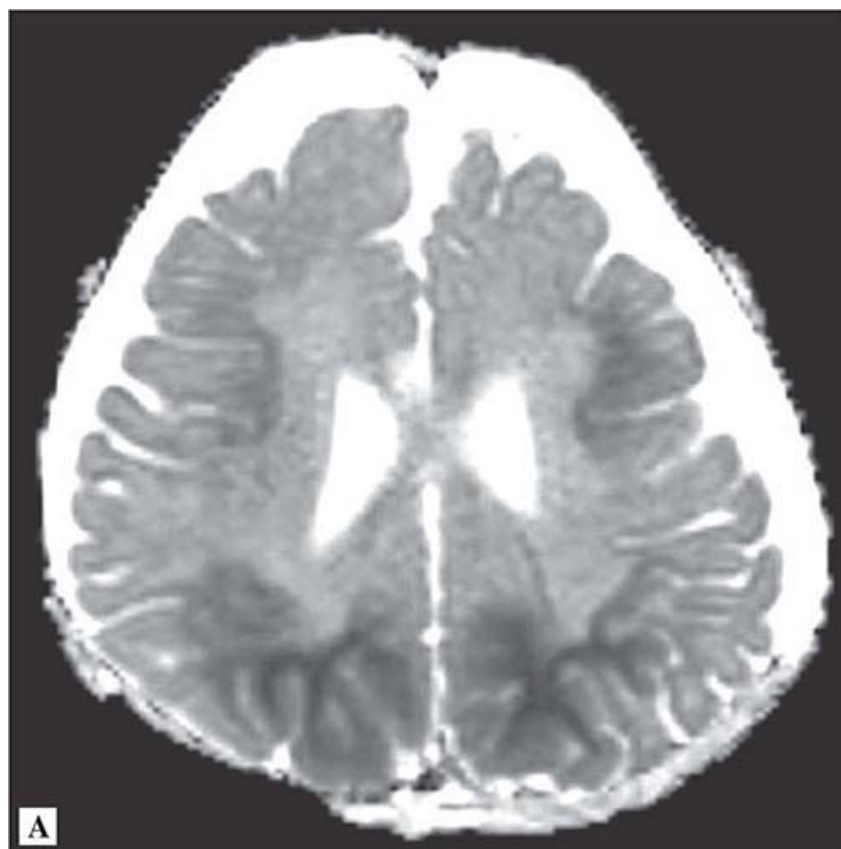


FIGURE 15.56 ■ Restricted Diffusion. This MRI demonstrates restricted diffusion within the brain parenchyma of a child with AHT. Symmetric restricted diffusion in the occipital and parietal region is dark on the apparent diffusion coefficient (ADC) sequence (**A**) and bright on the diffusion-weighted sequence (**B**). This parenchymal injury is common in children with AHT and is associated with seizures. (Photo contributor: Angie L. Miller, MD.)

Pearls

1. Think about abuse when a significant brain injury is attributed to a short fall, a young sibling, or other minor traumatic mechanism.
2. Infants and children with AHT may have no external signs of trauma and nonspecific or no neurologic deficits.
3. Significant retinal hemorrhages are exceedingly rare if neuroimaging is negative. Dedicated retinal exam may be omitted if traumatic brain injury is absent.
4. Three-dimensional skull reconstruction should be routine for all head CTs obtained in infants because skull fractures can be missed by plain skull films or by CT, especially if the fracture is horizontal, in the axial plane of the CT.
5. Significant retinal hemorrhages can sometimes be seen with MRI (especially using susceptibility-weighted imaging), but this should not preclude a dedicated retinal exam.
6. Nonabusive brain injuries can be seen with significant accidental trauma, in children with bleeding disorders, in home births, or in children who do not receive perinatal vitamin K.

SKELETAL FINDINGS

Clinical Summary

Fractures are the second most common abusive injury, behind only cutaneous injuries. Like other abusive injuries, fractures can be very difficult to identify clinically, and for this reason, a radiographic skeletal survey is recommended for most children < 24 months old (and for many up to 36 months) when abuse is being considered. To be useful, a skeletal survey needs to be conducted according to published guidelines, which require high-resolution technique and approximately 20 different dedicated films. When performed properly, a skeletal survey uses only 0.2 mSv of radiation, so transfer to an experienced pediatric

center is almost always to be preferred over performing an informal survey or a single-view “babygram” that does not conform to guidelines.

More than any other injury, radiographic signs of healing can be used to estimate the age of fractures, with periosteal reaction and callus formation commonly occurring between 7 and 14 days after injury. While no fracture is pathognomonic for abuse, rib fractures, classic metaphyseal fractures, and multiple fractures of different ages are highly concerning, and should prompt a thorough abuse evaluation. Fractures of the sternum, scapula, spine, pelvis, hands, or feet are uncommon in young children and should prompt a concern for abuse when there is not a clear history of significant trauma.



FIGURE 15.57 ■ Classic Metaphyseal Lesions (CMLs). Note multiple, bilateral CMLs in both distal femurs and both proximal and distal tibias, with the proximal humerus, these are the most common locations for CMLs. (Photo contributor: Angie L. Miller, MD.)

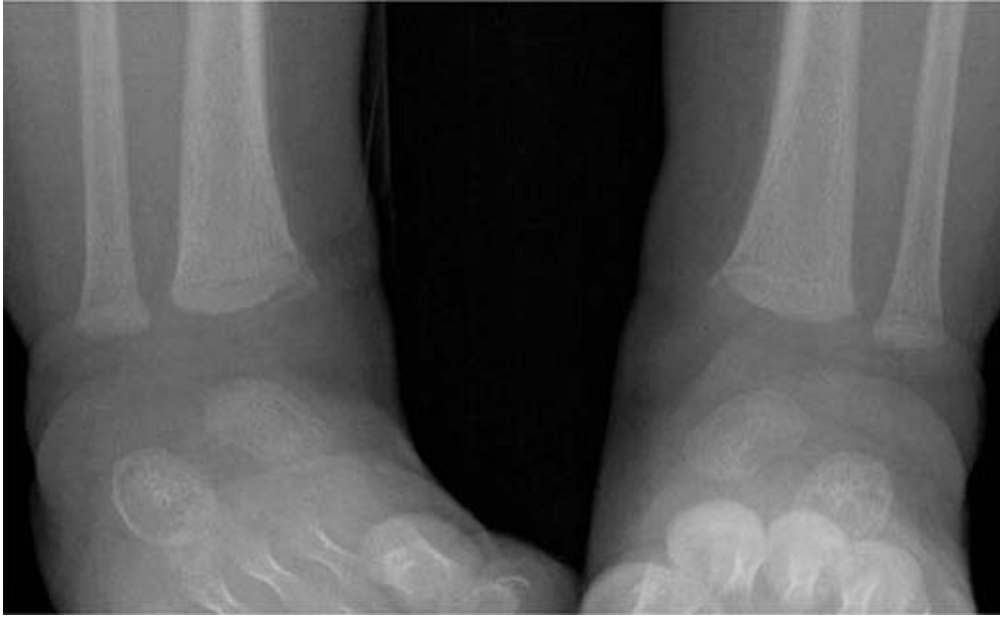


FIGURE 15.58 ■ Classic Metaphyseal Lesions (CMLs). This child had bilateral CMLs of the distal tibias, with bucket-handle morphology. (Photo contributor: Angie L. Miller, MD.)

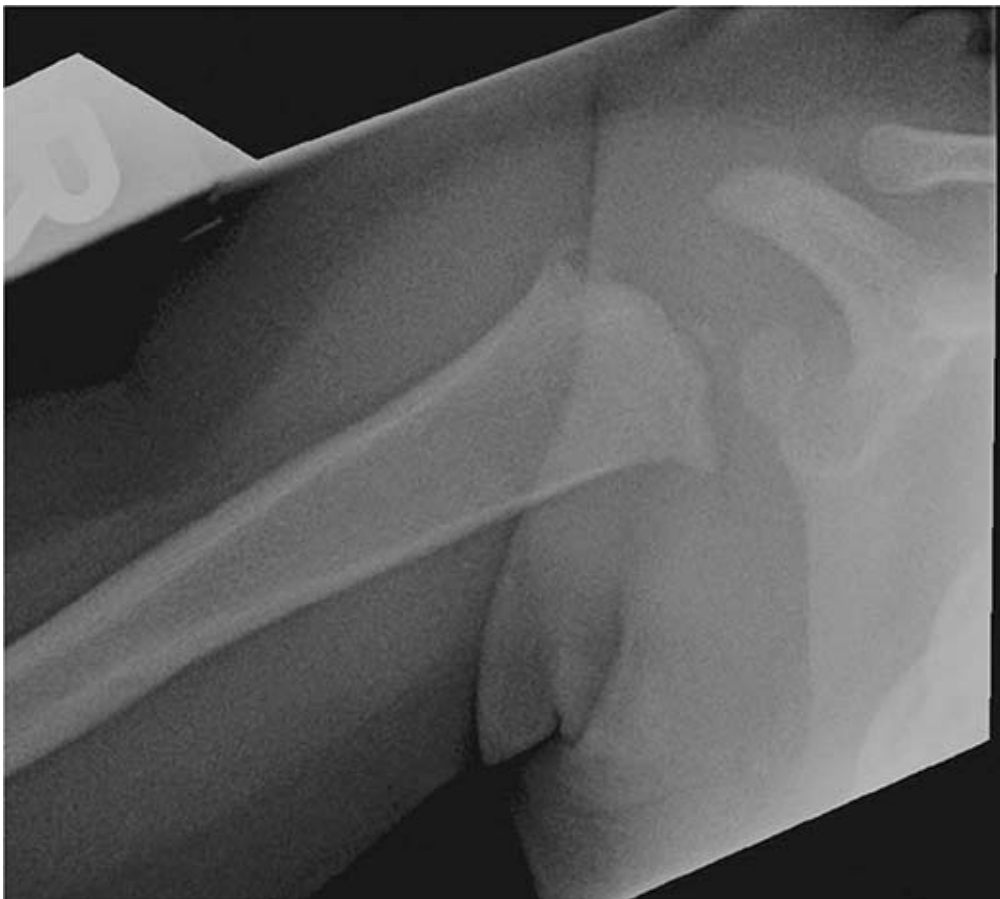


FIGURE 15.59 ■ Classic Metaphyseal Lesion (CML). Skeletal survey demonstrates a bucket-handle morphology of this metaphyseal fracture of the proximal humerus. (Photo contributor: Cincinnati Children's)

Management and Disposition

As with any abusive injury, abusive fractures should prompt testing for other abusive injuries or medical disease, should be reported to child protective services, and require the identification of a safe environment prior to discharge. Fractures that are most concerning for abuse (rib fractures and classic metaphyseal fractures) do not usually require splinting or casting and are commonly self-limited. Because healing can identify fractures that are missed initially, a follow-up skeletal survey is commonly obtained at least 10 to 14 days after the initial survey when the initial survey is indeterminate or when there is moderate or high concern for abuse. Films of the skull, spine, and pelvis are usually omitted from these follow-up surveys, unless there is concern for injury on the initial skeletal survey. Nuclear medicine bone scans have largely been replaced by the follow-up skeletal survey but are still used in some centers or, rarely, when follow-up skeletal survey cannot be obtained.



FIGURE 15.60 ■ **Classic Metaphyseal Lesion (CML).** Two views of this metaphyseal fracture (**A, B**)

demonstrate that the “corner fracture” or “bucket-handle” morphology of these fractures is a function of the view obtained, rather than properties intrinsic to the fracture itself. (Photo contributor: Cincinnati Children’s Hospital Medical Center.)

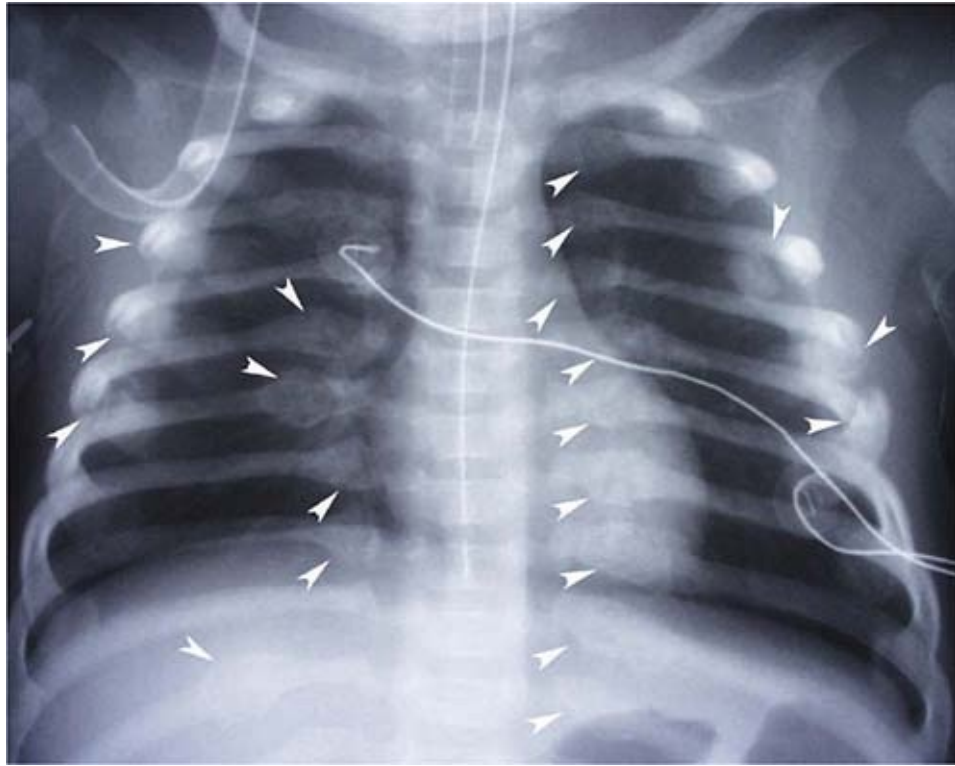


FIGURE 15.61 ■ Posterior Rib Fractures—Healing. This antero-posterior chest x-ray demonstrates multiple, bilateral, acute, and healing rib fractures in the posterior portion of the ribs adjacent to the spine (arrowheads). Posterior fractures result from levering of the ribs against the transverse processes of the vertebrae. (Photo contributor: Cincinnati Children’s Hospital Medical Center.)



FIGURE 15.62 ■ Rib Fractures—Acute and Healing. This oblique radiograph demonstrates acute fractures of ribs 6 and 8 and a healing fracture of rib 7, implying multiple episodes of trauma. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

Pearls

1. The physical examination is not sensitive for bony injury, especially in young infants. Bruising is absent in the majority of abusive fractures.
2. Skeletal survey should be obtained for most children < 24 months old with concern for physical abuse. Skeletal surveys are less likely to be helpful in children > 60 months (5 years).
3. Radiographic signs of healing (periosteal reaction, callous) are typically seen approximately 7 to 14 days from the time of injury.



FIGURE 15.63 ■ Epiphyseal Separation. This film shows a healing, abusive fracture of the distal humerus. Technically a displaced Salter-Harris I fracture, the lesion extends through the growth plate and results in separation and displacement of the epiphysis relative to the metaphysis, seen here as posterior displacement of the capitellum relative to the distal humerus metaphysis. These injuries require significant force and should raise concern for abuse in the absence of a history of significant trauma. (Photo contributor: Susan Scherl, MD.)



FIGURE 15.64 ■ Healing Fracture. This 24-day-old's birth-associated clavicle fracture shows signs of normal healing with periosteal reaction and callus formation. (Photo contributor: Angie L. Miller, MD.)

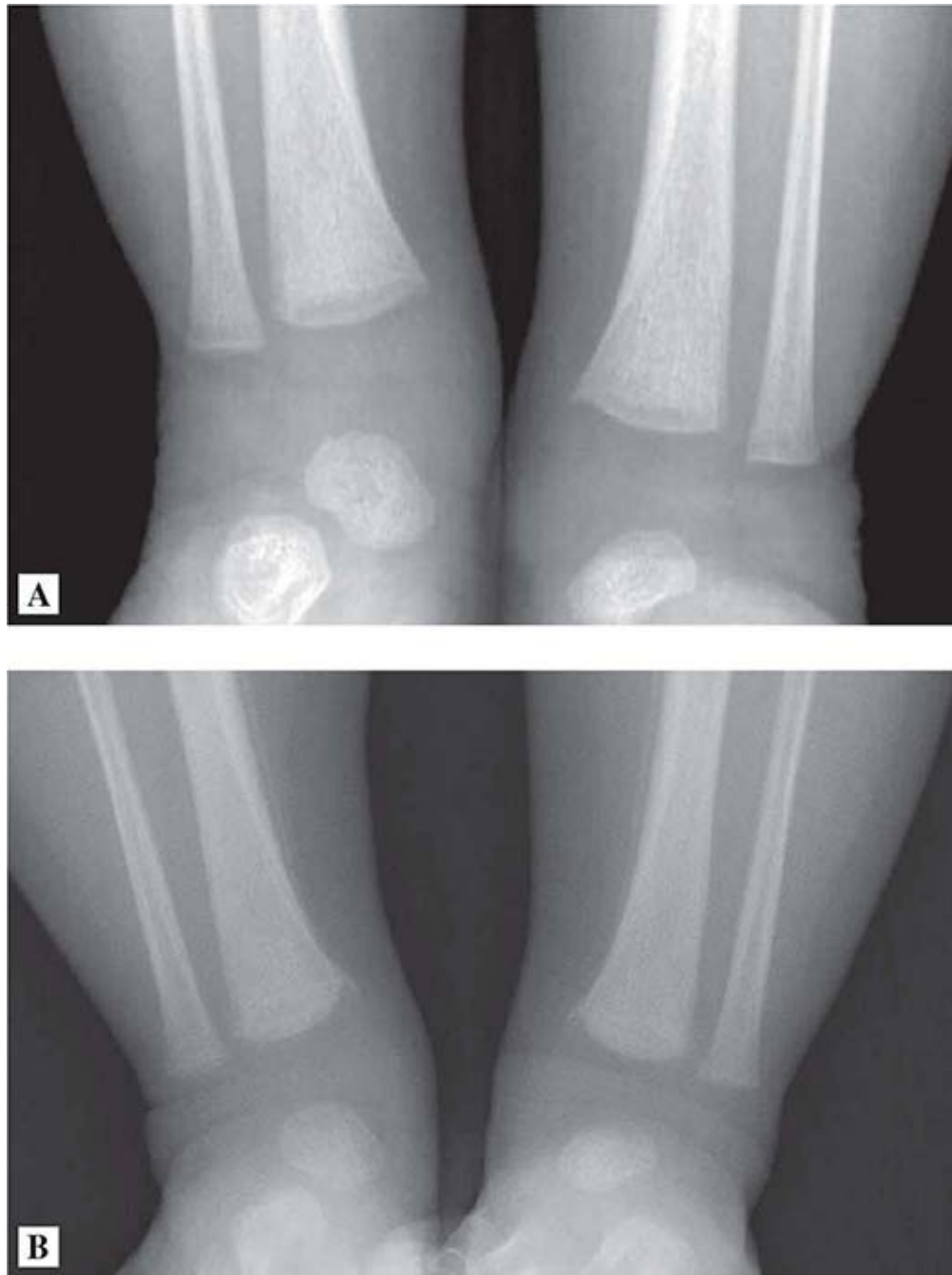


FIGURE 15.65 ■ False-Negative Bone Scan. Technetium-based nuclear bone scans were historically used to identify occult fractures. These have been almost completely replaced in most centers by follow-up skeletal survey obtained at least 14 days after the initial skeletal survey. Fluoride-based (^{18}F) bone scans have increased resolution relative to technetium and can sometimes be used when the initial skeletal survey is inconclusive and it is not possible to wait for follow-up skeletal survey. Even ^{18}F bone scans are limited, however, in the evaluation of metaphyseal injuries. This young infant's initial skeletal survey (**A**) was inconclusive for a classic metaphyseal injury of the right distal tibia. An ^{18}F bone scan was negative for injury with symmetric uptake in the distal tibial metaphyses. However, follow-up skeletal survey (**B**) showed bilateral healing metaphyseal fractures with periosteal reaction along the medial tibias. (Photo contributor: Angie L. Miller, MD.)

4. Suspect abuse when a child has metaphyseal fractures, rib fractures, fractures at different stages of healing, or unsuspected fractures.
5. Toddler's fractures (spiral fractures of the tibia) are nonabusive fractures common in children learning to walk.
6. When fractures are the only abusive injury type identified, consider bone fragility disorders including osteopenia of prematurity (in children with significant prematurity), rickets (look for radiographic signs), and osteogenesis imperfecta (consider testing and look at the sclera, teeth, growth chart, and family history).



FIGURE 15.66 ■ Blue-Gray Sclera. This school-aged child has osteogenesis imperfecta and a history of multiple fractures. While some patients demonstrate the classic “robin’s egg blue” sclera, a blue-gray tint is more common, and some patients have normal scleral pigmentation. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.67 ■ Osteogenesis Imperfecta (OI). This child with OI demonstrates osteopenia and bony deformities related to multiple, healing fractures. Even within families, OI has variable penetrance and x-

ray is not sufficient to exclude the diagnosis. (Photo contributor: Angie L. Miller, MD.)



FIGURE 15.68 ■ Osteomyelitis. This young infant presented with decreased use of the arm in the setting of low-grade fever and multiple social risk factors. This x-ray, showing a metaphyseal lesion, initially prompted concern for abuse. The lytic nature of the lesion, with bony destruction, led to the ultimate diagnosis of group B *Streptococcus* osteomyelitis. Osteomyelitis should be considered in children with even low-grade fever when the main indication of abuse is bony lesions that are not clearly traumatic. (Photo contributor: Angie L. Miller, MD.)

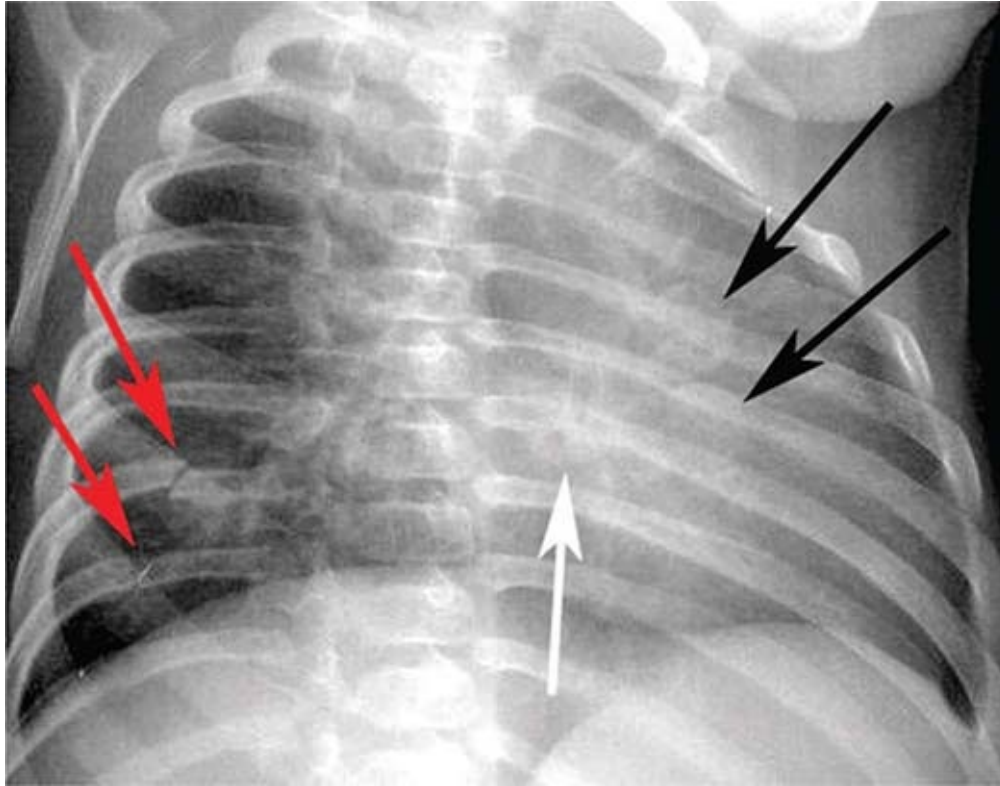


FIGURE 15.69 ■ Sternal Ossification Centers. This oblique view results in overlap of the round sternal ossification centers and the ribs, which can sometimes appear as if the child has healing rib fractures (black arrows). Look for this effect when fractures are seen in oblique, but not other, views. Notably, this child also has acute (right seventh and eighth; red arrows) and healing (left sixth) posterior rib fractures. (Photo contributor: Laura Z. Fenton, MD.)



FIGURE 15.70 ■ Skeletal Changes with Rickets. Cupping of the metaphysis (proximal to the growth plate), as shown here in both the radius and ulna, is one of the earliest signs of rickets or calcium deficiency (white arrows). Periosteal reaction is demonstrated along the shaft of both bones (red arrows). (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.71 ■ Toddler's Fracture. The toddler's fracture is a commonly seen, nonabusive, spiral fracture of the distal tibia that often presents in children learning to walk, sometimes without a specific history of trauma beyond the normal falls associated with this developmental stage. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.72 ■ Phalanx Fracture (Second, Third Metacarpal). Fractures to the phalanges of the fingers or toes are rare in both accidents and abuse. Without a history of significant trauma, identification of these fractures, which may be very subtle, should prompt a thorough evaluation for child abuse. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

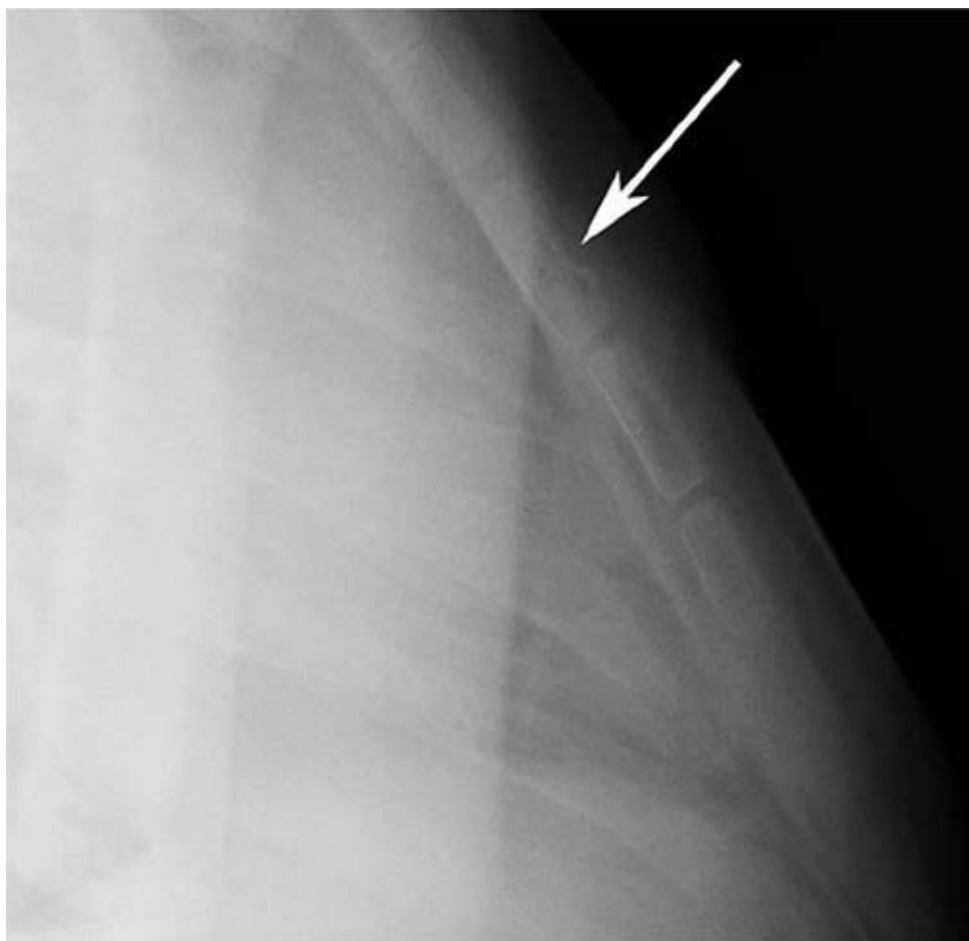


FIGURE 15.73 ■ Sternal Fracture. This lateral view shows a buckle fracture of the anterior, upper sternum (arrow). Like fractures of the scapula and fingers, these are very concerning for abuse unless there is a clear history of significant trauma. (Photo contributor: Angie L. Miller, MD.)



FIGURE 15.74 ■ Vertebral Fracture. The wedging of T-12 (arrow) and probably L-1 indicates vertebral compression fractures. These are the result of significant forces applied to the spinal column and often indicate child abuse. (Photo contributor: Alan E. Oestreich, MD.)



FIGURE 15.75 ■ Vertebral Fracture. Anterior wedging and marrow edema on MRI demonstrate bilevel spinal fractures (arrows). Without a clear history of significant trauma, this is highly concerning for abuse. (Photo contributor: Angie L. Miller, MD.)

VISCERAL FINDINGS

Clinical Summary

Intra-abdominal and intrathoracic injuries are identified in a small but important fraction of abused children. These injuries can be deadly and can significantly affect the plausibility of an offered history for other injuries. Clinical signs of abdominal injury (tenderness, bruising, distention, altered bowel sounds) are relatively specific but relatively insensitive. Hepatic transaminases (AST and

ALT) can identify abdominal injuries that are missed by clinical examination alone and should be obtained in children < 5 years old with concern for physical abuse and a significant injury. When AST or ALT are > 80 IU/L, abdominal CT with intravenous (IV) contrast should be obtained to identify intra-abdominal injury.

Significant intra-abdominal injuries are uncommonly the result of short falls or stairway falls, although relatively low-energy injuries with a direct blow to the abdomen (eg, a fall onto handlebars) can produce significant injuries.

Management and Disposition

Most solid organ injuries in well-appearing children are self-limited, but hollow viscus injuries, pancreatic injuries, and vascular injuries have high likelihood of deterioration and require early surgical consultation. Children with identified intra-abdominal or intrathoracic injuries require admission for observation and surgical consultation. The identification of abusive intra-abdominal or intrathoracic injuries should prompt screening for other abusive injuries with a careful physical examination, skeletal survey (for children < 24 months old), and neuroimaging (for children < 6 months old or with signs of brain injury).



FIGURE 15.76 ■ Abdominal Bruises. Abdominal bruising is unlikely in accidental injury and frequently indicates intra-abdominal injury. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.77 ■ Positive Focused Assessment with Sonography for Trauma (FAST) Examination. This anechoic (dark) fluid in Morrison pouch represented blood in this patient. As with nonabusive trauma, ultrasound is a rapid, noninvasive way to screen for abdominal injury in an unstable patient but is not sufficiently sensitive to detect smaller abdominal injuries that may, nevertheless, have important forensic significance. (Photo contributor: Jason W. Fischer, MD, MSc.)

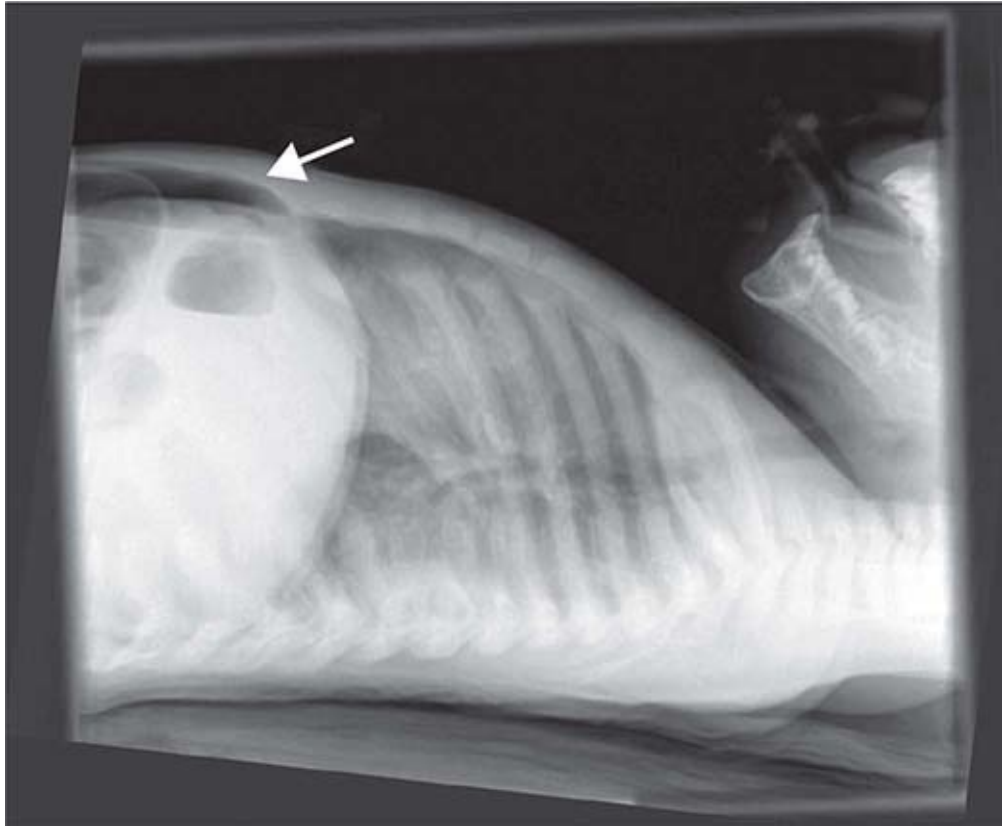


FIGURE 15.78 ■ Hollow Viscus Perforation. This lateral chest x-ray demonstrates free air anteriorly, inferior to the diaphragm, suggesting hollow viscus perforation, an injury that, like pancreatic injuries, is overrepresented in abused children. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

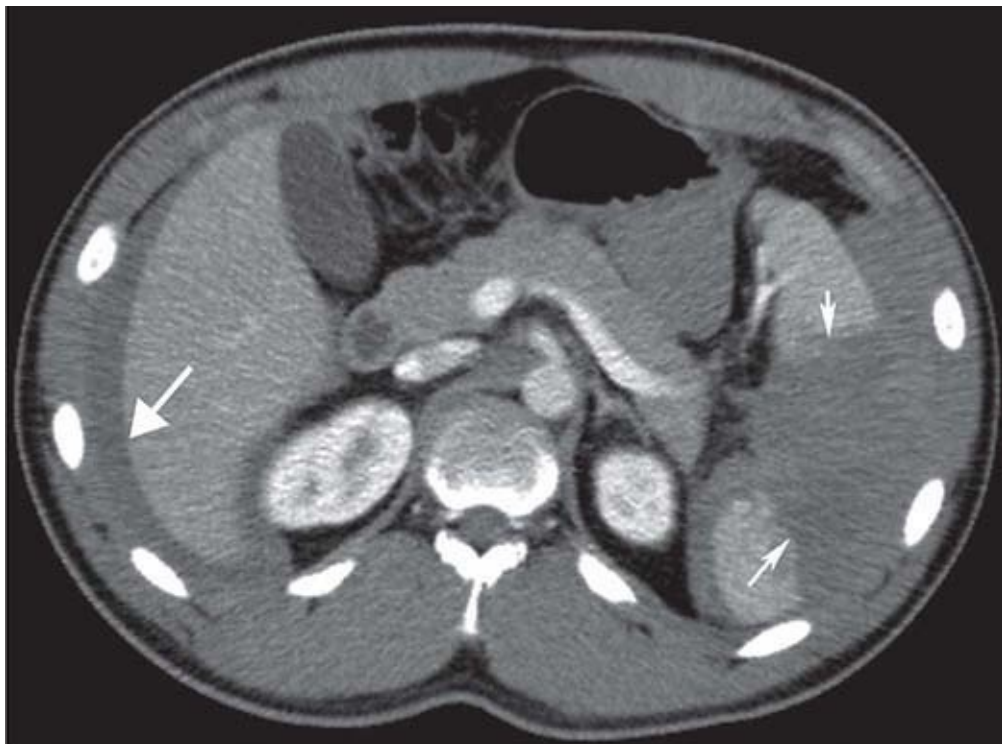


FIGURE 15.79 ■ Splenic Laceration. A severe laceration of the spleen is seen. Note the large separation of the splenic parenchyma with fluid density (blood) visible between the sections of the spleen (white arrows) and free fluid within the peritoneum (large white arrow). In young children, motor vehicle collisions and abuse account for the large majority of such injuries. (Photo contributor: Marguerite Caré, MD.)



FIGURE 15.80 ■ Splenic Laceration. This splenic laceration was identified in a child with abusive head trauma and retinal hemorrhages in the course of an investigation for unexplained anemia. (Photo contributor: Daniel M. Lindberg, MD.)

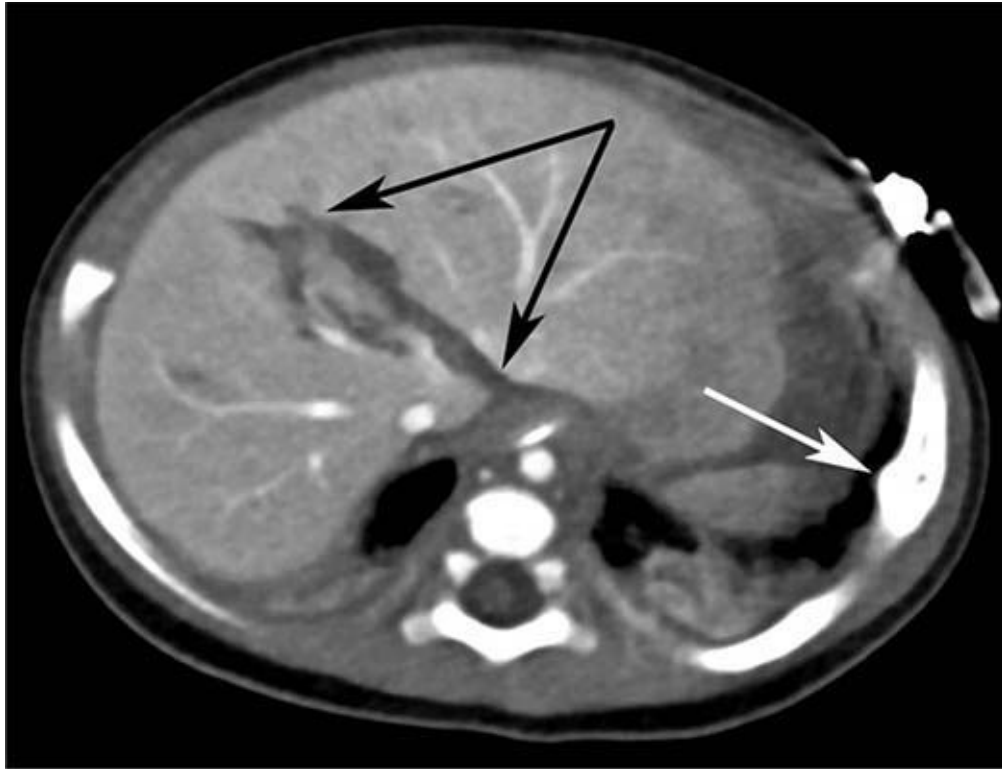


FIGURE 15.81 ■ Abusive Liver Laceration and Rib Fracture. This CT demonstrates a clinically occult hepatic laceration (black arrows) identified in a child with other abusive injuries. A left lateral rib fracture, better appreciated in bone windows (white arrow), is also apparent, a reminder to examine ribs closely in abdominal CTs obtained with concern for abuse. (Photo contributor: Angie L. Miller, MD.)

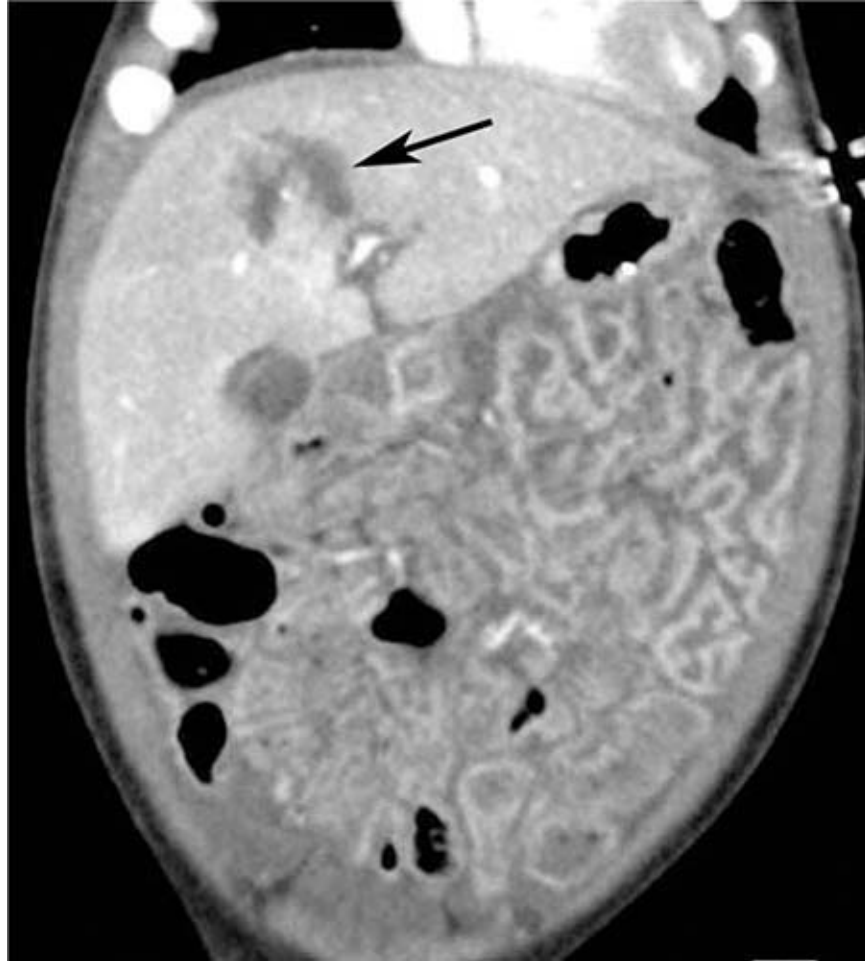


FIGURE 15.82 ■ Abusive Liver Laceration and Shock Bowel. This abusive liver laceration appears as a hypodense area in the central liver (arrow). Diffusely thickened and hyperenhancing bowel shows evidence of hypoperfusion, termed “shock bowel.” (Photo contributor: Angie L. Miller, MD.)

Pearls

1. Abdominal CT with IV contrast is the first choice for imaging in children with suspicion of abusive abdominal injuries. Neither ultrasound nor serial lab testing is sufficiently sensitive.
2. Hollow viscus injuries, vascular injuries, and pancreatic injuries have a high likelihood for decompensation and require early surgical consultation.
3. Amylase and lipase do not identify a significant number of abdominal injuries that are missed by physical examination and AST/ALT and should not be obtained routinely. If they are obtained and are significantly elevated, abdominal CT should be obtained.
4. Many children with significant abusive abdominal injury will have no bruising on physical examination.

Sexual Abuse

CHILD SEXUAL ABUSE EXAM AND GENITAL FINDINGS

Clinical Summary

The diagnosis and management of child sexual abuse differs from adult sexual assault. The majority of prepubertal victims of sexual abuse will have a normal genital exam without evidence of acute or healed trauma. Young children may present due to nonspecific findings such as behavior changes or genital rash. The history and context of concerns are often most important to make the diagnosis. Knowledge of child development and behavior informs the management and diagnosis. An emergency department (ED) physician may ask questions as necessary for the medical evaluation of the patient. As a rule, the history is obtained from the caregiver out of the presence of the child. Care should be taken to avoid leading questions, and both the question and the child's answer should be recorded verbatim when possible. Forensic interviews should be performed by professionals trained in interviewing children for possible sexual abuse.

A pubertal female can be examined in the supine position with feet in stirrups. A prepubertal female should be examined in the supine frog-leg position (hips and knees bent; soles of feet touching) and can be examined on the examination table or while sitting on a caregiver's lap. Gentle, even labial traction (pulling toward the examiner, not laterally) will allow visualization of the vulva and the hymen. Special attention should be given to the hymen and posterior fourchette, the most common sites of injury. Use the clock-face designation when documenting locations around the hymen. The prepubescent hymen is thin and has smooth edges and is extremely sensitive to touch. A speculum is never used in the examination of a prepubertal girl, except in rare examinations under anesthesia. In postpubertal females, the hymen is thickened and redundant; a cotton-tipped applicator can be used in the postpubertal female to unfold and examine the edges of the hymen.

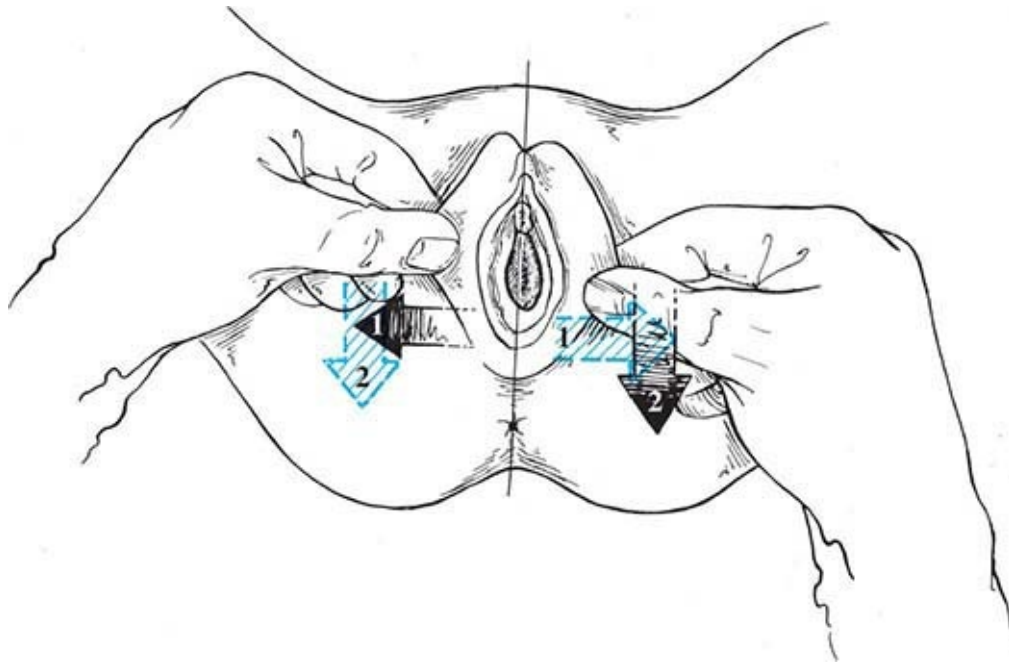


FIGURE 15.83 ■ Labial Traction Examination Technique. Position the child in a supine position with her knees out and soles together. Hymenal inspection in prepubertal girls is best accomplished when lateral (1) and posterior (2) traction to the labia is applied as shown here. (Adapted with permission from Giardino AP et al. *A Practical Guide to the Evaluation of Sexual Abuse in the Prepubertal Child*. New York, NY: Sage Publications; 1992.)

A male patient can be examined in the supine position, with care to inspect the penis and scrotum in entirety. The anus can be examined in the prone or lateral decubitus position.

Management and Disposition

When patients with a concern of sexual abuse present to the ED, social work should be notified. If sexual abuse is suspected, a report of alleged sexual abuse should be made to child protective services and law enforcement.

Children who are seen in the ED for acute sexual assault should receive a complete physical examination including genital and anal examination. Any injuries or concerning lesions should be described in detail with the aid of diagrams and, when available, photo documentation.

Evidence collection should be considered if the contact occurred within the last 72 hours (for all males and prepubertal females) and within 120 hours in pubertal females. The decision to perform evidence collection and the types of evidence collected are based on the contact described.

In most cases, an anxious patient will cooperate fully with the examination if

a parent or other support person remains for the examination. Reassurance and distraction techniques are helpful. Sedation is rarely needed and can be ineffective. An examination should never be forced upon an unwilling patient. When an examination is deemed medically necessary (eg, vaginal bleeding without a known source) and the patient is not cooperative, examination under anesthesia should be considered.

Sexual assault can coincide with intentional or covertly administered substance use. Do not begin the evidence collection or genital/anal examination until the patient is coherent and can consent or assent to the process and examination.



FIGURE 15.84 ■ Proper Labial Traction Allows Visualization of the Prepubertal Hymen. (A) Lateral labial traction only. The hymenal margins cannot be visualized. (B) Lateral, posterior, and caudal labial traction provides complete visualization of the hymenal rim. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

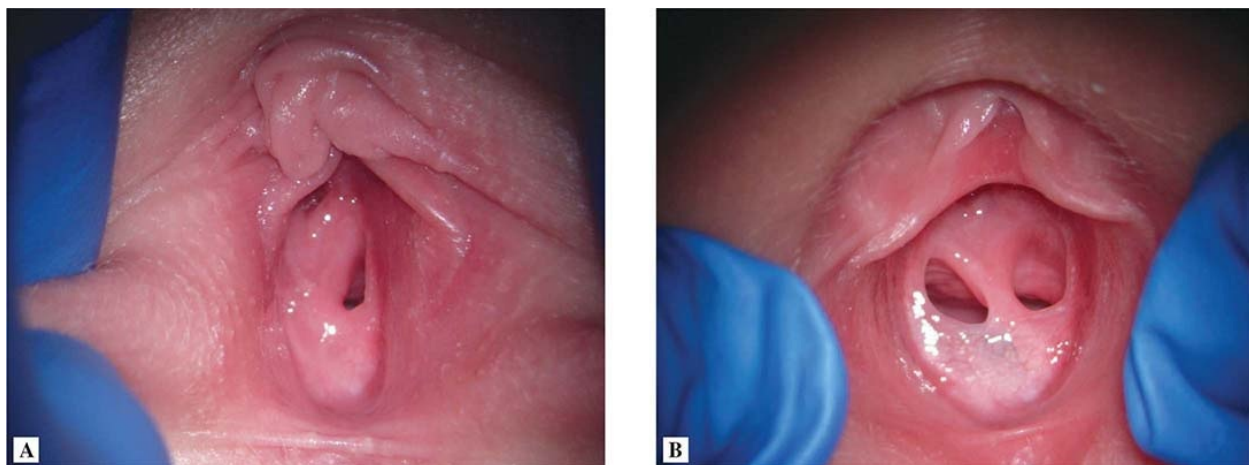


FIGURE 15.85 ■ Proper Labial Traction Allows Visualization of the Prepubertal Hymen. The hymen is partially visible in the first photo (A). When traction is applied, a hymenal septum is found, which creates the appearance of two vaginal openings (B). A septum is a normal hymenal variant. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.86 ■ Normal Estrogenized Newborn Hymen. Infants often have thickened annular hymens.

Maternal estrogens cause this effect. Do not confuse with traumatic swelling. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.87 ■ Normal Annular Hymen. The hymen is doughnut shaped without any defects. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

Pearls

1. A normal examination is the most likely finding, even in abused children, and does not exclude the diagnosis of sexual abuse. Examination findings (genital and anal) specific for sexual abuse are found in only a small percentage of abused patients.
2. The hymen is a ring of tissue that surrounds the vaginal opening. The shape and appearance of the prepubertal hymen is variable. Crescentic and annular configurations are most common. Hymens can also be sleeve-like, septate, imperforate (which eventually requires surgical opening), and cribriform (multiple small openings).
3. The prepubertal hymen is extremely sensitive and should not be touched; doing so will end the useful portion of the exam. If you need to manipulate the hymen, you can sometimes use the medial labia.
4. The inner edge of the prepubertal hymen is usually smooth and uninterrupted. Notches at the 3- and 9-o'clock positions are normal. Minor irregularities are

most likely insignificant.

5. In the United States, questions about HIV postexposure prophylaxis can be directed to a Centers for Disease Control and Prevention (CDC) hotline (888-HIV-4911).
6. Presumptive treatment for gonorrhea and chlamydia is not recommended for a prepubertal patient because of the very low incidence of infection in these patients and also because of the low risk of ascending infection (infection is a lower-tract disease in prepubertal females) and the need for confirmatory testing.
7. A history of sexual abuse is strongly associated with increased risk of suicidal thinking and suicide and self-harm attempts among older children and adolescents. Patients evaluated for sexual abuse should be carefully assessed for suicidal thoughts.
8. Before the patient is discharged from the ED, it is necessary to ensure a safe discharge plan.



FIGURE 15.88 ■ Normal Crescentic Hymen. The hymen is crescentic shaped without any hymen present from the 11- to 1-o'clock position. This is a normal hymen and the most common configuration in

prepubertal girls. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.89 ■ Normal Hymenal Mound—Crescentic Hymen. The hymen is crescent shaped, tissue is absent near the urethra between 11- and 1-o'clock position. A nonspecific mound of hymenal tissue is noted at 3-o'clock position. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.90 ■ Foley Catheter Technique to Better Visualize the Adolescent Hymen. In addition to using a cotton-tipped applicator (A), a Foley catheter can be inserted into the adolescent vagina, filled with a mixture of air and water, and gently retracted (B). Side-to-side displacement of the catheter exposes different hymenal sections for inspection (C). (Photo contributor: Cincinnati Children's Hospital Medical Center.)

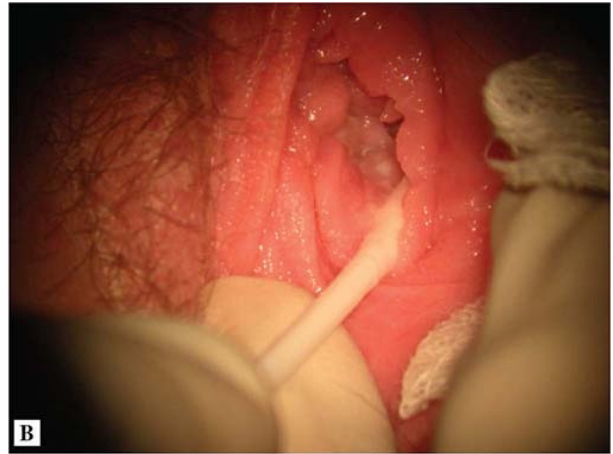


FIGURE 15.91 ■ Cotton Swab Technique. These images (A, B, C) show how a cotton-tipped applicator is used to ensure that all parts of the hymen are visualized during the examination of a pubertal patient. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.92 ■ Hymenal Septum. This picture shows a common hymenal variant—hymenal septum. A septum that extends into the vagina must be excluded either by examination (demonstrating that a cotton-tipped applicator can pass behind the septum) or with an ultra-sound. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.93 ■ Normal Anal Dilation. A relaxed external sphincter causes anal dilation in all children. Stool in the rectal vault contributes to dilation. This is not a finding of sexual abuse. Other conditions that can cause anal dilatation include chemical sedation, death, or a prolonged period of hypoxia. (Photo

INJURIES AND FINDINGS INDICATIVE OF GENITAL OR ANAL TRAUMA, ABUSE, OR INFECTION

Clinical Summary

Most physical examinations in cases of sexual abuse or assault will be normal. Findings specific for acute trauma, although not necessarily diagnostic of sexual abuse or assault, include bruising, lacerations, and abrasions. Most of these injuries will heal quickly, usually within a few days, and will heal completely without scarring. Nonacute examination findings caused by trauma can be difficult to diagnose and should be interpreted by a qualified child abuse expert.

Sexually transmitted infections diagnosed in a prepubertal child usually indicate sexual abuse. *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, *Trichomonas*, and syphilis are almost always transmitted by intimate sexual contact (intimate contact with infected secretions or lesions) unless acquired perinatally. HIV (unless acquired perinatally) and hepatitis are transmitted by intimate sexual contact or contact with infected blood. *Condylomata acuminata* (human papillomavirus or genital warts) and herpes simplex virus may be transmitted to the prepubertal child through sexual or nonsexual contact.

Management and Disposition

Victims of an acute sexual assault may have genital and/or anal bleeding. Patients who have active genital and/or anal bleeding need immediate attention and evaluation.

Also see “Child Sexual Abuse Exam and Genital Findings.”



FIGURE 15.94 ■ Hymenal Bruise. The hymen has bruising from 3-to 5-o'clock position after an acute sexual assault. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.95 ■ Posterior Fourchette Laceration. A laceration is present in the posterior fourchette, after sexual assault. This is a common location for injury after sexual assault/abuse. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.96 ■ Acute Genital Injury. This prepubertal girl has bruising to her left labia minora, and an acute, bleeding transection of her inferior hymen. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.97 ■ Scrotal Bruise. Bruising to the scrotum is present as well as an abrasion to the tip of the penis. Physical abuse as well as sexual abuse should be considered when genital injury is noted. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.98 ■ Penile Abrasion. Abrasion to the underside of the penis illustrates that it is important to examine all parts of the genitalia. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.99 ■ Acute Deep Perianal Lacerations and Bruising. An acute rectal laceration is visible at 8-o'clock position with bruising on the right side of the perianal area. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.100 ■ Vaginal Discharge—*Neisseria gonorrhoeae*. Vaginal discharge in a prepubertal child may be an indication of a sexually transmitted infection even when a history of abuse is denied. All children with vaginal discharge should be cultured for *N gonorrhoeae* and *Chlamydia*. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.101 ■ Condyloma Acuminata. Several small warts are visible in the perineum in this young child. (Photo contributor: Hope K. Haefner, MD.)

Pearls

1. It is not necessary to measure the vaginal opening of prepubertal girls. The size of the opening is dependent on examination technique and degree of patient relaxation. There is no consensus of normal size opening among experts.
2. Eyelash lice are pubic lice, and sexual abuse must be considered.
3. Rectal abuse often causes no visible trauma. Superficial anal fissures can be common findings in children; consider abusive trauma, however, if deep fissures are present.



FIGURE 15.102 ■ Perianal Condyloma Acuminata. Several peri-anal warts are visible. Warts can be transmitted sexually and nonsexu-ally. Workup should include an evaluation (examination, forensic interview) for sexual abuse and testing for other sexually transmitted infections. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.103 ■ Perianal Condyloma Acuminata. Extensive perianal warts are visible. The diagnosis of genital or anal warts does not automatically mean that they were sexually transmitted (see above). (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.104 ■ Perianal Condyloma Lata (Secondary Syphilis). Perianal condyloma lata are visible around the rectum. This should not be confused with condyloma acuminata. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

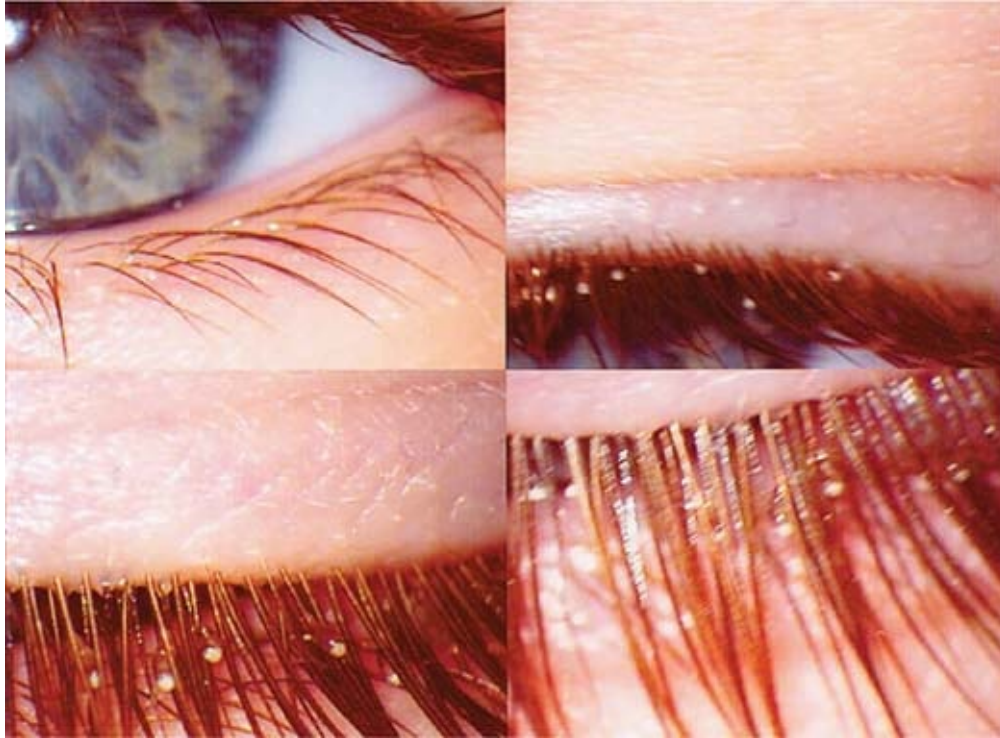


FIGURE 15.105 ■ Nits. Nits (the larval form of the louse) from *Pthirus pubis* are seen firmly adherent to the eyelashes in this child. Sexual abuse should be strongly considered; these are not from head hair. (Photo contributor: Robert A. Shapiro, MD.)

MIMICS OF ABUSE: ACCIDENTAL TRAUMA

Clinical Summary

Genital injury can result from a fall (straddle) onto an object. A straddle injury usually causes damage to the external genitalia with unilateral bruising and swelling of labia and periurethral tissues. Occasional penetrating trauma to the hymen with internal injury can be seen. The history and the type of object involved are important historical elements. Obtain a detailed history from the child (if developmentally appropriate) separately from the accompanying parent/adult. Factors that increase concern of possible sexual abuse include no history of injury, injury in a nonambulatory child, vaginal or hymenal injury without history of penetrating trauma, history inconsistent with physical findings, and/or additional nongenital trauma.

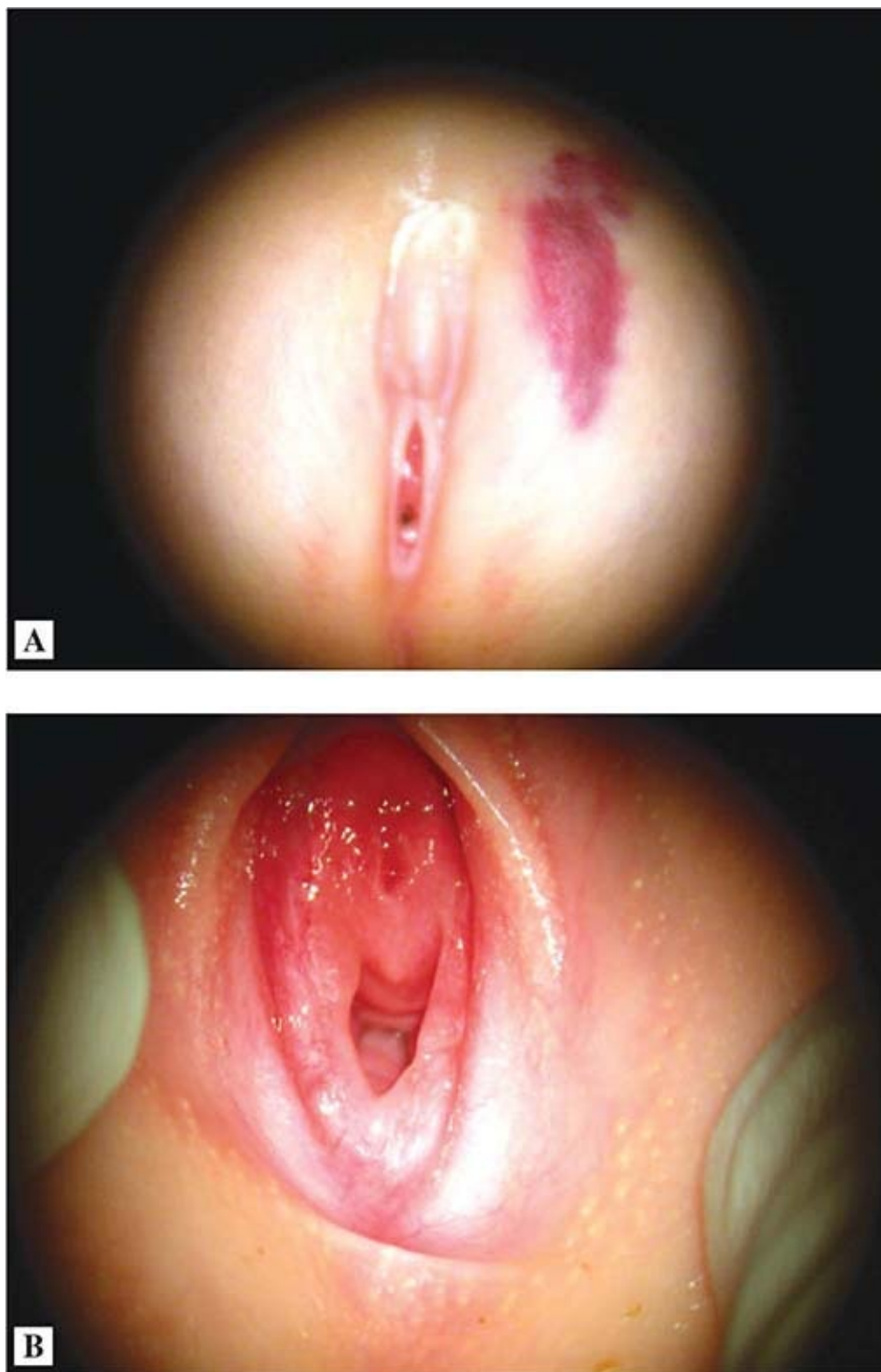


FIGURE 15.106 ■ Straddle Injury. This young child fell on to a pool edge sustaining a hematoma to the left labia majora (A, B). The remainder of her examination, including her hymen, was normal. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.107 ■ Straddle Injury. Bruising to the left labia majora and clitoral hood following a fall onto a bicycle bar. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.108 ■ Straddle Injury. Ecchymosis, swelling, and contusion of the perineum in a 3-year-old female child who tripped and fell on a large plastic toy. (Photo contributor: James Mensching, MD.)



FIGURE 15.109 ■ Toilet Seat Injuries. This toddler presented with a straightforward history of the toilet bowel falling onto his penis while voiding. He was able to void without difficulty. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 15.110 ■ Toilet Seat Injuries. The toilet seat fell onto this toddler's penis and caused bruising to the penis. Discharge is appropriate if the child can void without difficulty. (Photo contributor: Charles J. Schubert, MD.)

Acute bruising to the glans and corona of the penis can occur if the toilet seat falls onto the penis during voiding, trapping the penis between the seat and toilet bowl. This injury is not uncommon in boys of about 3 years of age whose height

and development set the stage for this injury.

Management and Disposition

Check for urethral injury in children with straddle injuries. Treatment is usually symptomatic. Sitz baths and antibacterial ointment or barrier creams promote healing and minimize discomfort. If the child has difficulty voiding, encourage voiding in a bath with a few inches of warm water to reduce dysuria. If severe injuries occur, surgical repair may be necessary with consultation with pediatric gynecology or surgery.

No specific treatment is needed for toilet seat injuries unless the child is unable to void. Consult urology if the child cannot void. Consider abuse in any child with a genital injury.



FIGURE 15.111 ■ Urethral Prolapse. A round reddish-purple donut-shaped mass is seen in this child's introitus. Careful examination reveals that the mass originates from the urethra. The hymen is normal. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

Pearls

1. Straddle injuries usually present with a clear mechanism of injury and a physical examination that supports the history.
2. Straddle injuries do not typically involve the hymen or internal vaginal mucosa.
3. Toilet seat injuries are most common in boys who are toilet training.

MIMICS OF ABUSE: MEDICAL CONDITIONS

Clinical Summary

Urethral prolapse is a condition that only occurs in females, usually in school-aged children. Patients will present with painless bleeding and, on exam, will have what appears as swelling, but is actually the prolapsed portion of the urethra. Urethral prolapse is commonly mistaken for vaginal injury. The etiology is unknown, but estrogen deficiency is thought to be a contributing factor. Other risk factors include increased intra-abdominal pressure that results from coughing or constipation and anatomic defects. Urethral prolapse is more common in African American girls.

Lichen sclerosus et atrophicus (LSA) is found often in older women but occasionally in prepubertal girls. Findings include thin hypopigmented (as white or yellow plaques) and easily friable skin in the genital and anal areas that can bleed even without trauma; itching and pain can also occur. The hemorrhagic form of LSA includes subepithelial hemorrhagic lesions to the affected skin, which can be mistaken for trauma.



FIGURE 15.112 ■ Urethral Prolapse. A round donut-shaped mass consistent with urethral prolapse is seen

in this school-aged child. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.113 ■ Lichen Sclerosus. This image shows both the hypopigmentation (white plaque area) and the multiple hemorrhagic areas characteristic of lichen sclerosus. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.114 ■ Lichen Sclerosus. This close-up image shows the hypopigmentation (white plaque area) and the differentiation between it and the surrounding normal skin. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.115 ■ Lichen Sclerosus. Hemorrhagic lichen sclerosus atrophicus. The perineum surrounding the vagina and anus is hypopigmented in an hourglass configuration. The hemorrhagic lesions are not traumatic in etiology. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

Management and Disposition

Medical treatment of urethral prolapse includes sitz bath and topical estrogen cream. A referral to urology should also be made, as well as reviewing with the family indications for emergent return including increased pain or bleeding, which are signs of strangulation. Surgical repair may be needed when conservative treatment fails or when the pro-lapse is necrotic.

Treatment of LSA consists of high-potency corticosteroids and referral to dermatology or pediatric gynecology.

Pearls

1. Urethral prolapse often presents with painless bleeding in school-aged girls.



FIGURE 15.116 ■ Labial Adhesions. Adhesions of the labia minora occur in young girls and may persist until puberty. A thin translucent line is seen where the labia meet. Involvement is often limited to the posterior portion of the labia, but more extensive lesions, as in this child, can obscure the hymen. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.117 ■ Labial Adhesions. Labial adhesions obscure the hymen in this prepubertal girl. (Photo contributor: Larry B. Mellick, MD.)

2. The hemorrhagic form of LSA can be mistaken for trauma; surrounding hypopigmented skin is the clue to the diagnosis.
3. Estrogen cream (Premarin) can be prescribed and applied gently over the adhesions twice daily for 2 to 4 weeks. Recurrence is common.
4. Labial adhesions may be mistaken for scars, and dehiscence from labial separation during the medical examination can cause minor bleeding, but should not be considered a sign of abuse.



FIGURE 15.118 ■ Perianal Streptococcal Infection. Intense erythema around the anus consistent with perianal streptococcal infection. Signs and symptoms include erythematous perianal, vulvovaginal, or penile rash with itching, pain, fissures, bleeding, and/or discharge. Systematic symptoms (fever, pharyngitis) are absent but can precede the onset of the rash. (Photo contributor: Raymond C. Baker, MD.)



FIGURE 15.119 ■ Perianal Streptococcal Infection. Intense erythema around the anus consistent with perianal streptococcal infection. (Photo contributor: Kevin J. Knoop, MD, MS.)

5. Diagnose perianal strep by swabbing the affected areas and culturing for group A streptococci. Treatment is with oral penicillin or amoxicillin for 14 to 21 days. Erythromycin is used in patients who are penicillin allergic.



FIGURE 15.120 ■ Venous Pooling. Venous pooling refers to the purple discoloration of the perianal tissues caused by collection of venous blood in the external hemorrhoidal plexus. Venous blood pools in this plexus when a patient is immobile, causing the veins to become distended and visible. It can often be eliminated by getting the patient up and mobilized and reexamining in the prone or lateral decubitus position. (Photo contributor: Kathi L. Makoroff, MD.)

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Chapter 16

ENVIRONMENTAL CONDITIONS

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Multiple Lightning Strikes. (Photo contributor: Lawrence B. Stack, MD.)

HIGH-ALTITUDE PULMONARY EDEMA

Clinical Summary

High-altitude pulmonary edema (HAPE) is a form of noncardiogenic pulmonary edema, generally beginning within the first 2 to 4 days after ascent above 2500 m (8200 ft). Early symptoms are fatigue, weakness, dyspnea on exertion, and decreased exercise performance. Symptoms of AMS, such as headache, anorexia, and lassitude, may also be present, but HAPE may develop without AMS. The first symptoms usually include persistent dry cough and dyspnea followed by tachycardia, tachypnea, and cyanosis at rest. Patients suffering from HAPE often experience sudden onset of symptoms upon awakening after the 2nd night at altitude. Eventually the victim develops dyspnea at rest and orthopnea with audible crackles in the chest. Pink frothy sputum is a grave sign. Patients may experience concurrent mental status changes and ataxia due to hypoxemia or associated high-altitude cerebral edema.

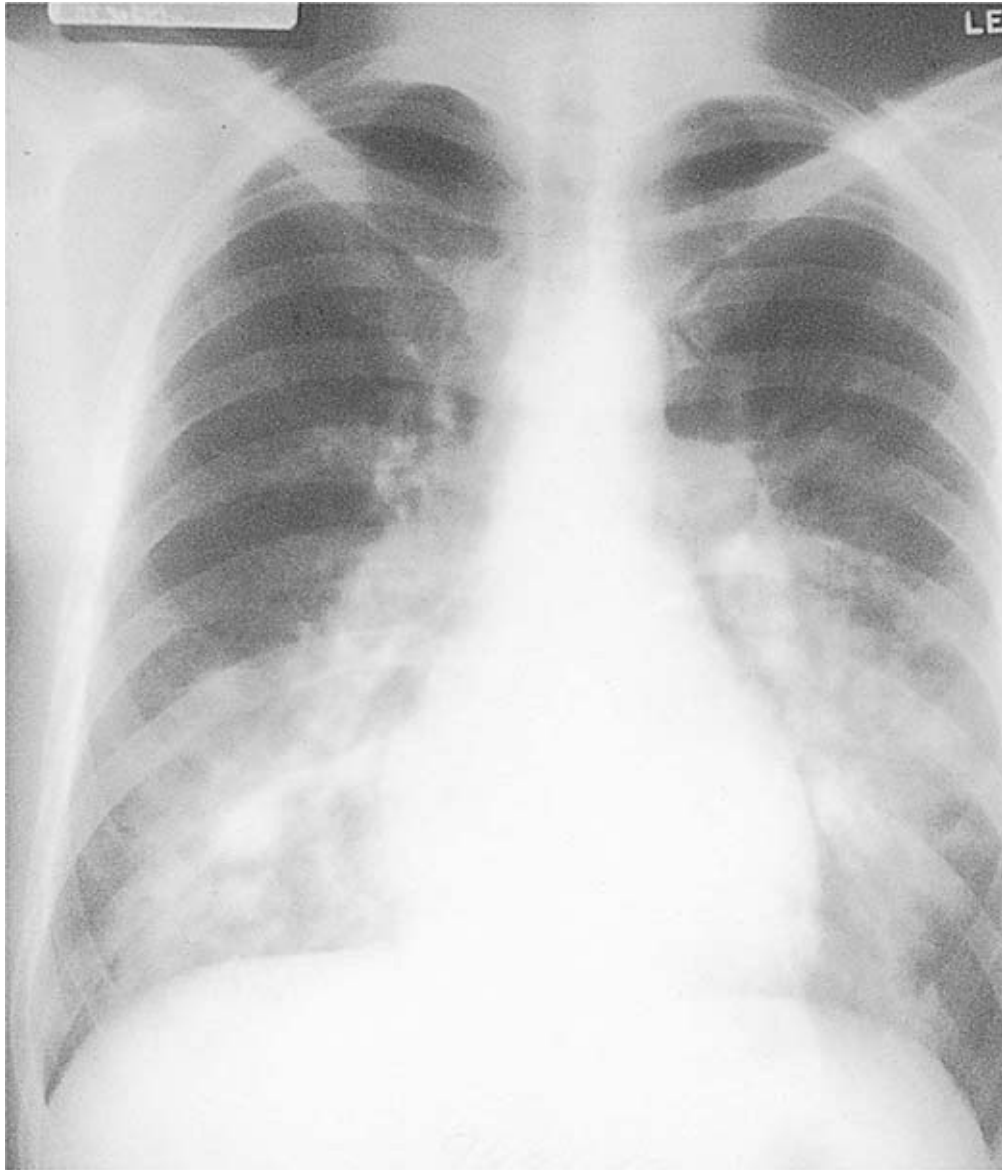


FIGURE 16.1 ■ High-Altitude Pulmonary Edema. Chest x-ray in patient with HAPE. Note normal heart size with bilateral “patchy” pulmonary infiltrates. (Photo contributor: Peter Hackett, MD.)



FIGURE 16.2 ■ “Gamow Bag.” Portable hyperbaric chamber (Gamow bag). A HAPE patient is being treated at 4300 m at Pheriche, Nepal. Due to orthopnea, the patient was unable to tolerate lying flat, so the bag was propped up (head elevated) immediately after inflation. (Photo contributor: Ken Zafren, MD.)

Management and Disposition

Mild cases (oxygen saturation in the 90s on low-flow oxygen) at moderate altitudes (below 3500 m [11,500 ft]) may be treated at altitude with bed rest and oxygen. If supplemental oxygen and a reliable person are available, the patient may be discharged with oxygen therapy and bed rest at home or in lodgings. Patients with more severe cases should descend immediately with as little exertion as possible. These patients may require admission to a hospital at a lower altitude and, in extreme cases, intubation and mechanical ventilation. Nifedipine, which lowers pulmonary artery pressure, is of benefit but is not a substitute for descent. Some experts use phosphodiesterase-5 inhibitors such as sildenafil or tadalafil instead of nifedipine. Hyperbaric therapy, especially with a portable hyperbaric chamber (Gamow bag), has an efficacy equal to that of supplemental oxygen and is mainly helpful in prehospital settings where oxygen availability is limited.

Pearls

1. Crackles may be unilateral or bilateral but usually start in the right middle lobe and are heard first in the right axilla.

2. HAPE limited to the left lung in association with a small right hemithorax without pulmonary markings on chest x-ray is pathognomonic for unilateral absent pulmonary artery syndrome. These patients develop HAPE at relatively low altitudes, sometimes below 2500 m.
3. Patients treated for HAPE may resume normal activities once they are asymptomatic and may ascend further during the same trip.

HIGH-ALTITUDE RETINAL HEMORRHAGE

Clinical Summary

Retinal hemorrhages are common above 5200 m (17,000 ft) and are not usually associated with acute mountain sickness (AMS). High-altitude retinal hemorrhages (HARHs) are rarely symptomatic, but if found over the macula, these hemorrhages may cause temporary blindness. The diagnosis can be established by ophthalmoscopy. Without visualization of the lesion, the differential diagnosis of unilaterally decreased vision or blindness at high altitude includes migraine equivalent, cerebrovascular accident, and dry eye (often unilateral, due to strong winds), as well as all conditions found at sea level.

Management and Disposition

HARHs generally resolve spontaneously after descent to lower altitude. No treatment is necessary for asymptomatic HARH. Patients with HARH associated with a decrease in vision should be referred to an ophthalmologist for follow-up.

Pearls

1. Patients with blurred vision and unilateral mydriasis at the high altitude should be asked about use of medications, including transdermal scopolamine patches.
2. As with almost all altitude-related problems, descent is the primary treatment. This is not emergent unless associated with severe altitude illness or progressive visual loss.
3. Although most symptomatic HARHs resolve completely in 2 to 8 weeks,

cases of permanent paracentral scotomata have been reported.

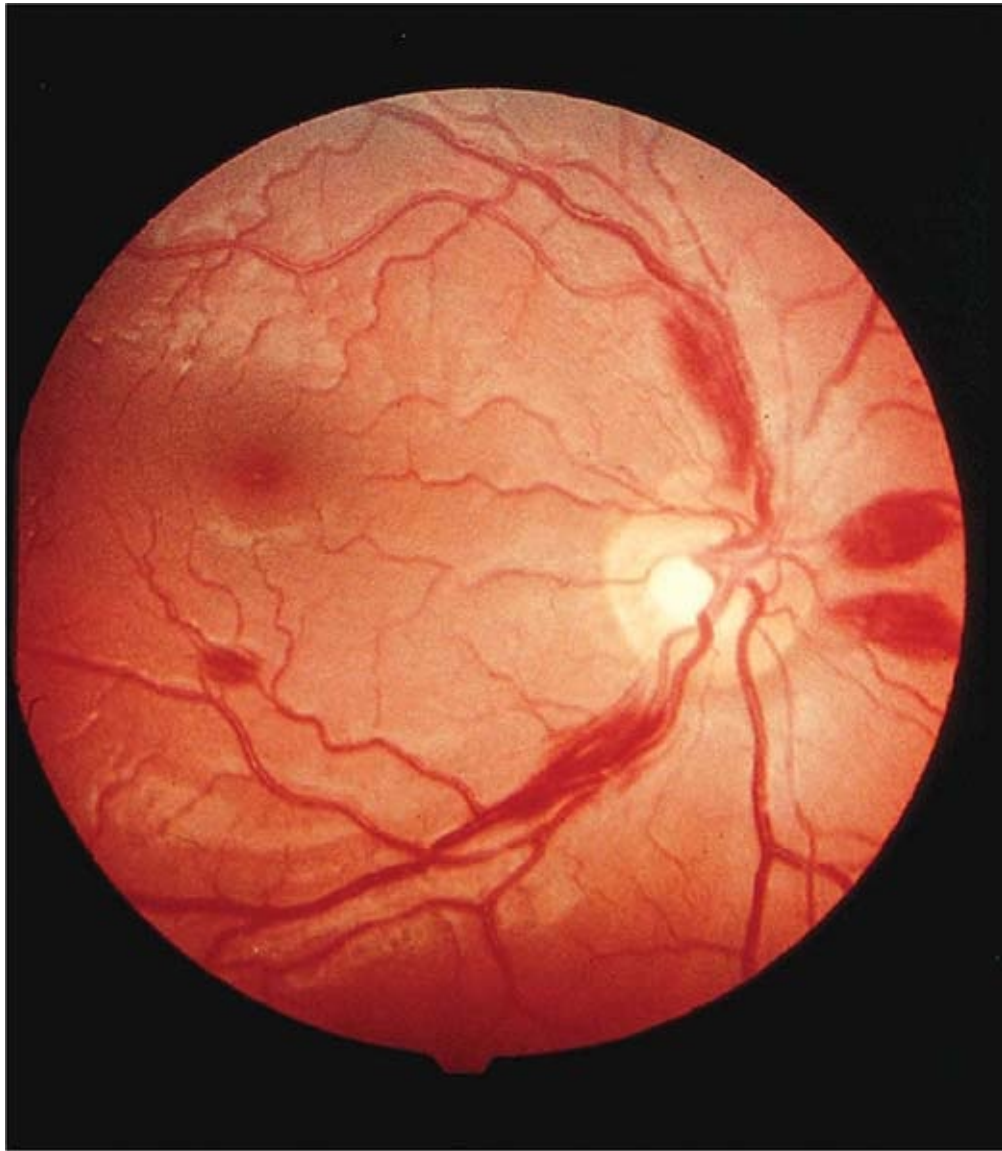


FIGURE 16.3 ■ High-Altitude Retinal Hemorrhage. Fundoscopic appearance of high-altitude retinal hemorrhage. (Photo contributor: Peter Hackett, MD.)

HIGH-ALTITUDE CEREBRAL EDEMA

Clinical Summary

Acute mountain sickness (AMS) is a symptom complex that usually begins 12 to 24 hours after ascent to high altitude and consists of headache and one or more

other symptoms, including gastrointestinal symptoms, fatigue and/or weakness, dizziness and/or lightheadedness, and difficulty sleeping. High-altitude cerebral edema (HACE) is a severe form of AMS, clinically defined by the presence of acute truncal ataxia, altered mental status, or both. Usually this occurs as a progression from AMS to HACE, but HACE may occur without antecedent AMS. If not effectively treated, HACE may progress to coma or death. Focal neurologic findings other than truncal ataxia are rare and should suggest another diagnosis, such as acute stroke or venous sinus thrombosis.

Management and Disposition

Treatment of HACE consists of immediate descent or evacuation, oxygen, and high-dose dexamethasone. Simulated descent using a portable hyperbaric bag (Gamow bag) may be more effective than oxygen alone and may be used in place of oxygen in field settings. Actual descent may be complicated by the inability of the patient to walk unassisted or at all. Patients with HACE may not ascend during the same trip and probably should not reascend to altitude for several months.



FIGURE 16.4 ■ HACE-Related Ataxia. The patient (middle) developed HACE and severe truncal ataxia at 5600 m (18,400 ft) in the Himalayas. He required full ambulatory assistance. Descent was started immediately. With descent, the patient's breathing improved, but he remained too ataxic to walk. (Photo contributor: Ken Zafren, MD.)

Pearls

1. The first sign of HACE is usually truncal ataxia. This may be tested by assessing tandem gait (heel-to-toe walking).
2. HACE may occur without symptoms of AMS.
3. HACE is often associated with some degree of HAPE.

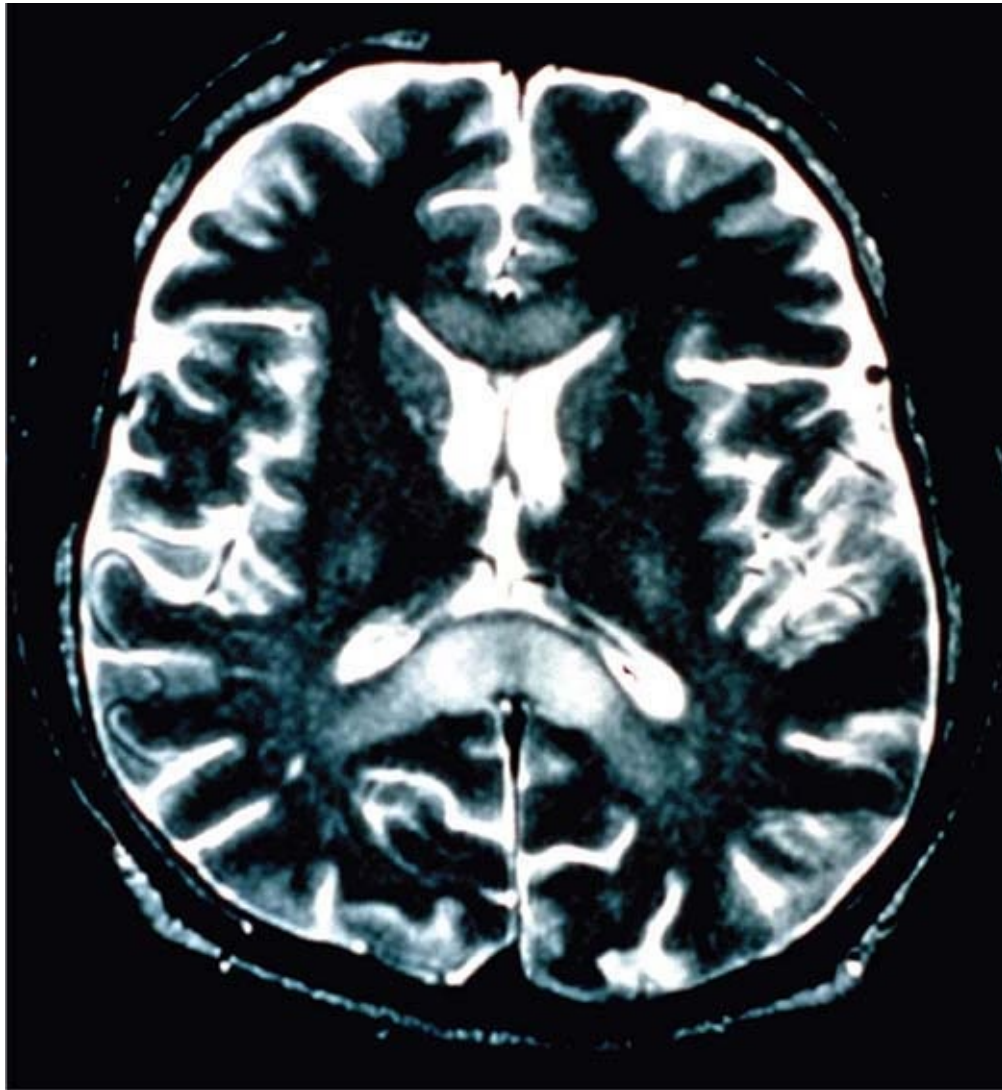


FIGURE 16.5 ■ MRI Brain—HACE. MRI of a patient with HACE. Note the increased white matter signal, especially in the splenium of the corpus callosum, in this T2-weighted image. (Photo contributor: Ken Zafren, MD.)

Clinical Summary

Accidental hypothermia is an unintentional decline in core temperature below 35°C (95°F). Presentation may be obvious or subtle, especially in urban settings. Symptoms vary from vague complaints to altered levels of consciousness. Physical findings include progressive abnormalities of every organ system. Following initial tachycardia, there is progressive bradycardia (50% decrease in heart rate at 28°C [82.4°F]) with decline in blood pressure and cardiac output. Electrocardiogram (ECG) intervals are prolonged, beginning with the PR interval followed by the QRS interval and finally the QT interval. A J wave (Osborn wave; hypothermic “hump”) may be seen, but is neither pathognomonic nor prognostic. The J wave is present at the junction of the QRS complex and the ST segment. J waves may also be associated with central nervous system lesions, focal cardiac ischemia, young age, and sepsis. In mildly hypothermic patients, an invisible increase in preshivering muscle tone may obscure P waves.

Management and Disposition

Core temperature measurement is best made with an esophageal probe inserted into the lower third of the esophagus. Rectal temperature is less accurate and requires the use of a special low-reading thermometer. Gentle handling and appropriate warming methods are the mainstays of emergency department (ED) treatment. Cardiovascular instability often complicates rewarming; Advanced Cardiac Life Support (ACLS) guidelines for hypothermia provide guidance. If not obvious, a precipitating cause such as hypothyroidism, hypoglycemia, or sepsis should be sought, as should associated pathology. Most patients require admission for observation or to treat associated injuries or comorbidities.

Pearls

1. The most common problem with the misdiagnosis of hypothermia in the ED stems from incomplete vital sign data.

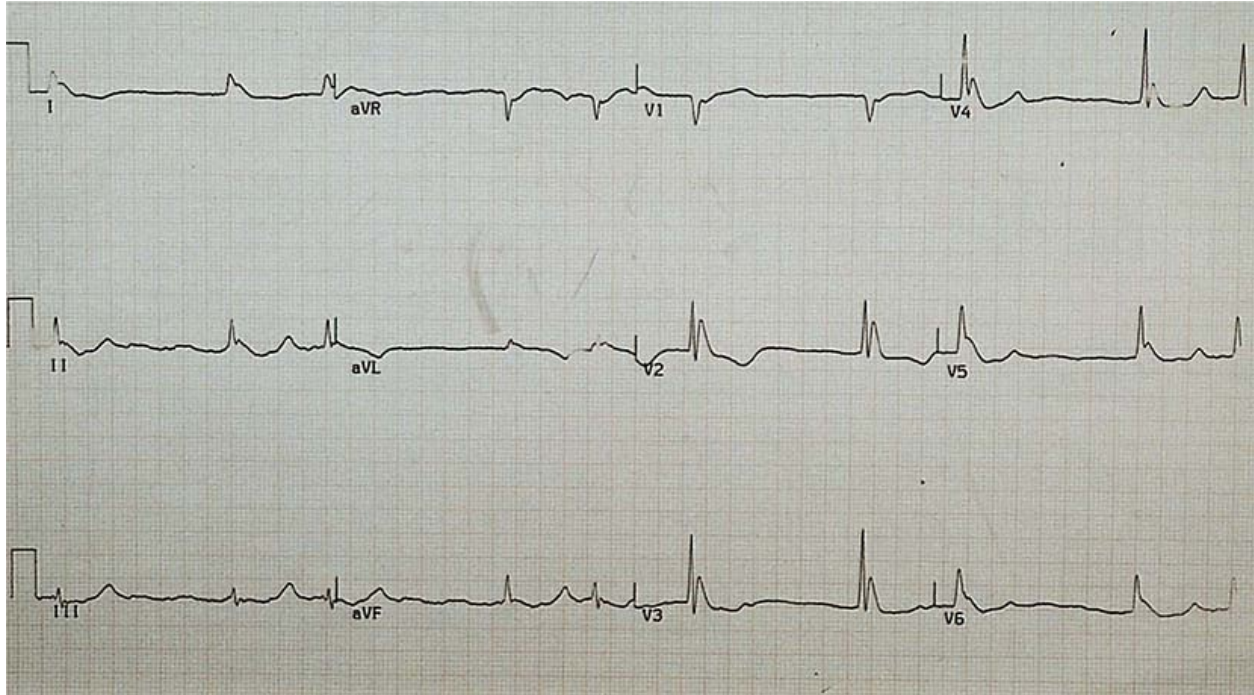


FIGURE 16.6 ■ J Waves. This patient's core temperature was 25.5°C. J waves occur below 32°C, especially in leads II and V₆. Below 25°C, they are larger and most common in precordial leads (especially V₃ and V₄). They are usually upright in aVL, aVF, and left precordial leads (see also [Fig. 23.45A](#), Hypothermia with Osborne Waves ["J" Waves] Present). (Photo contributor: Alan B. Storrow, MD.)

2. Accurate core temperatures, preferably by esophageal probe, and continuous cardiac monitoring are crucial to appropriate management.
3. Atrial arrhythmias are generally benign and should not be treated. They generally resolve with rewarming.
4. Most cardiovascular drugs are inactive during hypothermia and should be given only after the body temperature is above 35°C (95°F).

FROSTBITE

Clinical Summary

Frostbite is tissue freezing resulting from heat loss sufficient to cause ice crystal formation in superficial or deep tissue. Frostbite usually affects the extremities, nose, or ears (and the scrotum and penis in joggers). A sensation of numbness with accompanying sensory loss is the most common initial complaint. Often, by the time the patient arrives in the ED, the frozen tissue has thawed. The initial

appearance of the overlying skin may be deceptively benign. Frozen tissue may appear mottled blue, violaceous, yellowish-white, or waxy. Following rapid rewarming, there is early hyperemia even in severe cases.

Favorable signs include return of normal sensation, color, and warmth. Edema should appear within 3 hours of thawing; lack of edema is an unfavorable sign. Vesicles and bullae appear in 1 to 24 hours. Early formation of large clear blebs that extend to the tips of affected digits is a good indicator. Small dark blebs that do not extend to the tips indicate damage to subdermal plexi and are a poor prognostic sign. When seen early or soon after rewarming occurs, frostbite may be indistinguishable from nonfreezing cold injury such as immersion foot. Mixed injuries are common. Tissue loss is rare in uncomplicated non-freezing cold injury.

Management and Disposition

If other injuries are ruled out by history and physical examination, rewarm frostbitten areas in warm water bath (37°C-39°C [98.6°F-102.2°F]). If associated with severe hypothermia, active core rewarming should precede frostbite rewarming. If swelling occurs, surgical consultation is advisable to determine the need for fasciotomy. Admit all patients with associated hypothermia or in whom swelling occurs. Patients with superficial frostbite (minimal skin changes and erythema) may be treated by home care with nursing instructions. Patients with deep superficial frostbite (clear, fluid-filled blebs, swelling, pain) may be treated by home care in a reliable patient. The presence of deep frostbite (proximal hemorrhagic blebs, no swelling, no pulses) mandates hospital admission.



FIGURE 16.7 ■ Thawed Frostbite. Appearance of frostbite soon after rewarming. Deep frostbite was caused by wearing mountaineering boots that were too tight in extreme cold at high altitude. Note the deceptively benign appearance of this devastating injury, which ultimately resulted in bilateral below-the-knee amputations. (Photo contributor: Ken Zafren, MD.)



FIGURE 16.8 ■ Deep Frostbite. Deep frostbite at Everest Base Camp in Nepal, at 5360 m (17,585 ft), 3 days after exposure at 6400 m (21,000 ft). Dusky appearance and lack of distal blistering are poor prognostic signs. The great toe eventually required partial amputation. (Photo contributor: Chris Imray, MD.)



FIGURE 16.9 ■ Frostbite Blebs. Intact proximal blebs, both clear and hemorrhagic, indicate deep frostbite and a poor prognosis. (Photo contributor: Scott W. Zackowski, MD.)



FIGURE 16.10 ■ Frostbite-Ruptured Blebs. Frostbite injury to the hand with ruptured blebs. (Photo contributor: R. Jason Thurman, MD.)

Pearls

1. Early transfer of the patient to a center experienced in the care of frostbite injuries (even if far away) should be considered. On the other hand, transfer of the patient to a major medical center that does not generally manage frostbite is seldom in the patient's best interest.
2. Treatment of clear versus hemorrhagic blisters is controversial. One approach is to debride clear blisters and use topical aloe vera, while leaving hemorrhagic blisters intact.



FIGURE 16.11 ■ Late Frostbite. Late appearance of frostbite with demarcation starting to occur. Early surgery should be avoided in favor of autoamputation unless infection supervenes. (Photo contributor: James O'Malley, MD.)



FIGURE 16.12 ■ Late Frostbite. Late appearance of deep frostbite with clear demarcation. (Photo contributor: Wael Alrifai, MD.)

PERNIO

Clinical Summary

Pernio, also known as perniosis or chilblains, is the result of nonfreezing cold exposure in susceptible individuals. Pernio appears within 24 hours of cold exposure, most frequently on the face, ears, hands, feet, and pretibial areas. A large range of lesions may be seen, with localized edema, erythema, cyanosis, plaques, and blue nodules occasionally progressing to more severe lesions including vesicles, bullae, and ulcerations. The lesions persist for up to 2 weeks and may become chronic. They are typically very pruritic and associated with burning paresthesias. Following rewarming, pernio often takes the form of blue

nodules, which are quite tender. In the setting of recent cold exposure, pernio might be confused with the more severe syndrome of trench foot. If the history of cold exposure is not elicited, the differential diagnosis is potentially very broad.



FIGURE 16.13 ■ Pernio. Pernio or chilblains with localized erythema, cyanosis, and nodules. (Photo contributor: Ken Zafren, MD.)



FIGURE 16.14 ■ Pernio. Pernio of the hand with localized erythema and painful nodules. (Photo contributor: Alison Jarman, MD.)

Management and Disposition

Management is supportive. The skin should be warmed, washed, and dried. Affected extremities can be dressed in soft, dry, sterile dressings and elevated. Nifedipine (20-60 mg daily) may be helpful in chronic cases.

Pearls

1. Healing may be followed by hyperpigmentation.
2. Recurrences are possible following milder cold exposure.

3. Chilblains may be more frequent in young women, especially in association with Raynaud phenomenon, and may also be associated with underlying dermatologic or vascular disease.

IMMERSION INJURY (TRENCH FOOT)

Clinical Summary



FIGURE 16.15 ■ Immersion Injury. Immersion injury of the hands (an unusual location) several hours after rewarming. The patient spent 18 hours bailing out a boat in waters just above freezing in Alaska. (Photo contributor: Ken Zafren, MD.)

Immersion injury is a peripheral nonfreezing cold injury resulting from exposure to water, usually at temperatures just above freezing. However, the condition can occur during prolonged exposure to any cold wet environment. Dependency and immobility predispose to immersion injury. The degree of injury depends on exposure time and temperature. The first symptoms usually appear within hours. Tissue loss may occur after many days of exposure, but is more likely due to tissue necrosis or compartment syndrome due to swelling inside boots than to immersion injury. Prior to rewarming, the distal extremities are numb and swollen. The skin is first red, then changes to pale, mottled, or black. Cramping of the calves may occur. Immersion injury is distinct from tropical immersion

foot or warm-water immersion foot as seen in the Vietnam War. Tropical immersion foot was typically seen after 3 to 7 days of exposure to water at 22°C to 32°C. Warm-water immersion foot was seen after 1 to 3 days at 15°C to 32°C. These syndromes were characterized by burning in the feet, pain on walking, pitting edema, and erythema, with wrinkling and hyperhydration of the skin. They resolved completely after rest and removal from the wet environment.



FIGURE 16.16 ■ Immersion Foot. Early appearance of immersion foot in a mentally ill homeless patient. (Photo contributor: Ken Zafren, MD.)

Management and Disposition

Hypovolemia, hypothermia, and associated injuries are the rule and should be treated first. Extremities with immersion injuries should be allowed to rewarm gradually at room temperature with bed rest, elevation, and air drying unless frostbite that has not yet thawed is present. Extremities that are frozen require rapid rewarming in warm water. Swelling of immersion injuries may produce compartment syndrome and require fasciotomy. Most patients require admission to the hospital.



FIGURE 16.17 ■ Severe Trench Foot. This unfortunate homeless patient suffered prolonged exposure to a cold wet environment with resulting severe trench foot. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. Doppler ultrasound may be useful to identify peripheral pulses, as pulses are often difficult to palpate in affected extremities.
2. Mixed injuries (frostbite and immersion) are possible.

ULTRAVIOLET RADIATION EXPOSURE

Clinical Summary

Ultraviolet (UV) radiation causes both acute and chronic skin changes. Sunburn is a partial-thickness burn, which may become a full-thickness injury if infected. “Sun poisoning” is a severe systemic reaction to UV radiation. Patients may complain of nausea, vomiting, headache, fever, chills, and prostration. Excessive UV radiation may cause injury to the cornea and conjunctiva, termed UV keratitis (photokeratitis, snow blindness). This painful condition may occur in skiers, welders, or tanning salon patrons who do not wear proper eye protection.

There are several types of photosensitivity reactions (photodermatoses). Phototoxic reactions are abnormal responses to UV radiation caused by substances that are ingested (eg, prescription or over-the-counter medications) or applied to the skin. There is a direct relation between the amount of UV exposure and severity. Photoallergic reactions are clinically similar to contact dermatitis and, like phototoxic reactions, may be precipitated by ingested or applied drugs. Unlike phototoxic reactions, photoallergies may be precipitated by a small amount of light. Phytophotodermatitis is precipitated by skin contact with certain plants followed by exposure to UV radiation.



FIGURE 16.18 ■ Sunburn. Sunburn is characterized by erythema, edema, warmth, tenderness, and blisters. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 16.19 ■ Healing UV Exposure. This patient suffered significant UV exposure with resultant sunburn in the Himalayas. Darkly pigmented skin provides limited protection in high-altitude subtropical areas with very high amounts of UV exposure. Note the sparing of the periorbital areas secondary to the wearing of appropriate eye protection. (Photo contributor: Luanne Freer, MD.)



FIGURE 16.20 ■ Tanning Bed Burn. This patient suffered a severe diffuse partial-thickness burn from prolonged UV exposure in a tanning bed. Note the sharp demarcation on the buttocks at the point where the patient was partially protected by his pants. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.21 ■ Phytophotodermatitis. This reaction may require aggressive systemic steroid therapy. The case illustrated is a mild one caused by exposure to limes and UVA. A clue to the diagnosis is the patchy distribution with linear edges. More severe reactions resemble rhus dermatitis. (Photo contributor: Lee Kaplan, MD.)



FIGURE 16.22 ■ Phytophotodermatitis—Cow Parsnip. Severe phytophotodermatitis caused by exposure to cow parsnip (*Heracleum lanatum*) in Alaska. Another name for cow parsnip is “pushkie,” so this is also known locally as “pushkie burn.” (Photo contributor: Kathy McCue, MD.)



FIGURE 16.23 ■ Phytophotodermatitis—Lime. Phytophotodermatitis caused by exposure to limes and UV radiation in Jamaica. The patient had been exposed while riding shirtless on a horse through a lime orchard. (Photo contributor: Stephan Russ, MD.)

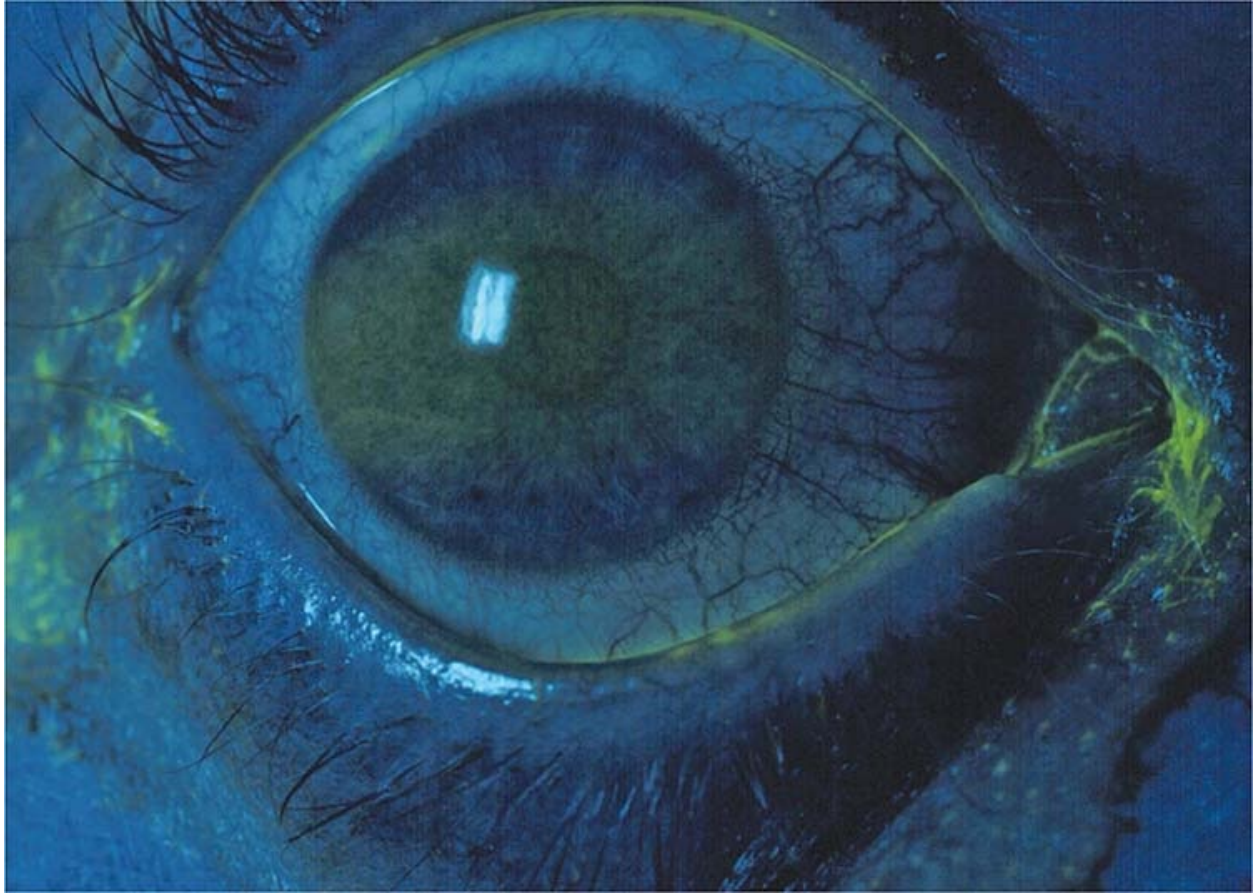


FIGURE 16.24 ■ Ultraviolet Keratitis. This patient presented with severe eye pain at 3 AM after a day of arc welding without proper eye protection. Notice the absence of fluorescein uptake where the eyelids were protective. (Photo contributor: Lawrence B. Stack, MD.)

Phototoxicity should be suspected in any patient with severe or exaggerated sunburn. Photoallergy is easily misdiagnosed as allergic eczema or contact dermatitis, especially since onset is often delayed up to 2 days after exposure. Phytophotodermatitis may mimic severe sunburn or contact dermatitis, especially rhus dermatitis. Endogenous photosensitizers (photodermatoses) include solar urticaria, porphyria cutanea tarda, polymorphous light eruption, and systemic lupus erythematosus. These may be provoked by visible light or by UV radiation.

Management and Disposition

Treatment of sunburn and sun poisoning involves standard burn therapy and supportive care. Sunburn is usually a self-limited problem. Cool compresses and nonsteroidal anti-inflammatory drugs may be beneficial. UV keratitis is treated with mydriatic-cycloplegic eye drops to decrease pain. Initial examination can

be facilitated by topical anesthetics. Severe cases may require antibiotic ointment, opiate analgesics, and bilateral eye patches for 12 to 24 hours, although the use of eye patches is controversial. Patients require 24- to 48-hour follow-up. Ophthalmology referral is indicated to rule out retinal damage.

Treatment of photosensitivity reaction has two components: treatment of the sunburn and recognition of the sensitizing agent or endogenous medical condition. Topical steroids and oral analgesics or antipruritics may be helpful. Systemic steroids may be necessary. Patients with severe reactions should be referred to a dermatologist for possible photo patch testing.

Pearls

1. *Para*-aminobenzoic acid (PABA) in sunscreens may be a photosensitizer and can cause a photoallergic reaction.
2. The properties of individual skin types produce marked differences in response to UV radiation.
3. Victims of UV keratitis typically present 2 to 12 hours after exposure. Patients may present during the night with severe unexplained bilateral eye pain.

LIGHTNING INJURIES

Clinical Summary

Lightning produces injury from high voltage, heat production, and explosive shock waves. Direct injuries include cardiorespiratory arrest, cardiac arrhythmias, and neurologic abnormalities such as seizures, deafness, confusion, amnesia, blindness, and paralysis. The patient may suffer contusions from the shock wave or from opisthotonic muscle contractions. Chest pain and muscle aches are common. One or both tympanic membranes (TMs) rupture in more than 50% of victims. Cataracts are usually a delayed occurrence. Hematologic abnormalities including disseminated intravascular coagulation have been described. Fetal demise may occur.

Burns may result from vaporization of sweat or moist clothing, heating of clothing and metal objects such as climbing equipment, belt buckles or bra wiring, and direct effects of the strike. Linear burns and punctate burns are thermal burns. Feathering burns, also known as ferning or Lichtenberg figures,

are not actual burns but likely represent superficial bruising. They are pathognomonic for lightning injury.

Diagnosis of lightning injury is straightforward when there is a thunderstorm, when there are witnesses to the strike, or when there are typical physical findings. Lightning on relatively sunny days (without thunder) striking a lone victim may produce a confusing picture. The scattering of clothing and belongings may mimic an assault. Side flashes from metal objects and wiring may produce indoor victims during storms.



FIGURE 16.25 ■ Linear Lightning Burns. Linear burns from lightning are due to thermal effects. (Photo contributor: William Barsan, MD.)



FIGURE 16.26 ■ Linear Lightning Burns. Linear lightning burns along the leg and foot of a strike victim. (Photo contributor: Sheryl Olson, RN, BSN, CCRN.)



FIGURE 16.27 ■ Punctate Lightning Burns. Punctate burns due to lightning are partial- or full-thickness thermal burns that range from a few millimeters to a centimeter in diameter. They are multiple and closely spaced. (Photo contributor: Arthur Kahn, MD.)

Management and Disposition

Care begins with the ABCs (airway, breathing, circulation). In the ED, a

thorough history and physical examination should be performed to identify associated injuries. Asymptomatic patients, including those with feathering, who have a normal ECG may be observed for several hours and discharged with referral to neurology, ophthalmology, and otorhinolaryngology because delayed sequelae are common. Patients with mild injuries should be admitted for neurologic and cardiovascular monitoring, with specialty consultation as needed. Patients with significant injuries should be admitted to a referral hospital with a full range of specialty services.



FIGURE 16.28 ■ Feathering. Feathering (furning) on the left side of a lightning strike victim. (Photo contributor: Sheryl Olson, RN, BSN, CCRN.)



FIGURE 16.29 ■ Feathering. Feathering (fern) burns are pathognomonic of lightning injury. These are not true burns but likely represent superficial bruising. (Photo contributor: Linda Katirji, MD.)

Pearls

1. The amount of damage to the exterior of the body does not predict the amount of internal injury.
2. Since lightning most commonly produces cardiac standstill by means of massive direct current countershock, prompt spontaneous return of normal heart rhythm, by virtue of cardiac automaticity, is the rule. However, respiratory arrest is often more prolonged. In a triage situation, the normal rules do not apply, since victims breathing spontaneously are already recovering. The rule in lightning strikes is to resuscitate the “dead.” Ventilatory support is often all that is required.
3. Keraunoparalysis (lightning paralysis) is a condition that is specific to lightning injury and is caused by extreme vasoconstriction, usually in the legs. It usually resolves spontaneously within a few hours.

4. Psychological sequelae following lightning injuries are underreported and may include memory loss, difficulty with concentration, and depression.



FIGURE 16.30 ■ Lightning Damage. Lightning can instantly heat metal objects, causing significant burns to skin in contact with the equipment. (Photo contributor: Nicholas Kanaan, MD.)

LARGE TERRESTRIAL ANIMAL ATTACKS

Clinical Summary

Both domestic and wild animals can attack humans. Injuries are caused by combinations of penetrating and blunt trauma. Penetrating injuries can be inflicted by teeth, claws, and horns, while severe blunt injuries may result from the victim being knocked over, trampled or otherwise crushed, thrown into the air, or dragged. Injuries may involve massive tissue injury or avulsion often associated with neurovascular damage and long bone fractures.

Management and Disposition

Evaluation and treatment are the same as for any other multiple trauma victim with a high-energy mechanism. The first priorities are to stop life-threatening hemorrhage, secure the airway, and manage other bleeding and circulation (the ABCs), which may be complicated by injuries of the face, neck, or chest. Victims should then be evaluated for less obvious blunt traumatic injuries. Open fractures and injuries to internal organs may not initially be clinically apparent, as with any victim of multiple trauma.

Pearls

1. Wounds often require operative debridement and repair by appropriate specialists. Consider tetanus and rabies prophylaxis in addition to antibiotic treatment for high-risk wounds.
2. Bite wounds can penetrate the skull or joints. Displaced teeth or claws may remain imbedded in wounds. Evaluation should include x-rays, ultrasound, and/or computed tomography (CT) scanning along with appropriate surgical consultations.



FIGURE 16.31 ■ Cougar Mauling. This patient sustained large wounds as a result of a cougar attack. Here, the weight and force of this large animal has resulted in severe shearing injuries. Penetrating injuries from large animals may be deeper than they appear. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.32 ■ Pit Bull Mauling. The left arm of a pit bull mauling victim. Note the open fracture of the radius. The patient sustained multiple severe defensive wounds to both arms, resulting in extensive soft-tissue and neurovascular injuries. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.33 ■ Grizzly Bear Attack—Open Scapula Fracture. Although grizzly claws are not very sharp, they can exert considerable force. This innocuous appearing injury was complicated by an open scapula fracture. (Photo contributor: Luanne Freer, MD.)



FIGURE 16.34 ■ Himalayan Black Bear Attack—Facial Trauma. Himalayan black bears often attack the face. Airway management may be facilitated by use of a surgical airway, as in this case from Bhutan. (Photo contributor: Charles Haviland Mize, MD.)

CORAL SNAKE ENVENOMATION

Clinical Summary

The most important species of coral snakes (Elapidae family) found in the United States are the eastern coral snake (*Micrurus fulvius*) and the Texas coral

snake (*Micrurus tener*). Coral snakes have small mouths. Bites are usually limited to fingers, toes, or folds of skin. Due to a less efficient venom apparatus than the Crotalids, coral snakes generally need to hold on or chew to effect a significant envenomation. The bite typically produces minimal local inflammation and pain. Paresthesias and muscle fasciculations are common. Systemic symptoms resulting from the powerful neurotoxic effects of the venom can include tremors, drowsiness, euphoria, hypersalivation, and respiratory distress. Cranial nerve involvement, manifested by slurred speech and diplopia, may be followed by bulbar paralysis with dysphagia and dyspnea. Death may result from respiratory and cardiac arrest. Onset of severe symptoms may be delayed up to 12 hours but may also be rapidly progressive.

Management and Disposition

In contrast to pit viper envenomation, prehospital application of a constrictive bandage may be of benefit in limiting the spread of neurotoxic coral snake venom. Severe systemic symptoms following envenomation by Elapidae may be delayed and cannot be accurately predicted by local wound reactions. Four to six vials of antivenom should be administered for any suspected envenomation by eastern or Texas coral snakes. Treatment of western coral snake (*Micruroides euryxanthus*) bites is purely supportive. Tetanus prophylaxis should be addressed.



FIGURE 16.35 ■ Coral Snake. United States eastern coral snake with typical coloring and red-on-yellow bands. (Photo contributor: Mike Cardwell, MS.)



FIGURE 16.36 ■ Coral Snake (Close-Up). A close-up view of the striking and distinctive markings of the highly venomous coral snake. (Photo contributor: Mike Cardwell, MS.)



FIGURE 16.37 ■ Nonvenomous Milk Snake. As opposed to the red-on-yellow rings seen in the venomous US coral snake, these red-on-black rings indicate a nonvenomous snake. Unfortunately, this applies only to animals native to the United States. (Photo contributor: Sean P. Bush, MD.)

Pearls

1. Treatment with antivenom should be initiated early in cases of eastern coral snake bites, since symptoms are often delayed and severe.
2. The adage “red on yellow, kill a fellow; red on black, venom lack” applies to all coral snakes found in the United States but does not hold true in other parts of the world.
3. As many as 60% of North American coral snake bites do not result in envenomation of the victim.



FIGURE 16.38 ■ Coral Snake Envenomation. Tooth mark from a coral snake bite. The snake latched onto this finger for 5 minutes. Ascending paresthesias after envenomation prompted intensive care unit admission and antivenom therapy. (Photo contributor: Lawrence B. Stack, MD.)

PIT VIPER ENVENOMATION

Clinical Summary

The pit vipers (Crotalidae family) indigenous to the United States include rattlesnake species, cottonmouths, and copperheads. Physical characteristics of pit vipers include a triangular head, heat-sensing pits, elliptical pupils, and a single row of subcaudal ventral scales. Pit viper venom is complex and produces hematologic, cardiovascular, and neuromuscular effects. Clinically, pit viper envenomations are divided into four categories. Bites without envenomation are characterized only by the direct tissue damage caused by the strike. Minimal envenomations consist of fang marks and local swelling only, with no systemic symptoms. Moderate envenomations include progression of tissue changes beyond the immediate location of the bite and/or systemic symptoms with mild changes in coagulation parameters. Severe envenomations include marked local

and progressive swelling and significant systemic symptoms with coagulopathy. Coagulopathy is manifested by subcutaneous ecchymosis and/or signs of bleeding).



FIGURE 16.39 ■ Eastern Diamondback Rattlesnake. The eastern diamondback is the largest US rattlesnake and has a characteristic diamond-shaped pattern on its dorsal aspect. Note the triangular head, which is characteristic of pit vipers. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.40 ■ Western Diamondback Rattlesnake. The western diamondback causes the most

fatalities from snakebites in Mexico and the second most in the United States behind the eastern diamondback. (Photo contributor: Mike Cardwell, MS.)



FIGURE 16.41 ■ Mojave Green Rattlesnake. The Mojave green is found in the southwestern United States and Mexico. There are two subspecies, Types A and B, with Type A thought to have the most toxic venom of all North American snakes. Unlike most rattlesnake venoms, Mojave Type A venom contains a potent neurotoxin. (Photo contributor: Mike Cardwell, MS.)



FIGURE 16.42 ■ Red Diamond Rattlesnake. The elliptical pupils and heat-sensing pits in this red diamond rattlesnake are characteristic of pit vipers. (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.43 ■ Cottonmouth. The cottonmouth is a semiaquatic venomous pit viper that may crawl or swim with its head raised at an angle of 45 degrees. When disturbed, it may open its mouth wide to reveal a white lining. (Photo contributor: Stephen J. Knoop.)



FIGURE 16.44 ■ Copperhead Snake. The copperhead is frequently encountered in wooded mountains, abandoned buildings, and damp, grassy areas. It is able to climb low bushes and trees in search of food. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.45 ■ Juvenile Copperhead Caudal Lure. Juvenile pit vipers may have bright yellow coloring on their distal tails, known as a caudal lure. The snakes are known to wiggle their tails to mimic insect prey, luring unsuspecting targets (such as frogs or lizards) into striking range. (Photo contributor: Timothy S. Forsythe, DO.)



FIGURE 16.46 ■ Rattlesnake Envenomation, Minutes Old. Timber rattlesnake envenomation on the leg of an unsuspecting victim. The fang marks are 4 cm apart. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.47 ■ Rattlesnake Envenomation, Day 1. This rattlesnake bite shows local swelling, some edema beyond the initial bite site, and a hemorrhagic bleb at 6 hours. (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.48 ■ Progression of Rattlesnake Envenomation, 7 Weeks. Seven weeks later, the patient shown in Fig. 16.47 has progressed to tissue loss, eschar formation, and mild changes in coagulation parameters. (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.49 ■ Moderate Rattlesnake Envenomation. This patient was bitten by a rattlesnake on the dorsal aspect of the right hand and presented with edema extending to the wrist as well as nausea and vomiting. (Photo contributor: Edward J. Otten, MD.)



FIGURE 16.50 ■ Severe Rattlesnake Envenomation. This patient sustained a rattlesnake bite to his hand and presented with marked and progressive swelling, subcutaneous ecchymosis, and a significant coagulopathy. (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.51 ■ Nonvenomous Snake. In contrast to the elliptical pupils and triangular head characteristic of pit vipers, this nonvenomous rat snake has a rounded head and circular pupils. (Photo contributor: Sean P. Bush, MD.)

Management and Disposition

Prehospital management of pit viper bites should include immobilization and rapid transport without delay. Lymphatic constriction bands and extractor devices should not be used. These devices may worsen focal complications of envenomation. Tourniquets and local incision are ineffective and generally do more harm than good. Electric shock, cryotherapy, and mouth suction are not recommended. ED management includes resuscitation, establishing a physiologic baseline, and determining the need for antivenom. CroFab (Savage Labs), a recombinant antivenom effective in neutralizing venom toxins, is the treatment of choice. Indications for antivenom administration include any progression of swelling, erythema, or ecchymosis beyond the immediate bite area, presence of any systemic signs of envenomation, or any coagulation abnormalities and/or bleeding complications associated with envenomation. The dose of antivenom increases with the severity of envenomation. In general, treatment is initiated with four to six vials of CroFab intravenously with further dosing regimen based on clinical course. Compartment syndrome is a possible complication of envenomation and should initially be treated with antivenom. Fasciotomy should be performed only if compartment pressures are greater than 30 mm Hg despite antivenom treatment. Patients who do not develop evidence

of envenomation after 8 hours of observation may be safely discharged home with close follow-up. Tetanus prophylaxis should be addressed and given when necessary.

Pearls

1. Up to half of all pit viper bites may be “dry” (without any injection of venom).
2. Intramuscular epinephrine (0.3 mg 1:1000) given prior to administration of horse serum antivenin may reduce the potential allergic response.

TROPICAL SNAKE ENVENOMATIONS

Clinical Summary

The epidemiology of snakebites in tropical regions differs considerably from that seen in more temperate climates. In general, the absolute number of venomous snakes is higher in the tropics, and snakes are often located in areas of high population density. The prevalence of bites is also higher due to differences in agricultural and hunting practices, frequent flooding, lack of adequate footwear in many locations, and housing that allows access of snakes into living areas. The annual mortality of snakebite in India may exceed 20,000. Snakebite is said to be the 5th most common cause of death in Myanmar. In some indigenous populations in South America, up to 20% of adult deaths are from snakebite. Places where snakebites are common are often in remote locations where medical care may not be immediately available. Signs and symptoms depend on the type of envenomation and the amount of toxin injected. Local pain, swelling, and blistering are seen with many snake-bites. Clotting disturbances, frank hemorrhage, and shock are seen with many viper bites, while neurotoxicity can be seen with elapid and sea snake bites.



FIGURE 16.52 ■ Fer-de-Lance (*Bothrops jararaca*). Fer-de-Lance, French for “lance head,” refers to a number of venomous pit vipers of the genus *Bothrops* in Central and South America (*B atrox*, *B asper*, *B jararaca*, *B lanceolatus*). This family of snakes is responsible for more deaths than any other New World snake. (Photo contributor: Cybele Sabino Lisboa, BSc.)



FIGURE 16.53 ■ Fer-de-Lance Bite. This patient was bitten on the index finger by a Fer-de-Lance snake while clearing bush in rural Peru. Note the ecchymosis proximal to the bite. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 16.54 ■ Fer-de-Lance Bite (Late). Local tissue destruction at the site of a *Bothrops atrox* bite in a Guyanese teenager. (Photo contributor: Ian Jones, MD.)



FIGURE 16.55 ■ Old World Viper Bite. Local hemorrhagic manifestations at the site of a snake bite in a Syrian man. Old World vipers lack the heat-sensing pits of the pit vipers. Bites can cause significant hemorrhagic, coagulopathy, and local tissue complications. (Photo contributor: Seth W. Wright, MD.)



FIGURE 16.56 ■ Clotting Test. Positive results of a 20-minute whole blood clotting test are seen with the blood from a man bitten by a Russell's viper. Lack of clotting at 20 minutes correlates closely with other clotting studies. (Photo contributor: Christian Medical College, Vellore, India.)

Management and Disposition

Patients should be reassured and kept as immobile as possible. Any involved extremity should be splinted. Tourniquets are generally discouraged but may be useful for treating some neurotoxic snakebites. Many modalities such as suction, incision, pumping apparatus, cryotherapy, and electrical shocks have been advocated, but are of no proven benefit and may be harmful. Specific antivenin is used as indicated but is often not available or prohibitively expensive.

Pearls

1. There are approximately 3000 species of snakes in the world, with 600 being venomous and over 200 considered to be medically important. Venomous snakes can be found at altitudes as high as 4000 m in equatorial tropical regions.
2. Neurotoxicity is seen in bites of elapids such as kraits, coral snakes, mambas, and cobras, but is not a feature of the African spitting cobra. The most common cause of death in neurotoxic envenomation is respiratory paralysis.
3. Snake venom ophthalmia is the syndrome caused by the spitting cobras. Severe pain, swelling, and corneal ulceration are seen. Blindness can be a secondary complication.

4. A simple bedside 20-minute blood clotting test can be used to determine the presence of significant coagulopathy in areas without laboratory coagulation capability.
5. The World Health Organization has an online database with all antivenom products currently under production.

GILA MONSTER ENVENOMATION

Clinical Summary

The Gila monster (*Heloderma suspectum*) is a venomous lizard found in the southwestern United States and northwestern Mexico. The only other venomous lizard in the Americas is the Mexican beaded lizard, found from Mexico to Guatemala. Both species are unlikely to bite unless provoked. They secrete venom into their saliva and increase saliva production when they are agitated. When biting their victim, the Gila monster chews rather than injecting, is known to hang on vigorously, and often must be forced to release its powerful grasp. Teeth may break off and contaminate wounds. Like snake venoms, Gila monster and beaded lizard venoms are complex mixtures of proteolytic enzymes and vasoactive substances. The bite may cause pain with local edema and proximal radiation. Tachycardia and hypotension may occur, along with anaphylactic reactions. Systemic complaints may include generalized weakness, nausea and vomiting, diaphoresis, and paresthesias.

Management and Disposition

If not already separated from the victim in the prehospital phase, the lizard must be removed. Putting the lizard under running hot water or prying the jaws apart using an appropriate tool are two options. Caregivers should take precautions against being bitten themselves and should also take care to ensure the victim is not bitten again in the removal process. Hypotension can be treated with volume resuscitation. Anaphylaxis should be treated with epinephrine. There is no anti-venom. Wound care should include copious irrigation and debridement as needed. Imaging should be used to rule out retained teeth. Tetanus prophylaxis is indicated. Prophylactic antibiotics are generally unnecessary. If the victim has no systemic symptoms several hours after observation, discharge with return

precautions is appropriate. Patients with systemic symptoms should be admitted.



FIGURE 16.57 ■ Reticulate Gila Monster. The Gila monster (*Heloderma suspectum*) is one of two venomous lizards found in the Americas. Its range is the southwestern United States and northwestern Mexico. (Photo contributor: Jeff Servoss, US Fish and Wildlife Service.)



FIGURE 16.58 ■ Gila Monster Bite. Gila monster bites may be extremely painful. They usually do not cause severe tissue damage. Retained teeth are a hazard. (Photo contributor: John Sakles, MD.)



FIGURE 16.59 ■ Gila Monster Bite, Tongue Swelling. Gila monster bites may cause anaphylactic reactions. (Photo contributor: John Sakles, MD.)

Pearls

1. Gila monsters and Mexican beaded lizards have very powerful jaws. Forcible removal is often necessary and must be carefully undertaken to avoid further bites.
2. Patients with systemic symptoms should be admitted, because progression to anaphylactic reactions is possible.

BLACK WIDOW SPIDER ENVENOMATION

Clinical Summary

The black widow spider (*Latrodectus mactans*) is the prototype for the genus *Latrodectus*, several members of which cause human disease. The black widow spider is not particularly aggressive but will defend her web, which is often found in woodpiles, basements, and garages. Most envenomations occur

between April and October, with bites most commonly located on the hand and forearm. The clinical presentation of severe and sustained muscle spasm is produced by a neurotoxic protein, which causes the release of acetylcholine and norepinephrine at the presynaptic neuromuscular junction. The initial bite may be mild to moderately painful but is often missed. Within approximately 1 hour, local erythema and muscle cramping begin, followed by generalized cramping involving large muscle groups such as the thighs, shoulders, abdomen, and back. Associated clinical features can include fasciculations, weakness, fever, salivation, vomiting, diaphoresis, localized sweating at the envenomation site, and a characteristic pattern of facial swelling called *Latrodectus* facies. Rare cases of seizures, uncontrolled hypertension, and respiratory arrest have occurred.

Management and Disposition

Treatment of the local wound should include cleansing and tetanus prophylaxis. Severe pain and spasm may require intravenous benzodiazepines and opiates. Calcium gluconate infusion has long been recommended to reduce symptoms, although evidence for its efficacy is controversial. Antivenom exists but carries the same risk as all horse serum products. Antivenom should be considered only in cases of respiratory arrest, seizures, uncontrolled hypertension, and pregnancy.



FIGURE 16.60 ■ Black Widow Spider (With Offspring). *Latrodectus mactans*, with characteristic hourglass marking on its abdomen. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 16.61 ■ Black Widow Spider Bite. The bite of the black widow spider is clinically subtle. Local reaction is usually trivial, as in this confirmed bite with a small patch of mild erythema. (Photo contributor: Gerald O'Malley, DO.)



FIGURE 16.62 ■ Black Widow Facies. A pattern of facial swelling, known as *Latrodectus facies*, may occur several hours after envenomation. (Photo contributor: Gerald O'Malley, DO.)

Pearls

1. Of the five *Latrodectus* species indigenous to the United States, only three are black and only one has the orange-red hourglass marking.
2. Envenomations by *L. mactans* can mimic an acute abdomen and should be considered in the differential diagnosis of patients presenting with severe acute abdominal pain.

BROWN RECLUSE SPIDER ENVENOMATION

Clinical Summary

The brown recluse spider (*Loxosceles reclusa*) is the prototypical member of the genus *Loxosceles*, which as a group can produce necrotic arachnidism following envenomation. These small spiders (approximately 1 cm in body length and 3 cm in leg length) have a worldwide distribution and are identified by fiddle-shaped markings on their anterodorsal cephalothorax. Initial envenomation may be painful, although patients often report no recollection of being bitten. Initial stinging gives way to aching and pruritus. The wound then may become edematous, with an erythematous halo surrounding a violaceous center. The erythematous margin often spreads in a pattern influenced by gravity, leaving the necrotic center near the superior aspect of the lesion. Bullae may erupt, and—over a period of 2 to 5 weeks—the eschar sloughs, leaving a deep, poorly healing ulcer. In approximately 10% of cases, systemic symptoms (loxoscelism) are present. Systemic features of brown recluse envenomation may include fever, nausea, vomiting, headache, morbilli-form rash, arthralgias, and, in severe cases, hemolytic anemia, coagulopathy, renal failure, and even death. Children are at higher risk of systemic disease.



FIGURE 16.63 ■ Brown Recluse Spider. Brown recluse spider with characteristic “fiddle” marking on the anterodorsal aspect of the cephalothorax. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.64 ■ Fiddle Back Marking. A close-up look at the characteristic fiddle back marking of the brown recluse spider. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.65 ■ Early Brown Recluse Spider Bite. Early brown recluse spider bite (approximately 8 hours) with a violaceous center surrounded by faint-spreading erythema. Note the “red, white, and blue” appearance. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 16.66 ■ Later Recluse Spider Bite (24 hours). Brown recluse spider bite at approximately 24 hours. Note asymmetric spread of erythema and early central ulcer formation. (Photo contributor: Edward Eitzen, MD, MPH.)



FIGURE 16.67 ■ Recluse Necrosis (Weeks). Within about 2 to 5 weeks, significant brown recluse envenomations may produce a deep, poorly healing ulcer with necrosis. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Most cutaneous lesions secondary to brown recluse spider bites can be managed with cold compresses, elevation, loose immobilization, and tetanus prophylaxis. Severe lesions may require reconstructive plastic surgery several weeks after wound stabilization. Dapsone, recommended in the past, should not be used. Any systemic reaction with evidence of hemolysis, hemoglobinuria, or coagulopathy should prompt admission. Hyperbaric oxygen therapy and antivenom (not available in the United States) have been suggested as possible adjuncts, but no clear consensus of preferred treatment has been established.

Pearls

1. The asymmetric spread of erythema, due to the local effects of gravity on the toxin, may help to distinguish a brown recluse spider bite from other arthropod envenomations.
2. Dapsone should not be used. The therapy is ineffective and may induce methemoglobinemia.
3. Urinalysis may be helpful in identifying hemolysis early in the clinical course of system reactions.



FIGURE 16.68A ■ Brown Recluse Spider Bite. Red, white, and blue appearance with significant asymmetric spread of erythema inferiorly down the arm beyond the envenomation site due to effect of gravity. (Photo contributor: Shannon B. Snyder, MD.)



FIGURE 16.68B ■ Systemic Loxoscelism. This patient suffered fever, headache, and a diffuse erythematous rash as a result of systemic loxoscelism. Note the brown recluse envenomation on the patient's right arm, seen also in Fig.16.68A. (Photo contributor: Shannon B. Snyder, MD.)

SCORPION ENVENOMATION

Clinical Summary

The most significant morbidity and mortality from scorpion stings is from the Buthidae family, characterized by a triangular central sternal plate. This family includes the venomous *Androctonus* genus in northern Africa, *Leiurus* in the Middle East, *Tityus* in South America, and *Centruroides* in North America.

Centruroides are found primarily in the southwestern United States and northern Mexico and are characterized by a variable subaculear tooth beneath the stinger. They may be striped and are yellow to brown in color. These scorpions tend to hide in crevices, woodpiles, bedding, clothing, and shoes. Envenomation produces a mild local reaction of pain, swelling, burning, and ecchymosis.

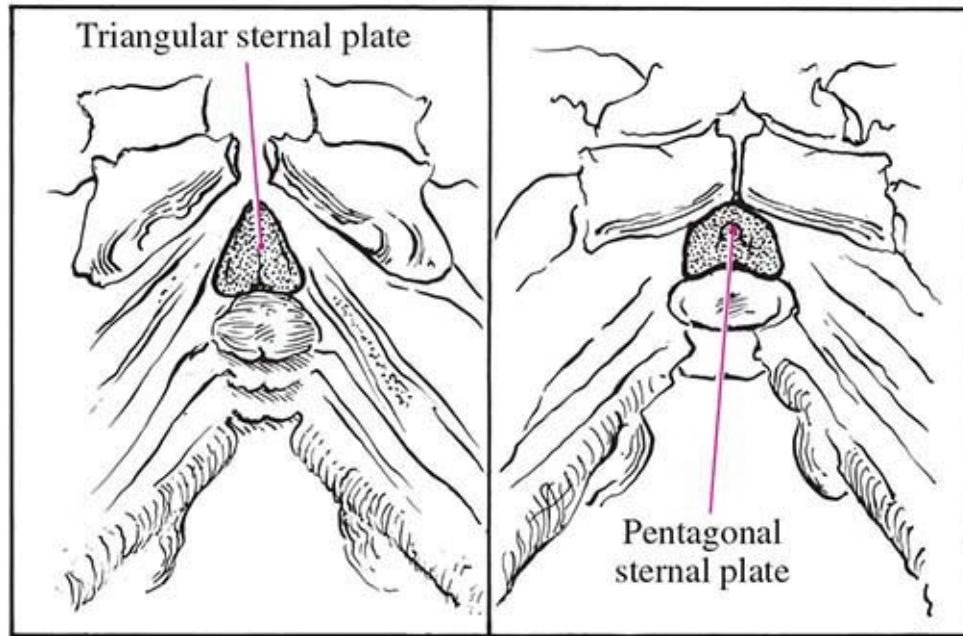


FIGURE 16.69 ■ Buthidae Sternal Plate. The Buthidae family is associated with the most significant envenomations and is characterized by triangular sternal plates (left). Members of the other scorpion families have pentagonal sternal plates (right).



FIGURE 16.70 ■ Buthidae Sternal Plate. The triangular appearance of the sternal plate is well seen in this scorpion, a member of the Buthidae family. (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.71 ■ *Centruroides exilicauda*. Members of this species are yellow to brown and usually less than 5 cm long. Below the stinger is the telson, within which are two glands containing venom. (Photo contributor: Sean P. Bush, MD.)

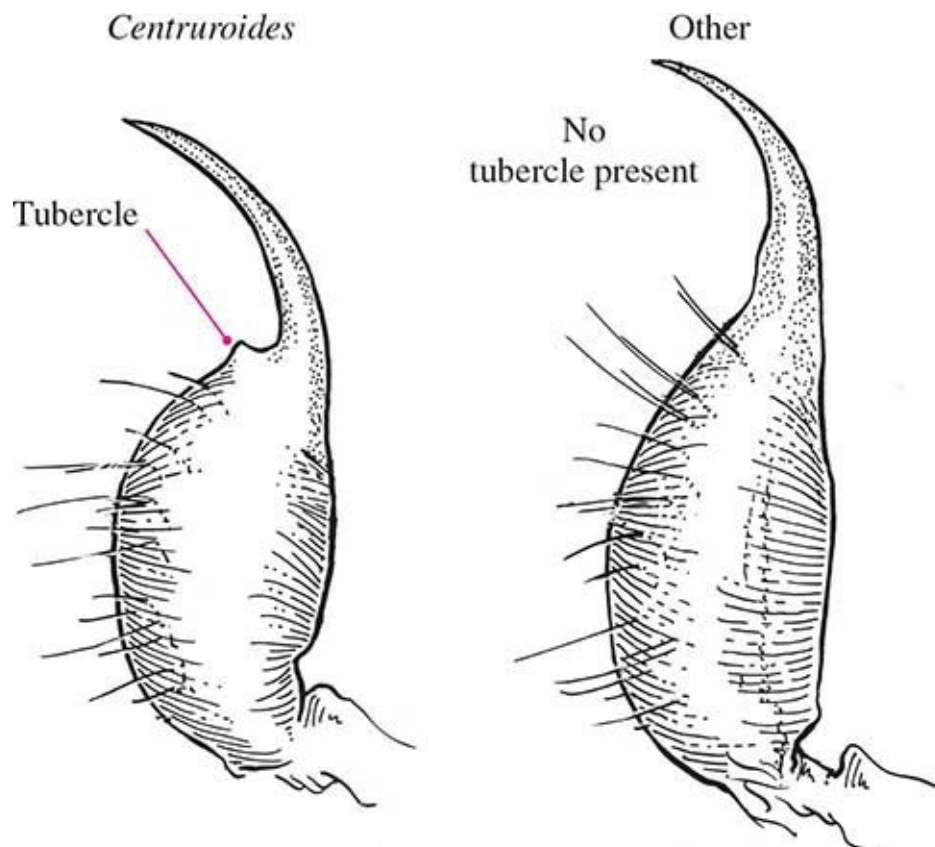


FIGURE 16.72 ■ **Subaculear Tooth.** The barb noted at the base of the stinger is variably present in *Centruroides* (left) and absent in other species (right).

Centruroides exilicauda (the bark scorpion) envenomation can lead to progressive symptoms and, very rarely, death. The venom of *C exilicauda* initially produces local paresthesias and pain (grade 1), which may be accentuated by tapping the involved area. More severe envenomations may produce remote paresthesias (grade II) and either somatic or autonomic nervous system dysfunction (grade III). Systemic symptoms may include tachycardia, nausea, wandering eye movements, blurred vision, difficulty breathing, trouble swallowing, restlessness, and involuntary shaking. Both somatic and autonomic dysfunction may be present (grade IV). Systemic reactions tend to be more severe in younger patients and may result in death, usually from respiratory arrest.



FIGURE 16.73 ■ *Centruroides limbatus*. Subaculear tooth. A variable subaculear tooth is characteristic of *Centruroides*. This is an example of a large subaculear tooth on the telson from *C limbatus*. *Centruroides exilicauda* typically has a smaller, sometimes subtle “tooth.” (Photo contributor: Sean P. Bush, MD.)

Management and Disposition

Treatment depends on the severity of envenomation. Grade I or II envenomations are treated with supportive care (ice, oral analgesia) and tetanus prophylaxis. Envenomations that progress to grade III or IV should be treated aggressively and may require paralysis and intubation for severe spasms. *Centruroides* antivenom (Anascorp) is available but is horse serum based and carries a risk of hypersensitivity reactions. Pain and paresthesias may persist for up to 2 weeks. Most systemic symptoms improve within 9 to 30 hours without antivenom treatment and usually peak at about 5 hours.



FIGURE 16.74 ■ Scorpion Sting. Most scorpion envenomations are mild and produce local pain, swelling, paresthesias, and mild ecchymosis. (Photo contributor: Stephen W. Corbett, MD.)

Pearls

1. Exercise caution when treating pain of *C exilicauda* envenomations with opioids, as synergistic respiratory depressive effects between venom and opioids may occur.
2. If the scorpion is brought in, it should be examined for the presence of a triangular plate and subaculear tooth.
3. Almost all scorpions, including *C exilicauda*, fluoresce with intense brightness under cobalt light.

TICKS

Clinical Summary

Ticks are blood-sucking parasites of people and animals. Ticks cause illness by acting as vectors for pathogens or by secreting toxins or venoms. Ticks carry more types of infectious pathogens than any other arthropods except mosquitoes. The most important pathogens include *Borrelia* (responsible for Lyme disease and relapsing fever), *Rickettsia*, including Rocky Mountain spotted fever (RMSF), *Ehrlichia* (ehrlichiosis), viral pathogens, such as Colorado tick fever, and babesiosis. Rashes are prominent in Lyme disease, RMSF, and southern tick-associated rash illness (STARI), sometimes present in relapsing fever, uncommon in Colorado tick fever, and absent in babesiosis.

Clinically important ticks in North America include *Ixodes dammini*, the deer tick (Lyme disease and babesiosis); *Dermacentor andersoni*, the wood tick (RMSF and Colorado tick fever); *Dermacentor variabilis*, the dog tick (RMSF, ehrlichiosis); and *Amblyomma americanum*, the lone star tick (a very widespread tick implicated in the transmission of Lyme disease outside of the range of *I. dammini* as well as STARI and ehrlichiosis). More than 40 species of ticks can cause tick paralysis. In North America, the most common cause is *D. andersoni*, but *A. americanum* and *Ixodes* species have also been associated with tick paralysis.

Tick paralysis develops 5 to 6 days after an adult female tick attaches. Over the next 24 to 48 hours, an ascending, symmetric, flaccid paralysis develops. Alternative presentations include ataxia and associated cerebellar findings without muscle weakness or isolated facial paralysis. Resolution of the paralysis after removal of the tick establishes the diagnosis.



FIGURE 16.75 ■ Deer Tick. *Ixodes dammini*, the deer tick, is a vector of Lyme disease and babesiosis. (Photo contributor: Centers for Disease Control and Prevention, Atlanta, GA.)



FIGURE 16.76 ■ Wood Tick. *Dermacentor andersoni*, the wood tick, is a vector of Rocky Mountain spotted fever and Colorado tick fever. (Photo contributor: Centers for Disease Control and Prevention, Atlanta, GA.)

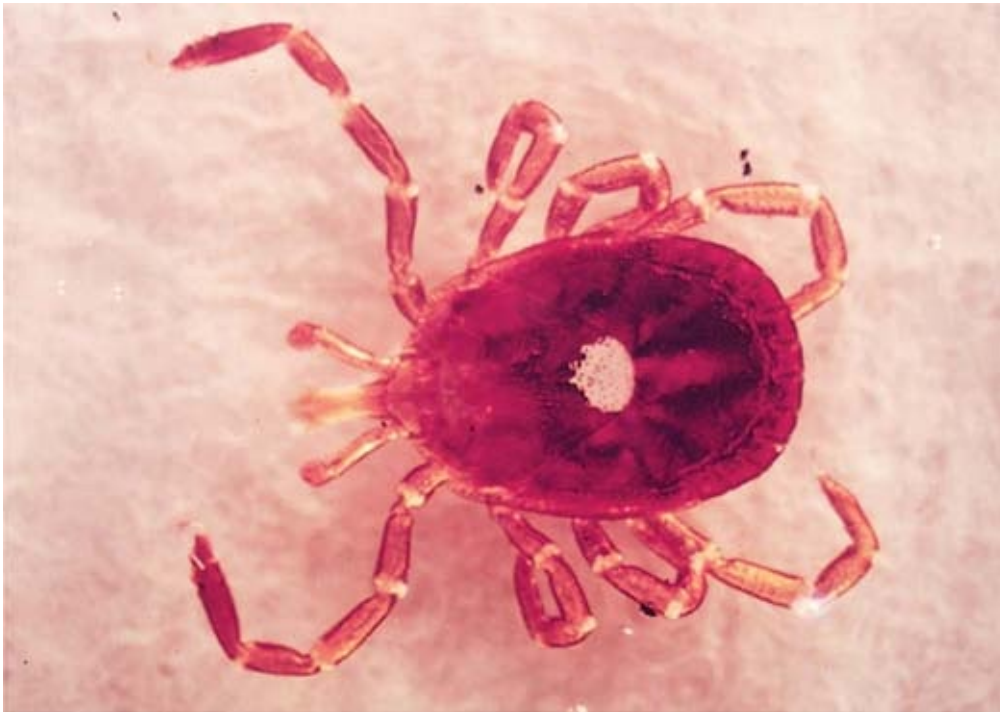


FIGURE 16.77 ■ Lone Star Tick. *Amblyomma americanum*, the lone star tick, has been implicated as a vector in STARI. (Photo contributor: Sherman Minton, MD.)



FIGURE 16.78 ■ Imbedded Tick. This lone star tick was found imbedded in the patient's shoulder. It was easily removed intact with tweezers. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.79 ■ American Dog Tick. *Dermacentor variabilis*, the dog tick, is a vector of Rocky Mountain spotted fever and ehrlichiosis. (Photo contributor: R. Jason Thurman, MD.)

Management and Disposition

If still embedded, the tick should be removed promptly by grasping it as close to the skin surface as possible, using blunt curved forceps or tweezers. The tick should be pulled out with slow, gentle traction, taking care not to crush or squeeze the body, which may result in injection of contaminated tick fluids. Other methods of tick removal—such as application of fingernail polish, isopropyl alcohol, or a hot match head—have not been proven to effect detachment and may induce regurgitation of tick contents into the wound.

Patients with tick paralysis may require supportive care, including mechanical ventilation. Patients with tick-borne illnesses may require admission for supportive care or intensive antibiotic treatment, but when clinically appropriate,

patients may be treated as outpatients with appropriate antibiotic therapy.



FIGURE 16.80 ■ Erythema Migrans Rash. This erythema migrans rash was located at the site of a tick bite in a patient who developed southern tick-associated rash illness (STARI). Erythema migrans rash is also associated with Lyme disease. (Photo contributor: Shannon B. Snyder, MD.)

Pearls

1. Prevention of tick bites includes the use of protective clothing containing permethrin.
2. A clear history of a tick bite is present in less than one-third of Lyme disease cases.
3. Unusual neurologic presentations, particularly bilateral peripheral seventh-nerve palsies, should prompt consideration of Lyme disease.
4. Patients with paralysis in endemic areas should be thoroughly searched for embedded ticks.

HYMENOPTERA ENVENOMATION

Clinical Summary

The order Hymenoptera includes wasps, hornets, yellow jackets, bees, and ants.

Envenomation usually results in local pain, mild erythema, swelling, and pruritus. Severe systemic or toxic reactions may occur from one or multiple stings, manifesting as gastrointestinal symptoms, headache, pyrexia, muscle spasms, or seizures. Anaphylaxis may occur within minutes from a single sting and may cause death from airway obstruction and/or cardiovascular collapse. A serum sickness–type reaction may occur 7 to 14 days after envenomation.



FIGURE 16.81 ■ Paper Wasp. Paper wasps are found throughout the world and often establish nests close to or within human dwellings. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.82 ■ Paper Wasp Nest. A typical paper wasp nest in a roof corner. Disturbance of a nest may result in swarming attacks. (Photo contributor: Clay B. Smith, MD.)



FIGURE 16.83 ■ Fire Ant Mound. This typical fire ant mound is a raised area of dirt in an urban yard. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.84 ■ Fire Ant Bites. These fire ant bites on the anterior knee occurred after this patient knelt on a mound. These bites are 3 days old; the initial sterile pustules have begun to crust over. (Photo contributor: Alan B. Storrow, MD.)

Solenopsis invicta was imported from South America and is the most prominent fire ant in the United States. These ants are primarily found in the South and build mound nests in open grass settings, commonly in urban yards. Disturbing the nests may result in severe swarming attacks, a common occurrence in the unsuspecting barefoot victim. Bites are painful and produce sterile pustules that crust over in a few days.

Management and Disposition

Anaphylaxis is treated with conventional therapy with careful attention to airway management. Local reactions may be treated with ice packs, steroid cream, and oral antihistamines. Opiates may be needed for severe pain.



FIGURE 16.85 ■ Honeybee Envenomation. Many honeybee sting-ers (barbs and venom sacs) are seen on this patient's cheek and ear and along the hairline. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.86 ■ Honeybee Stingers. The barbs and attached venom sacs (stinger apparatus) after removal from the patient. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. Honeybee stings are usually apparent since the stinger apparatus, including barb and venom sac, is often detached and present on the patient's skin. These should be removed by scraping them off, as grasping with tweezers may result in the release of more venom.
2. “Brazilian killer” or “Africanized” bees are present primarily in the southwestern United States. Their venom is not known to be more toxic than that of other bees, but their aggressiveness, tendency to swarm in large numbers, and ability to travel long distances make them potentially more dangerous to humans.
3. Immediately remove any rings when envenomations are located on the hands or feet as marked local swelling can occur, putting the distal digits at risk for ischemia.



FIGURE 16.87 ■ Imbedded Honeybee Stinger. Note the venom sac, still pulsating and attached to the honeybee stinger imbedded in the victim. (Photo contributor: Lawrence B. Stack, MD.)

ENVENOMATION

Clinical Summary

Caterpillar venom apparatus typically consists of barbed spines arranged in clumps or scattered on the dorsal surface of the insect. These are purely defensive in nature. Patients who are stung commonly have intentionally handled the insect or have had accidental skin contact while gardening. Envenomated patients typically present with acute pain followed by focal erythema and swelling. Caterpillars with a less sophisticated venom apparatus or low-potency venom may cause simple focal pruritus or urticaria, although some caterpillars are capable of producing a very painful sting requiring aggressive pharmacologic pain control. Systemic symptoms are rare. The puss caterpillar, or wooly slug (*Megalopyge opercularis*), is perhaps the most well-known and important venomous caterpillar in the United States. Wooly slug caterpillars have a widespread distribution, appear hairy and flat, and may reach a length of 4 cm.



FIGURE 16.88 ■ Caterpillar with Barbed Spines. Typical garden caterpillar with barbed spines arranged in clumps. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.89 ■ Caterpillar Sting. Appearance of a caterpillar sting at 2 hours. The patient presented with moderate pain and severe itching. Note how the erythema follows the pattern of the caterpillar. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.90 ■ Puss Caterpillar. The “puss caterpillar” or “woolly slug” is likely the most important venomous caterpillar in the United States. The hairy appearance and small hair tail are characteristic. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.91 ■ Centipede. Note the curved “fangs” (actually modified legs) on the first segment of this centipede from Texas. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.92 ■ Centipede. This dead centipede was retrieved from the photographer’s shoe the day after he developed diffuse urticaria for no apparent reason on a trek in Nepal. (Photo contributor: Ken Zafren, MD.)



FIGURE 16.93 ■ Chigger Bites. Chigger bites on a leg appear as puncta surrounded by erythema. (Photo contributor: Kevin J. Knoop, MD, MS.)

Chiggers are the larvae of trombiculid mites and may inflict multiple intensely pruritic bites on their victims. They are parasitic only as larvae. They infest humans by crawling onto them and latching on. Proper clothing precautions and repellents are usually effective in reducing unpleasant chigger infestations.

Centipedes are venomous arthropods that have one pair of legs per body segment. The 1st segment contains hollow curved “fangs” (really modified legs) bearing venom glands at the bases, which are capable of penetrating human skin. Centipedes generally use venom to kill prey but, when provoked, may envenomate humans and produce local intense burning pain, erythema, and swelling. Systemic reactions may occur but are uncommon.

Management and Disposition

Treatment of caterpillar, mite, and centipede envenomations is purely supportive and consists of pain control, antihistamines, topical antipruritic creams, and basic wound care.

Pearls

1. Attached caterpillar spines may be removed easily with adhesive tape.
2. Infiltration with local anesthetics may be useful in markedly painful centipede envenomations or for the removal of retained “fang” fragments.
3. The caudal appendages of centipedes are not associated with a venom apparatus.

SKEETER SYNDROME

Clinical Summary

Skeeter syndrome is not an envenomation. It is an allergic reaction to polypeptides in mosquito saliva. Widespread inflammation may mimic cellulitis. Initially characterized by erythema, pruritis, and warmth, the rash becomes swollen within hours. Papules, vesicles, and bullae may develop in 8 to 12 hours. Because salivary peptides vary among species, individuals may be allergic to some species of mosquitos but not others.

Management and Disposition

In mild cases, oral antihistamines and topical steroid creams are effective. In severe cases, systemic steroids are indicated.

Pearls

1. A history of mosquito bites and the time course of the rash can help distinguish skeeter syndrome from cellulitis.
2. Antibiotics are not indicated in the treatment of skeeter syndrome.



FIGURE 16.94 ■ Skeeter Syndrome. This patient presented with erythema, swelling, and itching of the anterior thighs and a large bullous lesion on the left anterior thigh less than 24 hours after mosquito bites. He was wearing shorts without repellent when bitten. (Photo contributor: Simant Singh Thapa, MD.)



FIGURE 16.95 ■ Skeeter Syndrome. The right leg of the patient in [Fig. 16.94](#). The area was highly pruritic, and smaller vesicles and bullae were noted. He was treated with oral antihistamines and topical steroid cream with resolution of his rash in 2 weeks. (Photo contributor: Simant Singh Thapa, MD.)

MIDDLE EAR SQUEEZE

Clinical Summary

Middle ear squeeze (barotitis media) results from a decrease in pressure within the middle ear as an individual descends through water or is exposed to an increase in atmospheric pressure that can be seen in descending aircraft or while driving in mountainous terrain. According to Boyle's law, as pressure increases, volume decreases proportionately. At a depth of approximately 1.2 m (4 ft), the pressure difference is great enough to collapse the eustachian tube and cause obstruction. If attempts to equalize the pressure such as a Valsalva or Frenzel maneuver fail, ascent is necessary or injury may ensue. If a diver continues to descend, hemorrhage and edema occur within the middle ear and rupture of the

TM may occur. The influx of water into the middle ear may cause extreme vertigo and lead to a diving disaster.

Barotitis media may present with pain only (grade 0), TM erythema (grade 1), erythema and mild TM hemorrhage (grade 2), gross TM hemorrhage (grade 3), free middle ear blood (grade 4), or free blood with TM perforation (grade 5).

Management and Disposition

Treatment includes decongestants and appropriate analgesia. Antihistamines may be of use for allergy-related eustachian tube dysfunction. Antibiotics are recommended for preexisting infections or for TM rupture. Most cases resolve spontaneously within hours to days. The patient should not resume diving until the condition has resolved or the TM is completely healed.



FIGURE 16.96 ■ Barotitis Media. Tympanic membrane erythema and mild hemorrhage consistent with barotitis media. (Photo contributor: Richard A. Chole, MD, PhD.)

Pearls

1. Barotitis media is the most common medical problem associated with diving.
2. Associated barotraumatic injuries should be considered when the diagnosis of barotitis media is made.

MASK SQUEEZE

Clinical Summary

Mask squeeze results when a diver fails to maintain the balance between the air pressure within the mask and the external water pressure during descent. If a diver descends without equalizing pressure by exhaling through the nose, significant negative air pressure will exist inside the mask. The net result may include rupture of capillary beds, leading to conjunctival hemorrhage and skin ecchymosis.

Management and Disposition

Treatment consists of ascent and supportive care. A history of recent eye surgery or use of anticoagulant medication should be sought. A thorough eye examination should be performed and ophthalmologic consultation considered if warranted.

Pearls

1. Diver education and proper diving technique minimize the risk of mask squeeze.
2. Special consideration should be given to patients with anticoagulant use or recent keratotomy, as corneal incisions heal relatively slowly.



FIGURE 16.97 ■ Mask Squeeze. Mask squeeze in a diver who descended to 45 FSW without exhaling into his mask. (Photo contributor: Kenneth W. Kizer, MD; reprinted with permission from Auerbach PS (ed). *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. 3rd ed. St. Louis, MO: Mosby-Year Book; 1995. Copyright © Elsevier.)

STINGRAY ENVENOMATION

Clinical Summary

Stingrays are found throughout the oceans of the world. Sting-rays are not typically aggressive, and the majority of envenomations are defensive in nature. Injuries typically involve a lower extremity if the animal is stepped on or an upper extremity if the animal is handled. Fatal injuries have been reported from chest trauma, which may result in perforation of the myocardium. Stingray envenomation occurs when a reflexive and forceful forward thrust of the caudal spine or spines of the animal impacts the victim, producing a puncture wound or laceration. The force of injection causes the integumentary sheath covering the spine to be driven into the wound, fragmenting and potentially releasing venom, mucus, pieces of the sheath, and spine fragments deep within the wound. Envenomation typically produces immediate and intense pain, edema, and bleeding. The initially dusky or cyanotic wound may progress to erythema, with rapid fat and muscle hemorrhage. Systemic symptoms may include nausea, vomiting, diarrhea, diaphoresis, muscle cramps, fasciculations, weakness, headache, vertigo, paralysis, seizures, hypotension, and syncope.

Management and Disposition

The wound should be irrigated immediately and primary exploration accomplished to remove any visible debris. Pain relief should be initiated early. Opiates may be needed. Stingray venom is made up of heat-labile polypeptides that may be inactivated by immersion in hot water (43°C-46°C [110°F-115°F]) for 30 to 90 minutes. After soaking, wounds should be formally explored, debrided, and dressed for delayed primary closure or primary closure with drainage. Surgical consultation may be warranted in certain injury locations. Imaging should be obtained after debridement to further examine for retained foreign bodies. Broad-spectrum antibiotics covering marine organisms are recommended. Patients can usually be discharged home after a 3- to 4-hour observation period if no systemic symptoms occur. Tetanus prophylaxis should be given if indicated.

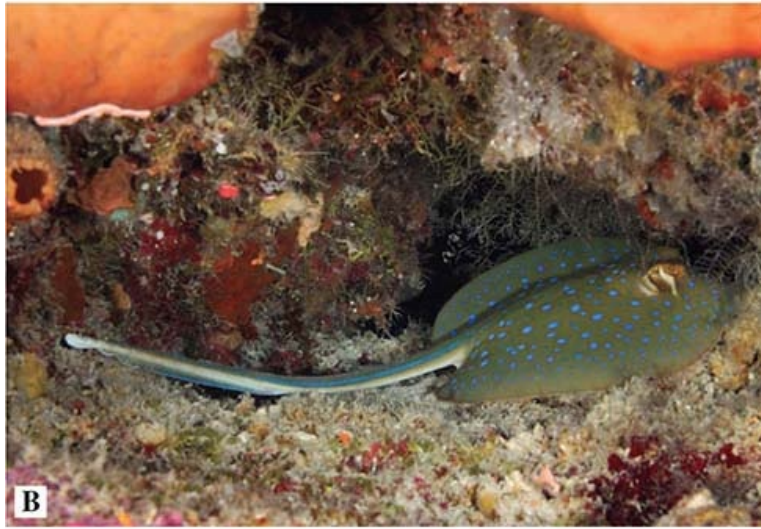


FIGURE 16.98 ■ Stingrays. (A) Spotted eagle stingray. This graceful stingray was photographed in waters off the coast of Bonaire. Note the three venomous spines at the base of the tail. (Photo contributor: Lynne Bentsen, RN.) (B) Blue spotted stingray. This stingray was photographed in waters off Indonesia. Stingrays often dwell on the ocean floor and may burrow into the sand, leading to envenomation by accidentally stepping on the animal. (Photo contributor: Ian D. Jones, MD.) (C) Southern stingray. This stingray was photographed in the Caribbean. The coloring of the stingray tends to blend with the ocean floor, leading to injury from inadvertent stepping on the stingray. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 16.99 ■ Buried Stingray. Stingrays like to burrow into the sand on the ocean floor, making them very difficult to see and easy to inadvertently step on. (Photo contributor: Keven Reed, OD.)

Pearls

1. Aggressive debridement is of primary importance in managing stingray wounds, as retained foreign bodies are a common problem.
2. Bacteria cultured from marine envenomations are extremely diverse. Antibiotics chosen should include coverage of staphylococci, streptococci, and *Vibrio* species.



FIGURE 16.100 ■ Stingray Barb in Forearm. This envenomation occurred after the fisherman inadvertently caught a stingray and was envenomated while taking the animal off the hook. The barb is imbedded in the patient's forearm. (Photo contributor: John Meade, MD.)



FIGURE 16.101 ■ Stingray Envenomation. Puncture wound from stingray envenomation in a lower extremity. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 16.102 ■ Stingray Barb on X-Ray. This x-ray demonstrates a retained stingray barb projecting over the forefoot and extending from the base of the 2nd metatarsal to the soft tissue between the 1st and 2nd metatarsal. The barb measured 5 cm in length. (Photo contributor: Sunny R. Patel, MD, MBA.)

SEA URCHIN ENVENOMATION

Clinical Summary

Sea urchins belong to the phylum Echinodermata and are nonaggressive, slow-moving creatures. Envenomation usually occurs after accidental contact with the organism. Long, brittle, venom-filled spines or specialized jaw-like appendages (pedicellariae) are responsible for the injury. Other echinoderms, notably the crown of thorns starfish, may also cause injury via similar mechanisms. The spines frequently break. Pedicellariae can remain attached and active for several hours. They may advance into muscle or joint spaces and cause infection or injury from the venom. The usual presentation is burning pain progressing to localized muscle aches. Erythema and edema may be present. Multiple

envenomations may produce systemic symptoms including nausea, vomiting, abdominal pain, paresthesias, numbness, paralysis, hypotension, syncope, or respiratory distress. While envenomation causes a reaction that may be quite painful, deaths, though reported, are exceedingly rare.



FIGURE 16.103 ■ Sea Urchin Spine on X-Ray. This is an x-ray of a patient who was reaching into a rock crevice in the ocean and experienced sudden sharp pain. Note the retained sea urchin spine in the fifth digit adjacent to the proximal phalanx. (Photo contributor: Marion Berg, MD.)

Management and Disposition

Following envenomation, the affected area should be submersed in hot water (43°C-46°C [109°F-115°F]) for 30 to 90 minutes. Pedicellariae may be removed by applying shaving cream and gently scraping with a razor. Obvious embedded spines should be removed. An x-ray should be performed to rule out retained foreign body. In certain cases, CT, ultrasound, or magnetic resonance imaging (MRI) may be helpful. Hand wounds often require surgical debridement. Retained spines may dissolve spontaneously, but granulomas may form, producing locally destructive inflammation. Antibiotics may be useful in certain

cases. Tetanus prophylaxis should be addressed.



FIGURE 16.104 ■ Long-Spined Sea Urchin. The majority of urchins are nonvenomous and only cause injury from puncture wounds. Their spines may break off in the victim, resulting in a high likelihood of infection. (Photo contributor: Ian D. Jones, MD.)



FIGURE 16.105 ■ Sea Urchin Envenomation. Note the multiple puncture wounds on the foot of a patient who accidentally stepped on a sea urchin in shallow water. (Photo contributor: Saralyn R. Williams, MD.)



FIGURE 16.106 ■ Fire Urchin. A beautiful fire urchin, found in the Indo-Pacific. (Photo contributor: Ian D. Jones, MD.)

Pearls

1. Sea urchin envenomation involving a joint may produce severe synovitis.
2. Some species of sea urchin contain dye, which may give the false impression of a retained spine.
3. Sea urchins known to be hazardous to humans are generally found in the Indian Ocean, Pacific Ocean, and Red Sea.

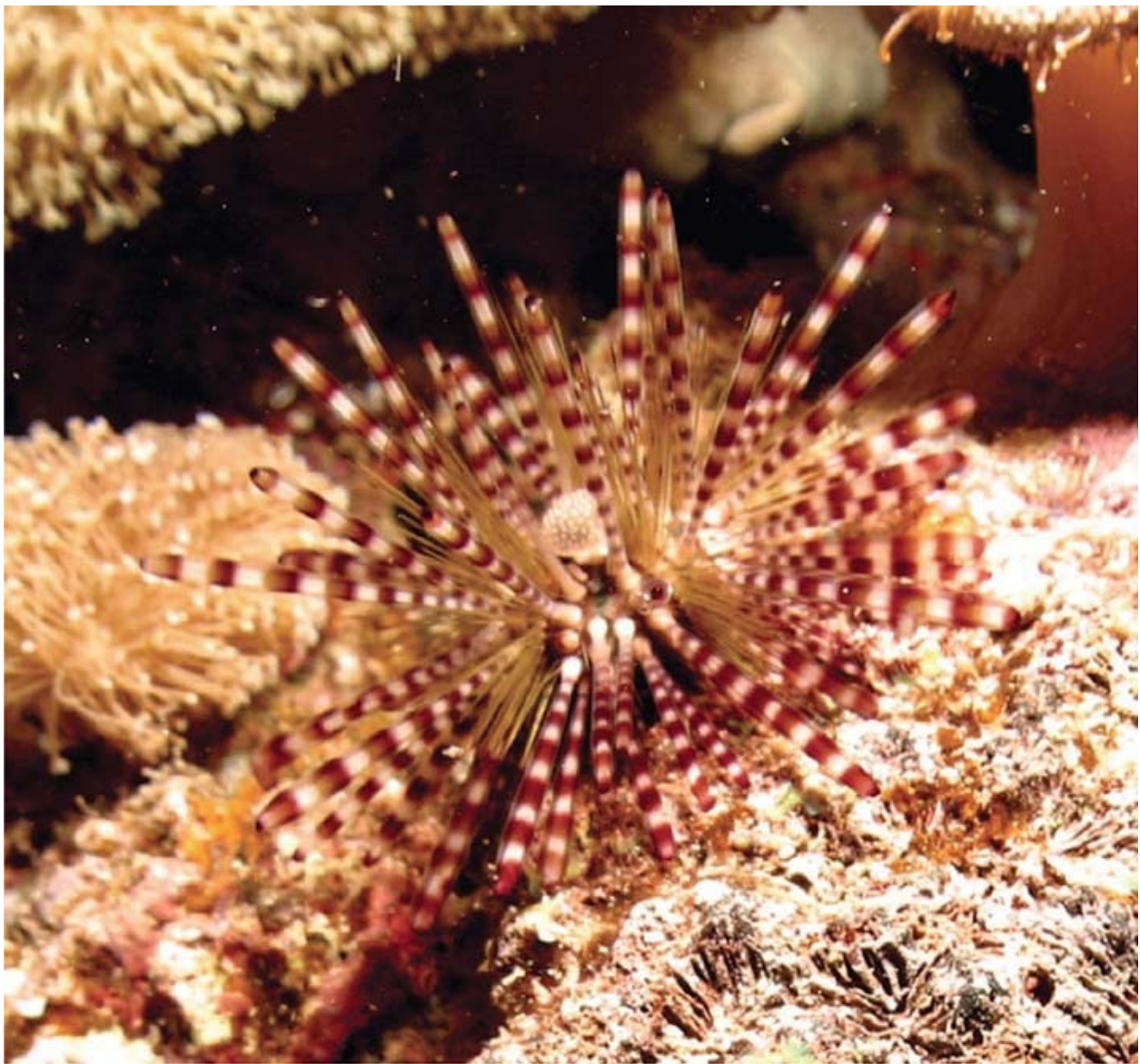


FIGURE 16.107 ■ Fire Urchin. Fire urchins are venomous sea urchins. Envenomations from fire urchins,

while extremely painful, are seldom if ever fatal. (Photo contributor: Ian D. Jones, MD.)

COELENTERATE ENVENOMATION

Clinical Summary

The phylum Coelenterata contains approximately 10,000 different species, of which several hundred are a danger to humans. This diverse group includes hydrozoans (including Portuguese man-of-war, stinging hydrozoans, and fire coral), scyphozoans (“true” jellyfish), and anthozoans (soft corals, stony corals, and anemones). They account for more marine envenomations than any other phylum. The important species involved in human injuries have stinging cells called nematocysts. Nematocysts are enclosed in venom sacs and are present in tentacles that hang from air-filled structures. After external contact, the nematocysts are discharged from their sacs, often penetrating the skin, and release their venom. Nematocyst venom is an extremely complex substance containing numerous proteins and enzymes. Clinical presentations following envenomation range from mild dermatitis to cardiovascular and pulmonary collapse. Mild envenomations usually result in a self-limited inflammatory eruption associated with burning and limited to areas of contact. Moderate to severe envenomations produce a spectrum of neurologic, cardiovascular, respiratory, and gastrointestinal symptoms. Anaphylactoid reactions—including hypotension, dysrhythmias, bronchospasm, and cardiovascular collapse—may occur, resulting in unexplained drownings.



FIGURE 16.108 ■ Stinging Hydrozoan. Similar to fire coral, contact with stinging hydrozoan results in local burning pain followed by erythematous papules or urticarial eruptions and blisters. (Photo contributor: Shawn Miller.)



FIGURE 16.109 ■ Hydrozoan Envenomation. This hydrozoan sting resulted from accidental direct contact with the diver's finger while taking an underwater photograph. (Photo contributor: Ian D. Jones, MD.)



FIGURE 16.110 ■ Fire Coral. (A) Fire coral. (Photo contributor: Shawn Miller.) (B) Fire coral envenomation. After contact, fire coral most commonly causes immediate local burning pain, followed by erythematous papules or urticarial eruptions. Pruritus may last for several days. (Photo contributor: Emily R. Stack.)



FIGURE 16.111 ■ Portuguese Man-of-War. A beautiful Portuguese man-of-war with multiple tentacles dangling in the water. The tentacles, filled with venomous nematocysts, can extend several meters in length. (Photo contributor: Adam Lavery.)

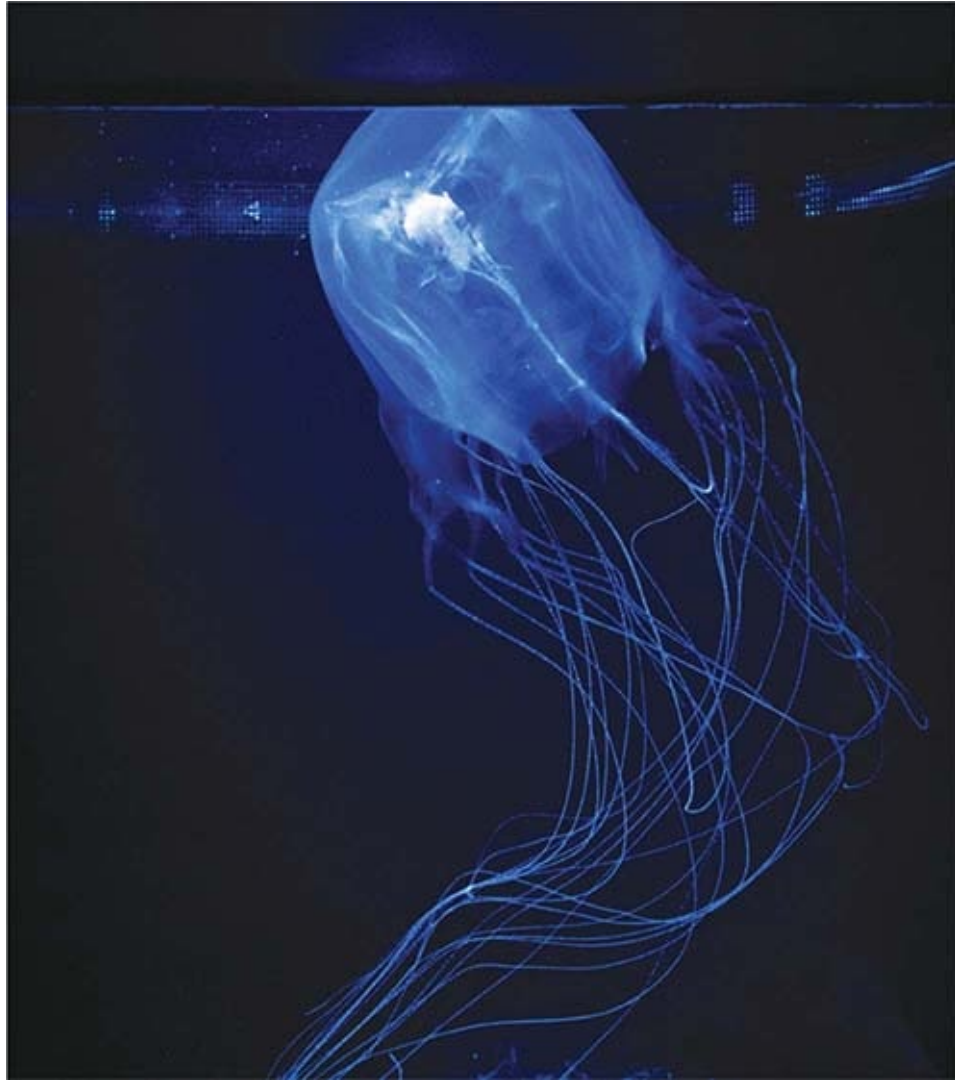


FIGURE 16.112 ■ Box Jellyfish (Under Blue Light). The box jellyfish delivers an unbearably painful sting. Envenomations can be life-threatening and require immediate treatment. (Photo contributor: Shawn Miller.)



FIGURE 16.113 ■ Coelenterate Envenomation. The sharp angulations and undulations characteristic of jellyfish envenomation. (From Halstead BH. *Venomous Marine Animals of the World*. Washington, DC: US Government Printing Office; 1965.)

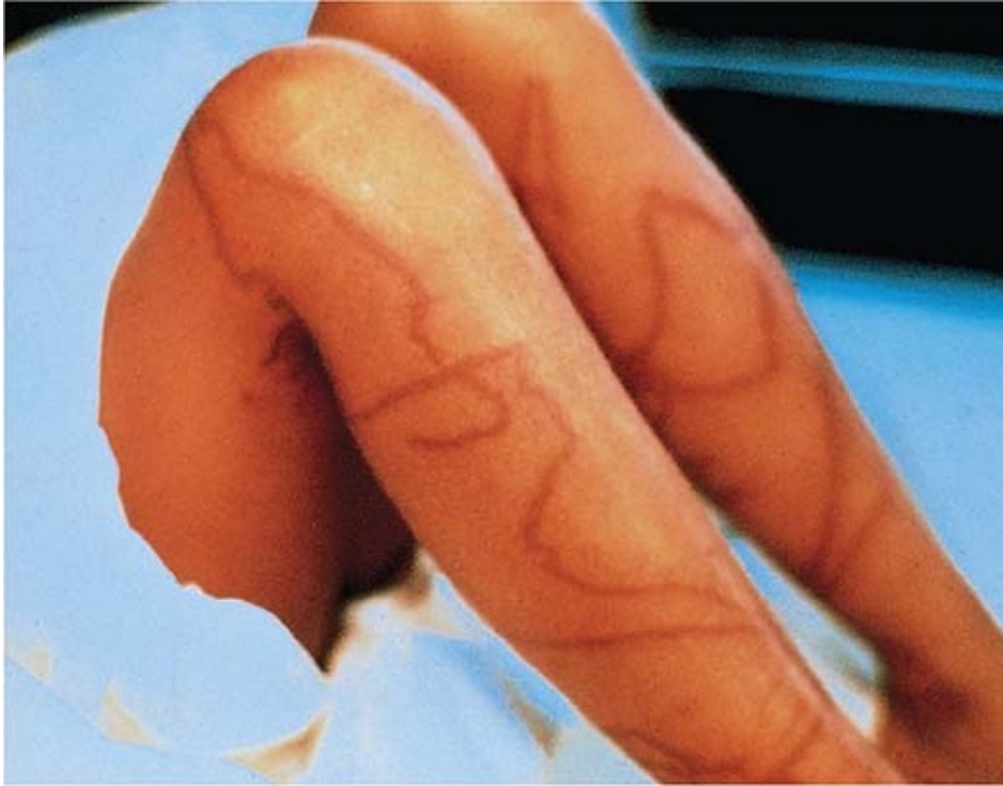


FIGURE 16.114 ■ Coelenterate Envenomation. Jellyfish envenomation on the lower extremities. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)



FIGURE 16.115 ■ Box Jellyfish Sting. This 5-year-old child was stung by a box jellyfish (*Chiropsalmus*

quadrigatus) off the coast of Japan. Severe envenomations may be fatal. (Photo contributor: Keven Reed, OD.)

Management and Disposition

Concurrently with primary resuscitation, nematocyst decontamination should be accomplished beginning with seawater flushing. Use of hypotonic fluids, such as fresh water or isopropyl alcohol, may cause additional nematocysts to fire and should be avoided. A 5% solution of acetic acid (vinegar) applied for at least 30 minutes is the most widely accepted method for inactivating nematocysts.

Tentacles can be removed with the application of shaving cream, followed in 5 minutes by a careful scraping with a firm, dull object, such as a tongue blade or credit card. Pruritus may be treated with antihistamines. Pain may be managed with immersion in hot water or with systemic analgesics. Any victim with systemic symptoms requires at least 6 to 8 hours of observation because rebound phenomena are common.

Pearls

1. The box jellyfish (*Chironex fleckeri*) is generally considered the most deadly of marine animals and is predominantly found in Australian and Southeast Asian waters.
2. The detached tentacles of some species may contain active nematocysts for months, even when fragmented on the beach or floating in water.
3. The Portuguese man-of-war is present on the Atlantic coast of Florida and in the Gulf of Mexico. It has a neurotoxin that may cause severe pain. Death, while reported, is extremely rare.



FIGURE 16.116 ■ Sea Anemone Envenomation. Contact with sea anemones results mainly in local skin irritation, initially manifesting with pruritus, burning, throbbing, and, sometimes, radiation of pain to other areas. The area involved may reveal blistering, local edema, and violaceous petechial hemorrhages. Skin lesions are confined to the areas of contact. (Photo contributor: Susan Scott.)

MARINE DERMATITIS

Clinical Summary

Marine dermatitis, also known as “sea bather’s eruption,” is a pruritic condition commonly mislabeled as *sea lice*. Symptoms usually occur a few minutes to 12 hours after exposure. The offending organisms are probably numerous and include the larval form of the thimble jellyfish and the planula form of the sea anemone, *Edwardsiella lineata*. The rash consists of erythematous wheals and papules, which may be extremely itchy. Systemic manifestations include fever, malaise, headache, conjunctivitis, and urethritis. Unlike cercarial dermatitis, marine dermatitis primarily affects areas of the body covered by caps, fins, and bathing suits.



FIGURE 16.117 ■ Marine Dermatitis. Typical appearance of marine dermatitis. (Photo contributor: Richard A. Clinchy III, PhD.)



FIGURE 16.118 ■ Cercarial Dermatitis. The discrete and highly pruritic papules of schistosome cercarial dermatitis commonly occur on exposed body areas. (Photo contributor: Edgar Maeyens, MD. Reproduced with permission from Auerbach PS, Cushing TA, Harris NS, eds. *Auerbach's Wilderness Medicine*, 7th ed. Philadelphia, PA: Elsevier; 2017. Copyright © Elsevier.)

Cercarial dermatitis, or “swimmer’s itch,” occurs when humans become accidental hosts of schistosomes that usually infect nonhuman hosts. This causes an immune response, resulting in itching, erythema, and mild edema. After 60 minutes, the classic signs are red macules that later become pruritic papules 3 to 5 cm in diameter and surrounded by erythema.

Management and Disposition

Marine dermatitis is self-limited, rarely persisting beyond 2 weeks. The dermatitis may be partially prevented by a vigorous soap-and-water scrub after saltwater bathing. Treatment is symptomatic. Calamine lotion with 1% menthol may bring relief. Topical steroids may provide additional relief. In severe cases, oral antihistamines and corticosteroids may be necessary.

Cercarial dermatitis is treated with isopropyl alcohol or calamine lotion. Severe cases may require systemic corticosteroids, while bacterial infection may require topical or oral antibiotics.

Pearls

1. Marine dermatitis primarily affects areas covered by caps, fins, and bathing suits.
2. During late spring and summer, incidence increases along the US east coast. In one reported outbreak, 25% of individuals entering the water were affected.

SCORPIONFISH STING

Clinical Summary

Scorpionfish are colorful venomous marine animals found primarily in tropical waters. Their exotic beautiful appearance has made them increasingly popular among marine aquarists in the United States. Many envenomations have resulted from mishandling. They are well camouflaged in the wild. Stings are usually

caused by accidentally stepping on them. Scorpionfish are grouped into the genera *Pterois* (lionfish), *Scorpaena* (scorpionfish proper), and *Synanceja* (stonefish), in order of increasing severity of envenomation. All have multiple spines associated with venom glands. Envenomation results from skin puncture followed by venom release. Immediately following a sting, the victim experiences intense pain that lasts for hours if untreated. The envenomation site may become warm, erythematous, and edematous, and vesicles may arise. Lionfish stings are painful but relatively mild, while systemic effects are more common with stonefish stings and may produce a constellation of cardiovascular, pulmonary, neurologic, and gastrointestinal sequelae. Death has been reported from stonefish stings.



FIGURE 16.119 ■ Lionfish (*Pterois volitans*). Envenomations occur by contact with the erectile spines on the dorsal, pelvic, and anal fins of the fish. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 16.120 ■ Scorpionfish. The scorpionfish is well-camouflaged, leading to accidental contact and envenomation in unsuspecting divers. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Hot water immersion (43°C-46°C [109°F-115°F]) for 30 to 90 minutes should be initiated as soon as possible. Rebound pain is common and can be treated with repeated hot water immersion. The wound should be inspected for pieces of spine and sheath. Thorough warm saline irrigation should be performed along

with wound exploration. Severe pain is treated with local injection of lidocaine without epinephrine and with opiate analgesia. Antibiotic prophylaxis should be considered in high-risk wounds and tetanus prophylaxis given, if indicated.

Pearls

1. Stonefish stings are the most dangerous. Severe systemic reactions may occur. Antivenin (Commonwealth Serum Labs, Australia) is available.
2. Scorpionfish venom is heat labile. Hot water immersion is effective in treating pain and inactivating venom.
3. Since 2002, Indo-Pacific lionfish have been reported in increasing numbers from New York to the Bahamas.



FIGURE 16.121 ■ Stonefish. The highly venomous stonefish is well-camouflaged. Envenomation usually results when the victim steps on the fish. (Photo contributor: Ian D. Jones, MD.)

CONE SNAIL ENVENOMATION

Clinical Summary

Cone snails, also referred to as cone shells or cone fish, are venomous predatory

marine gastropod molluscs capable of inflicting a painful, dangerous sting to humans. They may be found in wide distributions throughout oceans and seas worldwide. Cone snails prey on marine worms and fish, using their venom apparatus to inject the victim with paralytic toxins. The cone snail uses a dart-like tooth that fires out from the shell. Many have beautiful patterns on the shell, making them attractive for unsuspecting divers to collect. When disturbed, the snail may deploy its harpoon like tooth and envenomate the handler. Smaller species inflict a sting similar to that of a wasp, but envenomations from larger species may cause intense pain, swelling, paresthesias, and vomiting. Rarely, severe envenomations may progress to muscle paralysis, respiratory failure, and death.



FIGURE 16.122 ■ Cone Snail. The beautiful shells of the cone snail make it attractive for divers to collect. Envenomations commonly occur on the hand and fingers of unsuspecting victims due to handling the snail. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 16.123 ■ Geographic Cone Snail. This cone snail is seen in full view with tentacles, water siphon, and short pharynx (proboscis) visible. The geographic cone snail is highly venomous with a potentially fatal sting. (Photo contributor: Shawn Miller.)



FIGURE 16.124 ■ Cone Snail (Old). As cone snails age, the shells may become covered in adherent materials, making them difficult to see and increasing the risk of inadvertent contact. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Cone snail venom is heat-labile. Initial management consists of hot water immersion similar to the treatment of scorpionfish stings. Rebound pain is common and is treated with repeated hot water immersion. Inspect the wound for foreign material, and perform thorough warm saline irrigation. Severe pain may be treated with local injection of lidocaine without epinephrine and with opiate analgesia. Consider antibiotic prophylaxis in high-risk wounds and address tetanus status.

Pearls

1. Cone snail venom is heat-labile. Hot water immersion is effective in treating pain and inactivating venom.
2. Although fatal cone snail envenomations are rare, severe stings may cause death due to rapid onset of respiratory paralysis.

SEA SNAKE ENVENOMATION

Clinical Summary

Sea snakes are members of the elapid family of snakes that have evolved to survive in a variety of ocean habitats. There are over 70 species of sea snakes with the majority found in the tropical portions of the Indian and Pacific Oceans. Sea snakes are not found in the Atlantic Ocean or the Caribbean Sea. Most species of sea snakes live close to the coast, although there is a pelagic species, *Pelamis platurus*, that is found across a large swath of the Pacific Ocean. Reports vary regarding the docile nature of sea snakes. Some species have a much higher propensity to bite than others. Bites occur when the animal is disturbed or handled. There are many reports of bites occurring as fishermen are removing snakes from their nets. Because of the small size of the organism's fangs, bites may be inconspicuous and are often painless. The majority of bites do not result in envenomation.



FIGURE 16.125 ■ Sea Snake. The sea krait, seen here swimming free in the ocean, is very docile and rarely bites humans unless provoked. (Photo contributor: Ian D. Jones, MD.)

The most potent toxin in sea snake venoms, similar to other elapids, is a neurotoxin that competes for acetylcholine at the neuromuscular junction. Severe envenomations may ultimately lead to paralysis and respiratory failure. Additional components of the venom are myotoxic and may result in rhabdomyolysis and renal failure.



FIGURE 16.126 ■ Sea Snake. A close-up view of a sea snake found feeding on a reef. (Photo contributor: Kevin J. Knoop, MD, MS.)

The symptoms of sea snake envenomation generally occur within 30 minutes of envenomation and may be quite variable. Common symptoms include confusion, headache, myalgias, and weakness of the facial muscles, followed by an ascending flaccid paralysis and, ultimately, respiratory arrest.

Management and Disposition

Treatment for a sea snake bite includes a combination of supportive care and the immediate administration of polyvalent sea snake antivenom. Supportive care should include intubation if indicated. The patient should also be carefully observed for signs of rhabdomyolysis and hyperkalemia. The patient should be aggressively hydrated to avoid further complications such as renal failure. A toxicologist or individual experienced in managing sea snake envenomations should be consulted.

Pearls

1. An effective polyvalent antivenom exists and is effective against envenomation from all species of sea snakes.

2. In addition to respiratory support, victims of sea snake envenomation should be observed for the development of rhabdomyolysis and hyperkalemia.

CEPHALPOD ENVENOMATION

Clinical Summary

There are over 750 species of cephalopods found throughout the oceans worldwide. Cephalopods are members of the phylum mollusca and include cuttlefish, squid, octopuses, and the nautilus. Several species of cephalopods are known or thought to be venomous, but only the blue-ringed octopus, genus *Hapalochlaena*, poses a significant threat to humans.

There are four known species of blue-ringed octopus. All are small; none exceed a few inches in size. The blue-ringed octopus is found in tide pools and shallow reefs across a wide area of the Indo-Pacific ranging from Japan to Australia. The blue-ringed octopus is generally not aggressive and is relatively nondescript when left in peace. When threatened, however, the animal will flash or pulsate with striking iridescent blue rings. Bites occur when the animal is disturbed or inadvertently handled. It may be found hiding in old bottles or shells in tidal pools and in this way poses a hazard to collectors. Like other cephalopods, the blue-ringed octopus has a beak that can be powerful enough to penetrate a wet suit. Some bites may be relatively painless.



FIGURE 16.127 ■ Blue-Ringed Octopus. The beautiful but highly venomous blue-ringed octopus. (Photo contributor: Russell C. Gilbert, MD.)



FIGURE 16.128 ■ Blue-Ringed Octopus. The blue-ringed octopus demonstrates its iridescent blue colorations, seen when the animal is threatened. (Photo contributor: Kevin J. Knoop, MD, MS.)

Blue-ringed octopus venom has several identified components. The most powerful component of the venom, tetrodotoxin, is a potent sodium channel blocker that is identical to the toxin found in pufferfish. Once the victim is bitten, symptoms may develop within 10 minutes and include perioral paresthesias, facial weakness, nausea, and vomiting. In some patients, especially children, hypotension may develop. As symptoms progress, flaccid paralysis and respiratory arrest may result.

Management and Disposition

Treatment for a blue-ringed octopus bite is entirely supportive, as no antivenin currently exists. The patient almost always has a normal sensorium unless profoundly hypoxic or hypercarbic. Primary management of severe envenomation includes management of hypotension and prolonged ventilatory support until the toxin can be degraded and excreted.

Pearls

1. No antivenin exists for a blue-ringed octopus envenomation.
2. The victim's mental status is usually normal. The victim may be fully conscious despite being paralyzed and apneic.

ERYSIPELOID

Clinical Summary



FIGURE 16.129 ■ Erysipeloid Envenomation. Typical appearance of *Erysipelothrix rhusiopathiae* skin infection. (Photo contributor: Paul S. Auerbach, MD; reprinted with permission from Auerbach PS (ed). *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. 3rd ed. St. Louis, MO: Mosby-Year Book; 1995. Copyright © Elsevier.)

Erysipeloid, also known as “fish handler’s disease,” is a bacterial skin infection caused by *Erysipelothrix rhusiopathiae*. This condition is frequently seen in people who handle raw meat, fish, and shellfish. The offending organism enters the body through a break in the skin and causes a local infection within 2 to 7 days. Lesions are characterized by an edematous central purplish-red area, surrounded first by central clearing and then circumscribed by an advancing raised, erythematous ring. The area is usually pruritic and painful and may be associated with fever, malaise, and regional lymphadenopathy.

Management and Disposition

If left untreated, erysipeloid will usually resolve spontaneously in about 3 weeks. Skin infections may be treated with penicillin, a first-generation cephalosporin, or a macrolide antibiotic such as erythromycin. If severe infection occurs, it

should be treated with appropriate broad-spectrum antibiotics.

Pearls

1. A history of occupational or recreational exposure to fish or shellfish is the key to diagnosis.
2. *E rhusiopathiae* is usually resistant to aminoglycoside antibiotics. These should be avoided.

TOXICODENDRON AND OTHER PLANT EXPOSURES

Clinical Summary

Poison ivy, oak, and sumac cause more cases of allergic contact dermatitis in the United States than all other allergens combined. At least 70% of the US population is sensitive to these *Toxicodendron* species. The allergen urushiol is responsible for *Toxicodendron* dermatitis, also known as rhus dermatitis. Urushiol is found in many other plants, including in the skin of mangos. Mangos are a common cause of plant dermatitis in subtropical and tropical areas, including Hawaii.

The dermatitis begins with pruritus and erythema, usually within 2 days of exposure in susceptible persons. The degree of dermatitis depends on the patient's degree of sensitivity, the amount of allergen exposure, and the reactivity of the skin at exposed body locations. The dermatitis may range from erythema alone to papules, vesicles, and bullous eruptions. A linear distribution of cutaneous lesions is strongly suggestive of *Toxicodendron* dermatitis. This distribution occurs after plant parts have rubbed against the skin or when contaminated fingernails have scratched it.



FIGURE 16.130 ■ **Poison Ivy.** *Toxicodendron radicans* (poison ivy—shrub or climbing vine). Note that the leaves of poison ivy have three leaflets and the stems are commonly reddish orange. Poison ivy occurs throughout the United States. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 16.131 ■ **Poison Oak.** *Toxicodendron diversiloba* (poison oak). Like poison ivy, the terminal part of the branch has a cluster of three shiny leaves. It grows as a tree or woody shrub and occurs west of the Rocky Mountains. (Photo contributor: Ken Zafren, MD.)



FIGURE 16.132 ■ Poison Sumac. *Toxicodendron vernix* (poison sumac). Note that the leaves of poison sumac have 7 to 13 leaflets. It grows as a tree or woody coarse shrub. Only one species of poison sumac is found in the United States. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 16.133 ■ Poison Sumac Dermatitis. A moderately severe local reaction to poison sumac. Note the vesicles, bullae, and exudates characteristic of a contact dermatitis. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.134 ■ Poison Oak Dermatitis. Erythematous papules and vesicles. This firefighter was exposed to urushiol, the allergen of poison oak, ivy, and sumac, in smoke from burning poison oak. (Photo contributor: Ken Zafren, MD.)



FIGURE 16.135 ■ Poison Ivy. Erythematous papules and vesicles in a linear distribution consistent with *Toxicodendron* dermatitis. (Photo contributor: Alan B. Storrow, MD.)

Management and Disposition

An immediate rinse or shower with warm water and soap may minimize the reaction. If symptoms are limited to erythema and papules affecting a small surface area, calamine lotion or topical steroid sprays may provide adequate symptomatic relief. Pruritus may be decreased with oral antihistamines and oatmeal baths. Vesicles and bullae may benefit from Dome-boro compresses (60 minutes three times daily) to help dry the lesions and relieve pruritus. Systemic corticosteroids tapered over 3 weeks are used in severe reactions. Secondary infection should be treated with systemic antibiotics against staphylococcal and streptococcal species.



FIGURE 16.136 ■ Poison Ivy. A highly pruritic vesicular eruption of *Toxicodendron* dermatitis on the patient's forearm. Note the linear groupings of vesicles, likely from scratching with contaminated fingernails. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.137 ■ Mango Dermatitis. This patient was catching mangos as they dropped from a tree in Mexico. The rash progressed rapidly over a 48-hour period. This patient can eat mangos, but is sensitive to the skin of the fruit. (Photo contributor: Melissa Gray, WEMT.)

Pearls

1. Fluid from the vesicles or bullae does not contain any allergen.
2. Removal of the allergen from the skin within 30 minutes of exposure may prevent dermatitis.
3. Deliberate removal of allergen from under the fingernails may prevent spreading.
4. Treatment with systemic steroids for less than 3 weeks may result in rebound exacerbations of the dermatitis.

Clinical Summary

Sporotrichosis is a fungal skin infection caused by *Sporothrix schenckii*, an organism primarily found on plants and flowers and in soil. The problem is common among gardeners and florists. It also affects those who handle animals, since the fungus may inhabit claws. Infection occurs when contaminated thorns, spines, or claws penetrate the victim's skin. After an average incubation period of 3 weeks, localized infections become apparent. "Fixed" cutaneous infections are localized to the inoculation site and are manifest as 2- to 4-mm papules or nodules. They may ulcerate or become surrounded by raised erythema. They are typically painless. Progression to lympho-cutaneous infections occurs in about 70% of cases. Patients present with a nodule at the site of penetration, with appearance of subcutaneous nodules and skip areas along lymphatic tracks later. The lesions may wax and wane over months to years. Patients with cutaneous sporotrichosis typically lack systemic symptoms.

Management and Disposition

Sporotrichosis may be successfully treated with oral potassium iodide for 1 month after clinical manifestations have resolved. Alternative therapy includes oral itraconazole, ketoconazole, or terbinafine, whereas disseminated infections may require intravenous amphotericin B. Outpatient therapy is appropriate for nondisseminated infections. Tetanus status should be addressed.

Pearls

1. Fungal cultures and tissue biopsy cultures can be useful to confirm the diagnosis.
2. Treatment should be continued for 1 month following clinical resolution to eradicate *S schenckii*.
3. Although rare, a pulmonary form of sporotrichosis after inhalation exposure has been reported.



FIGURE 16.138 ■ Fixed Sporotrichosis. The ulcer and surrounding erythema of fixed cutaneous sporotrichosis could be confused with a brown recluse spider bite. (Photo contributor: Edward J. Otten, MD.)



FIGURE 16.139 ■ Lymphocutaneous Sporotrichosis. Lymphatic spread is common in cutaneous sporotrichosis. (Photo contributor: Kevin J. Knoop, MD, MS.)

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Chapter 17

TOXICOLOGICAL CONDITIONS

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Jimsonweed. Jimsonweed seed pod with dried seeds. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

AMPHETAMINE TOXICITY

Clinical Summary

Amphetamine toxicity may occur from abuse of prescription amphetamines, use of supplements, or use of illicit forms of the drugs. Routes of administration include ingestion, insufflation (“snorting”), parenteral injection, and smoking. Phenylethylamine is the backbone structure of amphetamines as a class, and supplements may be labeled as such. “Ice” refers to a pure preparation of methamphetamine hydrochloride in a large crystalline form. Designer amphetamines include 3,4-methylenedioxymethamphetamine (MDMA, “ecstasy”) and synthetic cathinones (ingredients in “bath salts”). While the central nervous system (CNS) targets of these compounds are serotonergic and dopaminergic pathways, the clinical presentation is typically a sympathomimetic toxidrome. The structure of the compound determines if the drug has more hallucinogenic effects.

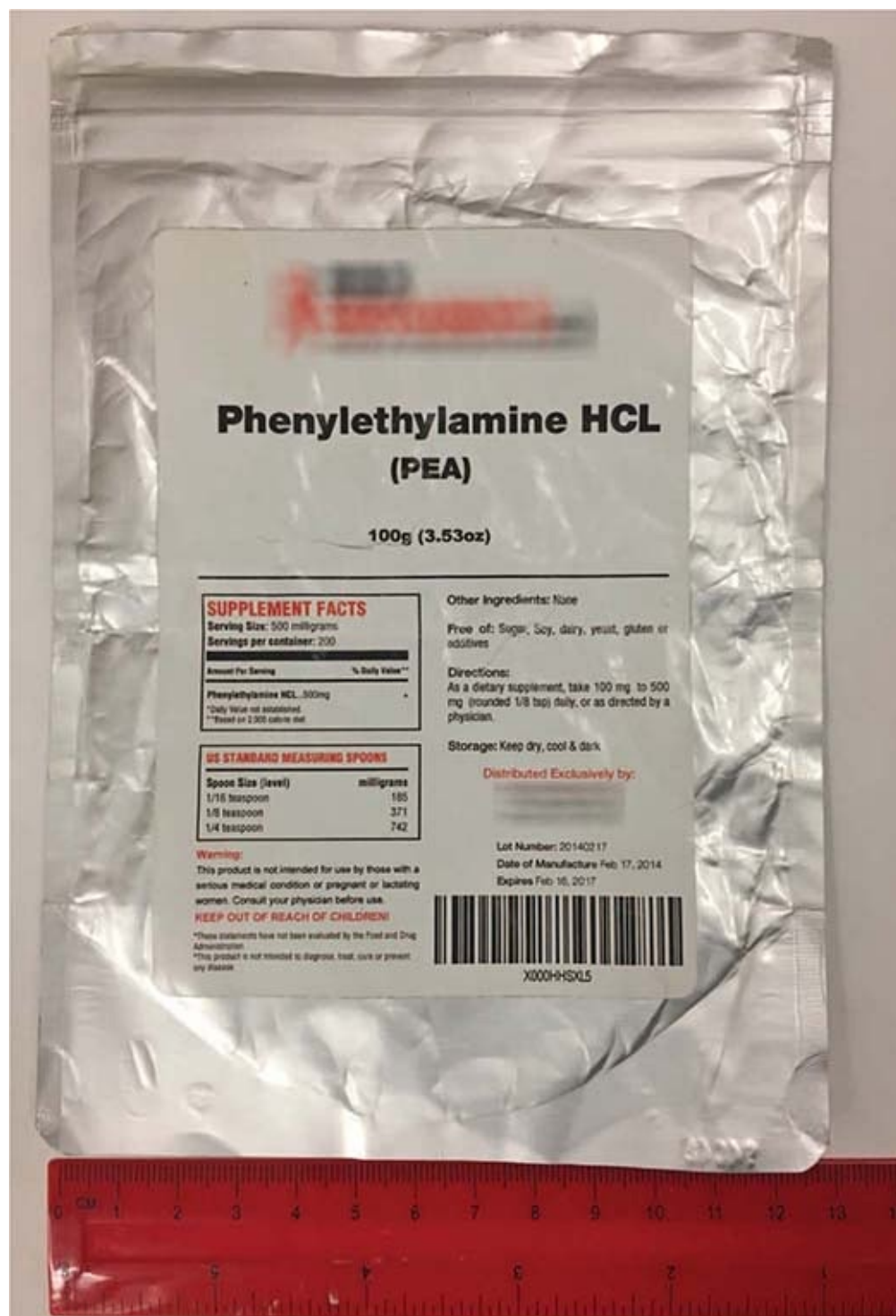


FIGURE 17.1 ■ Amphetamine Bulk Supplements. This packet was brought with a patient who exhibited a severe sympathomimetic toxidrome after ingesting the entire packet. Note the “serving” size of 500 mg with 200 “servings” per container. (Photo contributor: Saralyn R. Williams, MD.)



FIGURE 17.2 ■ “Ice” Methamphetamine. An example of the “ice” form of amphetamines with a pipe. (Photo contributor: US Drug Enforcement Administration.)



FIGURE 17.3 ■ Ecstasy. Examples of the candy-like appearance of ecstasy tablets. (Photo contributor: US Drug Enforcement Administration.)

Although clinically indistinguishable from cocaine toxicity, the duration of effects is appreciably longer. The most common cardiovascular manifestations are tachycardia and hyper-tension, although myocardial ischemia has been reported. CNS toxicity is the primary reason most amphetamine users are brought for medical care. Presentations may range from increased anxiety to life-threatening agitated delirium with hyperthermia. Visual and tactile hallucinations and psychoses are common. Poor dentition is seen among chronic methamphetamine users (“meth mouth”).



FIGURE 17.4 ■ Early “Meth Mouth.” “Meth mouth,” the extensive and accelerated dental caries associated with chronic methamphetamine abuse. (Photo contributor: R. Jason Thurman, MD.)

Management and Disposition

Treatment focuses on the signs and symptoms of toxicity. As with other causes of sympathomimetic toxicity, initial management includes control of the agitation to prevent other complications (eg, rhabdomyolysis). Benzodiazepines are the first-line therapy for agitation; large repeated doses may be required. Severe hyperthermia requires evaporative cooling techniques. Hypertonic sodium may be useful for MDMA-associated cerebral edema and seizures.

Pearls

1. In addition to the medical complications associated with chronic methamphetamine use, the manufacture of illicit methamphetamine is associated with exposure to toxic chemicals and risk for severe burns.



FIGURE 17.5 ■ Advanced “Meth Mouth.” Note the severe dental decay in this chronic methamphetamine abuser. (Photo contributor: Carson Harris, MD.)

2. The hyperthermia associated with acute amphetamine poisoning may result in end-organ damage similar to patients with heatstroke-like illness.
3. MDMA may result in syndrome of inappropriate antidiuretic hormone (SIADH) with subsequent hyponatremia and cerebral edema.

DESIGNER DRUGS: “BATH SALTS” AND “SPICE”

Clinical Summary

The cathinones are derivatives of naturally occurring phenylethylamines found in the leaves of the *Catha edulis* (Khat) plant. Some of these compounds include mephedrone, methylone, and methylenedioxypropylamphetamine (MDPV). Example names on the packaging include “Molly’s plant food,” “bath salts,” and “flakka.” Adverse effects of these compounds include cardiac, psychiatric, and neurologic signs and symptoms similar to effects seen with other sympathomimetics.

Synthetic cannabinoids have been promoted as “spice” or “incense” products. These compounds have full agonist effects at the cannabinoid receptors, in contrast with tetrahydrocannabinol (THC), which demonstrates only partial agonism. While cannabinoids such as marijuana do not typically result in sympathomimetic effects, the synthetic cannabinoids may also cause acute sympathomimetic toxicity including seizures and tachydysrhythmias. Myocardial infarction, stroke, and acute kidney injury have all been reported with the use of synthetic cannabinoids.



FIGURE 17.6 ■ Khat plant. The leaves of the Khat plant (*Catha edulis*) are chewed for the stimulant effects. The leaves must be fresh in order for cathinone to be present. (Photo contributor: US Drug Enforcement Administration.)



FIGURE 17.7 ■ Bath Salts. Examples of typical packaging for bath salts. (Photo contributor: US Drug Enforcement Administration.)



FIGURE 17.8 ■ Synthetic Cannabinoids—“Spice.” Examples of the packaging of synthetic cannabinoids, which are labeled “not for human consumption.” (Photo contributor: John G. Benitez, MD, MPH.)

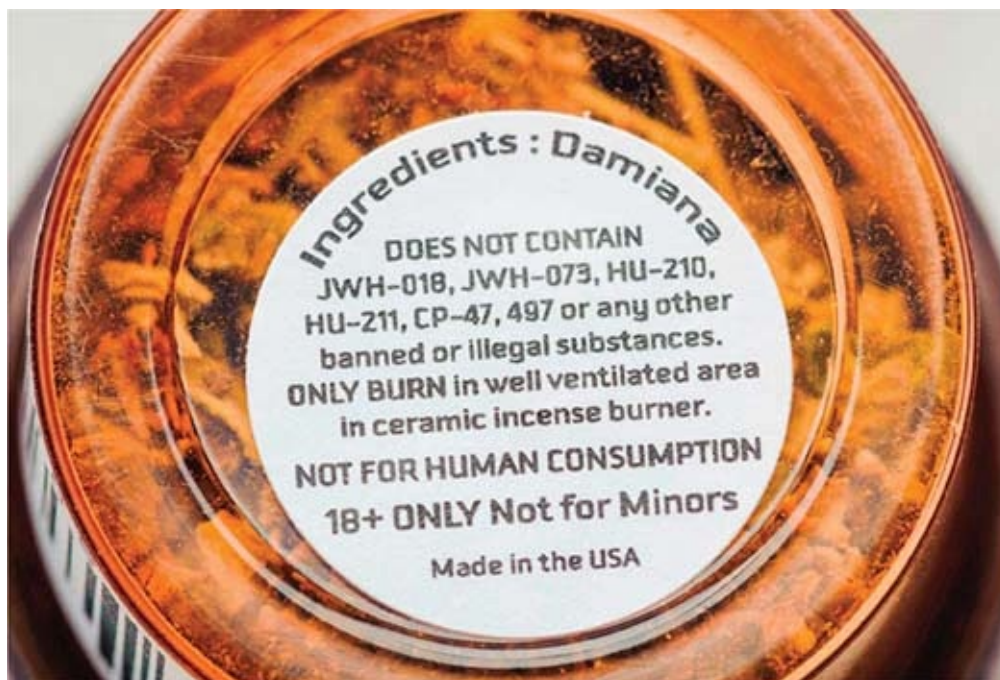


FIGURE 17.9 ■ Synthetic Cannabinoids Disclaimer. Close-up of the language used to suggest that the contents do not contain illegal substances. (Photo contributor: John G. Benitez, MD, MPH.)

Forensic analysis of these products has demonstrated significant variability in types and amounts of active products over time, both between and within marketed brands. As a result, individuals may experience different clinical effects with different exposures to the same product.

Management and Disposition

Patients under the influence of one of the synthetic compounds should be approached and managed similar to other acute sympathomimetic poisonings.

Pearls

1. The ingredients in “bath salts” are not detected by routine testing for amphetamines as a class.
2. Vaping has become a popular method for abuse of “K2/spice” as liquid formulations become available.
3. Adulterants such as long-acting anticoagulants (vitamin K inhibitors) have been found in synthetic cannabinoids.

COCAINE TOXICITY

Clinical Summary

Cocaine is a natural alkaloid derived from the leaves of *Erythroxylum coca*. Cocaine hydrochloride (powder cocaine) is a crystalline white powder. “Crack,” the free-base of cocaine hydrochloride, is an off-white substance named both for its rock-like appearance (“rock”) and the sound it makes when heated. “Crack” may be smoked as it vaporizes when heated. Inhalation of the vapor results in rapid absorption and distribution of the cocaine to the brain.

Cocaine intoxication manifests as a sympathomimetic toxidrome, with tachycardia, hypertension, diaphoresis, mydriasis, delirium, and hyperthermia. Increased muscular activity may result in rhabdomyolysis. Numerous neurologic complications have been reported after cocaine use, including subarachnoid hemorrhage, intracerebral hemorrhage, cerebral infarction, and seizures.



FIGURE 17.10 ■ Cocaine Powder. Cocaine powder. (Photo contributor: US Drug Enforcement Administration.)



FIGURE 17.11 ■ Drug Paraphernalia. Crack pipe sequestered in the rectum during a patient's arrest, resulting in laceration of the hemorrhoidal venous plexus and massive hemorrhage. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

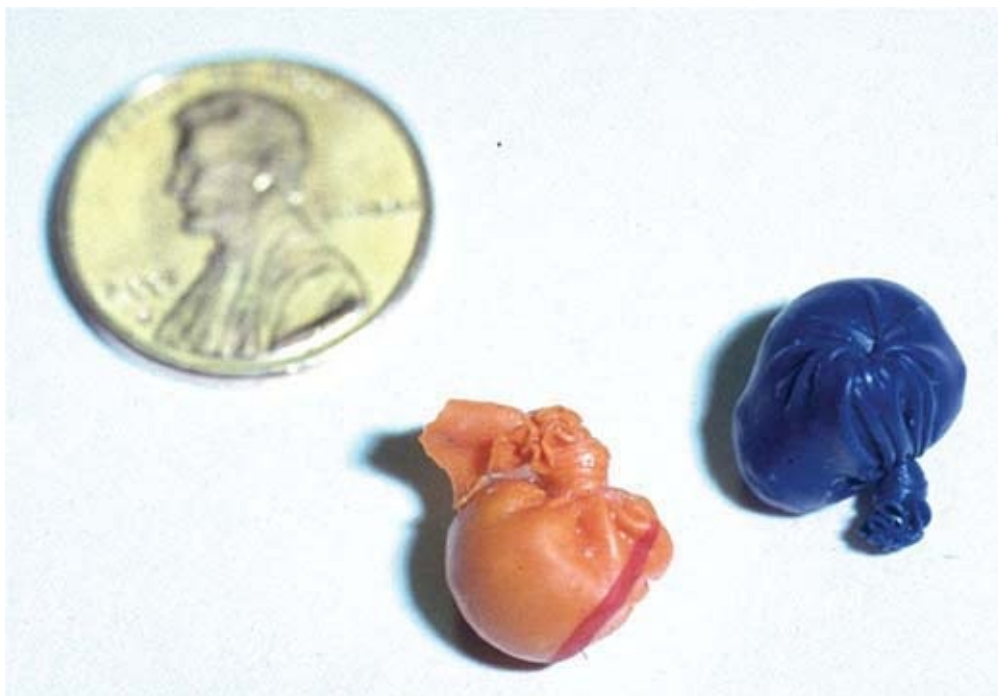


FIGURE 17.12 ■ Cocaine Body-Packing. Cocaine-filled balloon packets from the stool of a cocaine "body-packer" (penny used for scale). Radiopaque packets are often visible on KUB radiograph. Severe toxicity may result in the event of a ruptured packet. (Photo contributor: Alan B. Storrow, MD.)

Cardiovascular toxicity, including acute myocardial infarction, is well described after cocaine use. Dysrhythmias, including supraventricular tachycardia, atrial fibrillation and flutter, ventricular tachycardia, ventricular

fibrillation, and torsades de pointes, have been reported. Cocaine is a sodium channel blocker and may cause QRS widening on the electrocardiogram (ECG). Aortic dissection and rupture have been associated with cocaine use.

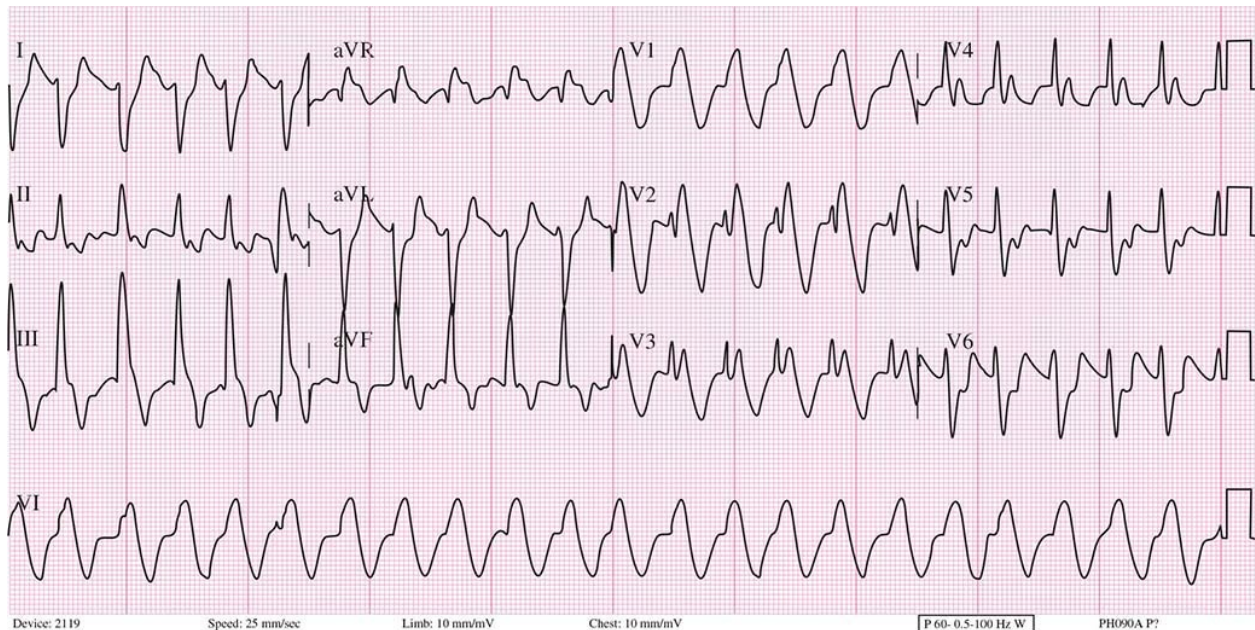


FIGURE 17.13 ■ Cocaine Cardiotoxicity. The initial 12-lead ECG of a patient with acute cocaine and cocaethylene poisoning demonstrating with wide complex rhythm from the sodium channel blocking effects. Note the profound terminal R wave changes in the lead aVR. The initial serum pH was 6.8. (Photo contributors: Thomas Babcock, MD, and Laurie Lawrence, MD.)

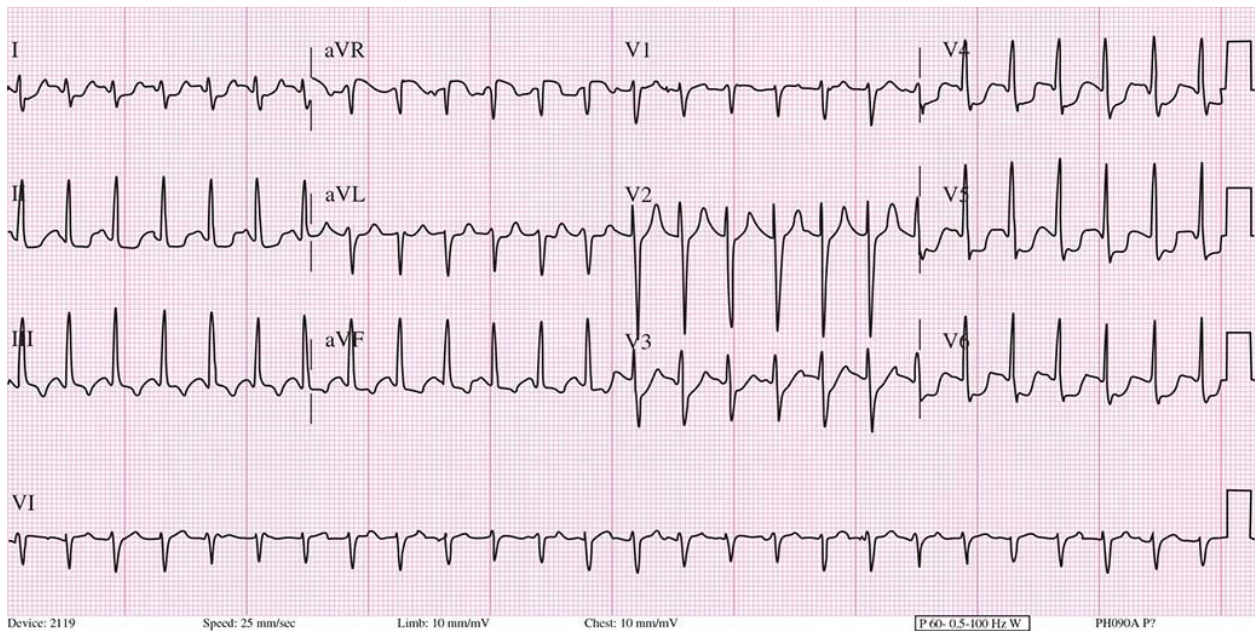


FIGURE 17.14 ■ Treated Cocaine Cardiotoxicity. The 12-lead ECG of the same patient in Fig. 17.13, 68 minutes after aggressive treatment with sodium bicarbonate to a serum pH of 7.26. (Photo contributors: Thomas Babcock, MD, and Laurie Lawrence, MD.)

Pulmonary complications include pneumothorax, pneumomediastinum, and cardiogenic and noncardiogenic pulmonary edema (NCPE). “Crack lung” refers to an acute pulmonary syndrome of dyspnea, hypoxia, and diffuse pulmonary alveolar infiltrates.

Management and Disposition

Treatment is primarily supportive and focuses on the signs and symptoms of toxicity. Cardiac monitoring is indicated for symptomatic patients. Initial management focuses on control of agitation, reduction of the hyperthermia, and prevention of complications (eg, rhabdomyolysis). Benzodiazepines are the first line of therapy for agitation and are beneficial for acute cocaine-associated chest pain. Neuroleptic agents are used with caution for cocaine-associated psychomotor agitation due to the negative effects of these agents on thermoregulation, seizure threshold, and the potential for dysrhythmias. Sodium bicarbonate administration should be considered for QRS widening in the setting of acute cocaine poisoning.

Pearls

1. The use of β -blockers for the management of an acute cocaine sympathomimetic toxidrome may result in worsening vaso-spasm and hypertensive crisis (“unopposed α -effect”).
2. Cocaine may induce coronary spasm, resulting in acute myocardial ischemia. Chronic cocaine use also accelerates atherosclerotic disease.
3. Urine drug screens usually check for the cocaine metabolite benzoylecgonine, which may be detectable in the urine for up to 3 days after cocaine exposure.
4. The rupture of a cocaine packet in a body-packer may result in fatal toxicity. Emergent surgical intervention may be considered for immediate removal of the packets.
5. Levamisole is an adulterant that is sometimes found in cocaine. Levamisole is an anthelmintic that can cause severe agranulocytosis and/or vasculitis.



FIGURE 17.15 ■ Cocaine-Induced Rhabdomyolysis. Rhabdomyolysis is a common clinical finding in patients with severe cocaine poisoning. Myoglobinuria may occur. (Photo contributor: Mohamud Daya, MD.)

LEVAMISOLE-INDUCED VASCULITIS

Clinical Summary

Levamisole was first used as an anthelmintic for human and veterinary use. Initial human use in the 1970s for inflammatory conditions resulted in publications of levamisole-associated agranulocytosis and vasculitis. In 2003, the US Drug Enforcement Agency identified levamisole as an adulterant in cocaine. In 2009, case reports of agranulocytosis and vasculitis associated with levamisole-contaminated cocaine were published. The dermatologic manifestations may include retiform purpura with possible skin necrosis and tend to appear on the ears and nose but can affect any area.

Cases have been reported from both cocaine hydrochloride and crack cocaine and all routes of administration. The reason for adulterating the cocaine with levamisole is not clear. Theories include that the levamisole enhances the effects of cocaine via one of several potential mechanisms: enhancing noradrenergic neurotransmission, inhibiting monoamine oxidase, inhibiting acetylcholinesterase, and stimulating ganglionic nicotinic receptors.



FIGURE 17.16 ■ Levamisole-Induced Vasculitis (Ear). The vasculitis associated with levamisole has a predilection for involving the ear. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Initial considerations for the differential diagnosis for agranulocytosis or the vasculopathy should be broad. The xenobiotic exposure history for the patient must also be carefully reviewed, inclusive of drugs of abuse. For both the agranulocytosis and the cutaneous vasculopathy, serologic testing assists with the differential diagnosis; however, no one classic pattern is diagnostic for levamisole as the etiologic agent.

In the setting of agranulocytosis, neutropenic fever may occur and should be managed accordingly with antibiotics. Deaths from infectious complications have occurred. Cessation of exposure to the levamisole is imperative—which means cessation of cocaine use.

Pearls

1. Case reports of levamisole-associated complications suggest that there is a high level of recurrence of complications upon reexposure to levamisole-contaminated cocaine.
2. Based on case reports, the ears and nose tend to manifest the skin necrosis more often than other areas.
3. Adulterants such as levamisole are not a part of the routine drug screens. Detection for levamisole requires additional techniques such as gas chromatography–mass spectrometry, liquid chromatography, or tandem mass spectrometry.



FIGURE 17.17 ■ Levamisole-Induced Vasculitis (Nose). Levamisole-induced vasculitis can induce significant tissue necrosis requiring reconstructive grafts. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.18 ■ Levamisole-Induced Vasculitis. Typical appearance of the purpuric lesions of levamisole-induced vasculitis. These lesions were acute and involved the leg of the patient in [Fig. 17.17](#) who also had necrotic lesions from a previous case of levamisole-induced vasculitis. (Photo contributor: R. Jason Thurman, MD.)

ANTICHOLINERGIC (ANTIMUSCARINIC) TOXIDROME

Clinical Summary

The anticholinergic toxidrome is best illustrated by the mnemonic: hot as a hare, blind as a bat, mad as a hatter, red as a beet, and dry as a bone. As the etiology reflects central and peripheral muscarinic receptor blockade, it is more accurately termed an antimuscarinic toxidrome. A centrally mediated delirium may occur, which is typically not violent but is associated with mumbling speech and persistent “picking” behaviors. Other manifestations include hyperthermia, mydriasis, dry mucus membranes and axillae, tachycardia, decreased

gastrointestinal motility, erythematous skin, and urinary retention.

Many xenobiotics are antimuscarinic. One of the more common is diphenhydramine. Tricyclic antidepressants, phenothiazines, cyclobenzaprine, carbamazepine, atropine, scopolamine, glycopyrrolate, and belladonna alkaloids all have antimuscarinic properties. Plants such as jimson weed contain belladonna alkaloids and may be used recreationally.

Management and Disposition

Initial assessments of the vital signs and the duration of the QRS on ECG are important. Since many antimuscarinic xenobiotics are also sodium channel blockers, QRS interval should be monitored. Hyperthermia occasionally occurs and is treated with evaporative cooling. Most of these patients require only supportive care, with the administration of benzodiazepines for agitation. A Foley catheter may be needed for treatment of the urinary retention. Occasionally, physostigmine is used as a diagnostic reversal agent for antimuscarinic poisoning, but its risks versus benefits must be considered. The half-life of physostigmine is only about 20 minutes.



FIGURE 17.19 ■ Anticholinergic Mydriasis. Mydriasis and flushing are some of the characteristic findings of anticholinergic toxidrome. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

Pearls

1. The antihistamine diphenhydramine has sodium channel– blocking properties and may cause QRS widening.
2. Physostigmine inhibits acetylcholinesterase, resulting in increased acetylcholine at the muscarinic synapses. Physostigmine crosses the blood-brain barrier, so it improves the antimuscarinic delirium; however, the improvement may be delayed by a few minutes after its administration given the mechanism of action.
3. Since the anticholinergic toxidrome may mimic the sympathomimetic toxidrome, the best physical examination finding to distinguish the two is presence or absence of sweat. Antimuscarinic patients are “dry as a bone.”



FIGURE 17.20 ■ Anticholinergic Delirium. Anticholinergic delirium is manifested by agitation, confusion, and a “picking” behavior. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

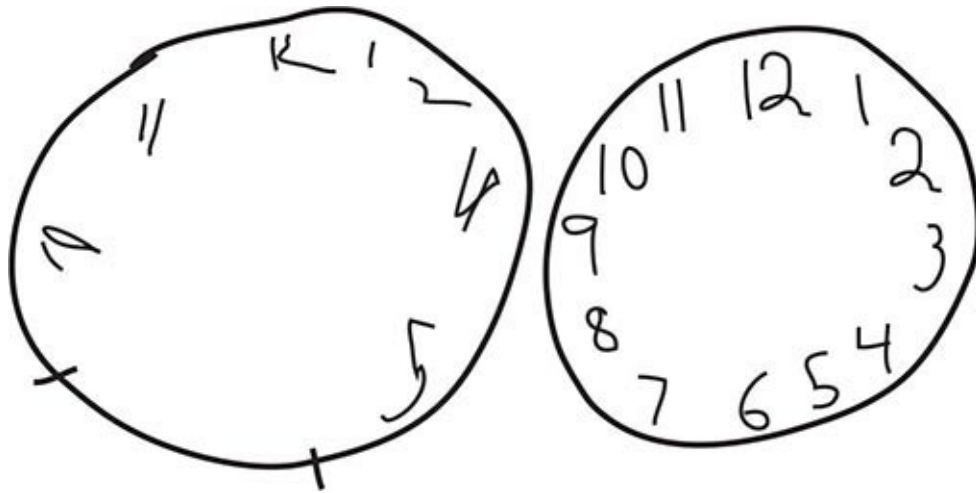


FIGURE 17.21 ■ Anticholinergic Delirium. Prior to treatment with physostigmine, a patient suffering acute anticholinergic delirium drew the clock on the left. Following physostigmine administration, the patient drew the clock on the right. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)

CHOLINERGIC TOXIDROME

Clinical Summary

Acetylcholine (ACh) is a neurotransmitter of both the central and peripheral nervous system. It is the neurotransmitter for both muscarinic and nicotinic receptors. Inhibition of acetylcholinesterase increases ACh in the synapse. This results in the clinical syndrome of both muscarinic and nicotinic effects. One mnemonic for the muscarinic effects is DUMBBEL(L)S: diarrhea, urination, miosis, bronchorrhea, bradycardia, emesis, lacrimation, and salivation. Nicotinic effects include the fasciculations, paralysis, and occasional sympathomimetic effects of mydriasis and tachycardia.

The most common cholinergic poisoning occurs after exposure to anticholinesterase insecticides. These include organic phosphorus (OP) compounds and carbamates. OP compounds bind to acetylcholinesterase, and after a period of time, this bond “ages” and becomes permanent. Carbamates are reversible binders of acetylcholinesterase and tend not to cross the blood-brain barrier.

Management and Disposition

The initial management of patients with acute cholinergic poisoning includes decontamination of their clothing and skin. The airway should be secured early in the resuscitation because most patients have difficulty oxygenating and ventilating due to bronchorrhea and the muscle weakness; however, succinylcholine should be avoided as a paralytic agent because succinylcholine requires acetylcholinesterase for its metabolism. Atropine is the initial therapeutic agent, and dosing is doubled until the pulmonary secretions are dried. Pralidoxime is administered as early as possible for patients with acute OP poisoning but would not be of benefit to patients who have known carbamate poisoning. Seizures should be aggressively treated with benzodiazepines.

Pearls

1. Atropine is the first-line agent to dry the secretions from cholinergic poisoning. Glycopyrrolate may be used if there is a limited amount of atropine available; however, glycopyrrolate does not cross the blood-brain barrier and thus would not treat the central cholinergic effects.



FIGURE 17.22 ■ Cholinergic Miosis. Significant miosis seen in a patient with acute cholinergic poisoning from a pesticide. (Photo contributor: Shannon Langston, MD.)



FIGURE 17.23 ■ Cholinergic Toxidrome. This patient is manifesting profound muscarinic effects from acute cholinergic poisoning and has intractable vomiting and diarrhea. (Photo contributor: Shannon Langston, MD.)

2. The nicotinic clinical findings can be remembered by the days of the week: MTWtHF: mydriasis, tachycardia, weakness, hypertension, and fasciculations.
3. Nerve agents such as sarin, tabun, and soman are OP compounds and are readily absorbed through inhalational routes.
4. The pharmaceutical agents physostigmine, neostigmine, and pyridostigmine are carbamates; only physostigmine crosses the blood-brain barrier.



FIGURE 17.24 ■ Atropine Therapy. This picture demonstrates the use of multiple vials of atropine required to dry the secretions of a patient with acute cholinergic poisoning. (Photo contributor: Shannon Langston, MD.)

OPIOID TOXICITY

Clinical Summary

The term *opioid* refers to all compounds with opium-like activity. Heroin is made from morphine and is usually sold as a white powder; however, the color may vary to dark brown. “Black tar” heroin may be sticky like roofing tar or hard like coal and is usually dark brown to black in color. Diversion and abuse of opioids is a common practice that is resulting in an increased number of opioid deaths. Opioid-dependent patients may use alternative supplements like kratom to reduce opioid withdrawal symptoms.



FIGURE 17.25 ■ Asian Heroin. Asian heroin tends to be available in a powder form. (Photo contributor: US Drug Enforcement Administration.)



FIGURE 17.26 ■ Black Tar Heroin. Black tar heroin has a different appearance and texture than the South American and Asian heroin. Because it has a “gummier” texture, it is usually injected or smoked. Black tar heroin is associated with wound botulism. (Photo contributor: US Drug Enforcement Administration.)

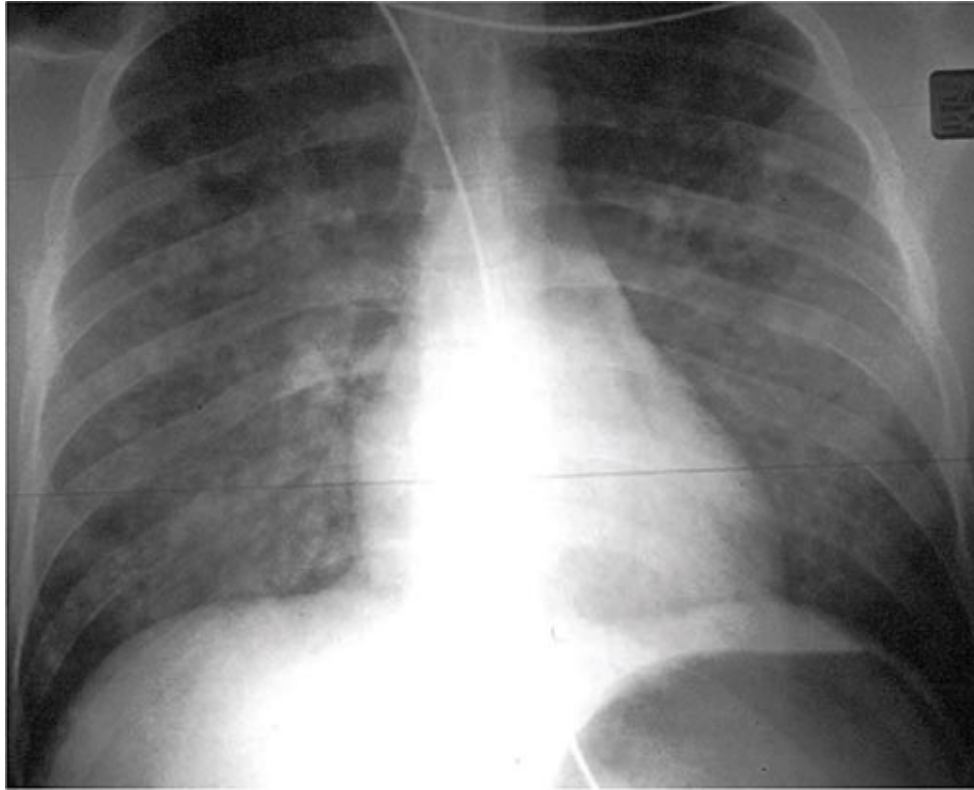


FIGURE 17.27 ■ Heroin-Related Noncardiogenic Pulmonary Edema. Noncardiogenic pulmonary edema may occur in the setting of opioid poisoning. The radiograph demonstrates the bilateral airspace opacities and the normal-sized cardiac silhouette. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)



FIGURE 17.28 ■ Opioid-Induced Noncardiogenic Pulmonary Edema. The endotracheal tube demonstrates the frothy expectorant in a patient with opioid-induced noncardiogenic pulmonary edema. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

The classic opioid toxidrome is a clinical triad of coma, respiratory depression, and miosis. However, opioid-related CNS depression can range from mild sedation to coma. Normal or dilated pupils may occur after overdose of meperidine or pentazocine, or in the setting of CNS hypoxia. Death is typically due to respiratory depression. Noncardiogenic pulmonary edema (NCPE) is associated with the use of certain opioids, particularly heroin, methadone, and morphine.



FIGURE 17.29 ■ Heroin Body-Packing. KUB radiograph of a “packer” demonstrating the presence of radiopaque foreign bodies. Rupture of a packet may result in severe opioid toxicity. (Photo contributor: Jason Chu, MD.)



FIGURE 17.30 ■ Piloerection. Piloerection may be noted with acute opioid withdrawal. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)



FIGURE 17.31 ■ Opioid Mucosal Necrosis. In this patient, significant necrosis of the soft palate is seen. The necrosis occurred secondary to repeated opioid abuse by snorting crushed oxycodone tablets. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.32 ■ Kratom. Kratom is derived from a plant that contains alkaloids that have activity at the mu opioid receptor, α_2 -receptor, and serotonergic receptors. Kratom has some opioid-like effects and stimulant effects, depending on the dose. It is promoted as a “natural” remedy for opioid withdrawal. (Photo contributor: Saralyn R. Williams, MD.)

Management and Disposition

Care of these patients focuses on airway management and antidotal therapy. Whole-bowel irrigation has been advocated after ingestion of sustained-release formulations or in the setting of body-packing and body-stuffing. In the latter, abdominal x-rays are indicated to look for evidence of foreign bodies. Chest radiographs are indicated for signs and symptoms of NCPE. Rhabdomyolysis and cerebral hypoxia may occur after prolonged periods of respiratory and CNS depression. Naloxone is the antidote of choice for significant opioid toxicity. In

administering naloxone, care should be taken not to precipitate acute opioid withdrawal. Continuous infusion of naloxone is indicated with longer acting opioids to avoid recurrence of respiratory depression.

Pearls

1. The presence of adulterants such as scopolamine or clenbuterol may mask or alter the appearance of the classic opioid toxidrome and may result in more significant toxicity than the primary drug.
2. Recurrent toxicity and life-threatening respiratory depression may occur following short-term reversal with naloxone administration, especially in body-stuffers or after ingestion of sustained-release formulations.
3. The use of naloxone in the setting of tramadol toxicity is relatively contraindicated due to the occurrence of seizures.
4. The use of black tar heroin has been associated with wound botulism.
5. Poisoning from α_2 -agonists such as clonidine mimics acute opioid poisoning.

DESOMORPHINE (KROKODIL)

Clinical Summary

Desomorphine, also known by its street name krokodil, is a powerful highly addictive synthetic opioid commonly made from codeine. The drug features a very rapid onset of action and is about 10 times as powerful as morphine. The medical use of this drug was terminated in 1981 due to its short duration and significant respiratory depression compared to other opioids, but recreational use has surged in the past decade. Due to the over-the-counter availability of codeine products in Eastern Europe and Russia, desomorphine may be easily manufactured through “krokodil labs” in a similar fashion to methamphetamine production. The drug is heated and usually intravenously injected by the user. Severe skin necrosis is caused by many contaminants, such as iodine and phosphorus from matchstick heads used in the production process. These contaminants are caustic, causing skin and soft-tissue breakdown as well as damage to blood vessels, muscles, bones, and organs. Lesions may become severe, causing deep space infections and gangrene, and may even result in the need for amputations. Desomorphine’s street name “krokodil” is thought to be

derived from the similarity of damaged skin to crocodile leather.

Management and Disposition

Management is generally supportive. Acute overdose is managed in the same fashion as other opioid toxicities, with particular attention to the patient's respiratory status. Infectious complications can be severe and may require long-term antibiotic therapy. Local skin necrosis may be self-limiting if minor, but severe necrosis of the tissues may result in the need for surgical management and even amputation in some cases.



FIGURE 17.33 ■ Desomorphine-Related Abscesses. Multiple abscesses and multiple necrotic eschars are seen on this patient's bilateral lower extremities. The patient had injected desomorphine into his legs 2 weeks prior to the appearance of the lesions. (Photo contributor: Brandon O'Keefe, MD.)



FIGURE 17.34 ■ Desomorphine-Related Abscesses. Closer view of the lesions seen in [Fig. 17.33](#). (Photo contributor: Brandon O’Keefe, MD.)

Pearls

1. The greater potency of desomorphine coupled with its short duration of action contributes to the highly addictive nature of the drug.
2. The severe tissue necrosis seen with desomorphine injection results from contaminants introduced in the “cooking” process. These adulterants are frequently not fully removed in production and are highly caustic to the tissues.

ACETAMINOPHEN POISONING

Clinical Summary

Acetaminophen is a widely available analgesic and antipyretic agent. It is commonly found in combination with opioids, decongestants, antihistamines, and other over-the-counter and prescription products. Patients may complain of nausea and vomiting shortly after a toxic ingestion, but patients may also be asymptomatic. Signs and symptoms of acute liver injury typically occur within 36 hours after acute ingestion. Occasionally, patients present to the emergency department after developing evidence of hepatotoxicity, not realizing that the large ingestion of an acetaminophen-based product is the etiology.

In the overdose setting, acetaminophen exerts its toxic effects via a metabolite that is created via the P450 enzyme system. The metabolite causes centrilobular necrosis of the liver, which may lead to fulminant hepatic failure. Renal failure may also occur. Fatalities from hepatic failure usually occur 3 to 5 days after the ingestion. Treatment includes the administration of *N*-acetylcysteine (NAC), which can prevent acetaminophen-induced hepatotoxicity if initiated within 8 hours of the acute ingestion.

Management and Disposition

Activated charcoal may be considered in patients who present within 2 hours of acetaminophen overdose. A serum acetaminophen level ($\mu\text{g/mL}$) drawn at 4 hours after a single acute ingestion can be plotted on the Rumack-Matthew nomogram to determine the need for treatment. If the serum level is at or above the treatment line, the patient should be treated with a standard course of oral or intravenously administered NAC. Patients who require administration of NAC should be admitted to the hospital.

Pearls

1. Acetaminophen is a common agent in many over-the-counter medications. Patients who overdose on these medications require routine checking of a serum acetaminophen level to identify those who may need treatment with NAC.



FIGURE 17.35 ■ Acetaminophen Overdose. Multiple acetaminophen-containing pills are seen in the vomit of an overdose patient. The patient had ingested the pills a few hours prior to presentation in a suicide attempt. (Photo contributor: Alan B. Storrow, MD.)

2. The formulation of oral NAC is available in a 20% solution. The 20% solution comprises 20 g of NAC per 100 mL of solution. For the average 70-kg adult, the initial oral loading dose of 140 mg/kg would be 9.8 g, or approximately 50 mL of the 20% solution.
3. To enhance palatability, oral NAC can be diluted into a beverage of choice and served in a cup with a lid and a straw.
4. Massive ingestions of acetaminophen may result in an anion gap metabolic acidosis.



FIGURE 17.36 ■ Acute Hepatotoxicity. This patient developed acute hepatic failure with marked jaundice as a result of an intentional acetaminophen overdose. (Photo contributor: R. Jason Thurman, MD.)

SALICYLATE POISONING

Clinical Summary

Salicylates are a common cause of analgesic poisoning. Acute ingestions of large quantities of aspirin may have delayed absorption due to the formulation of the drug or the formation of bezoars. Poisoning may occur with chronic ingestions as well, particularly in older patients.

Early effects after ingestion include gastrointestinal (GI) irritation, which may lead to nausea and vomiting. Classically, salicylate-poisoned patients present with a mixed acid-base picture. Central stimulation of the respiratory drive results in a primary respiratory alkalosis. As a result of disrupted energy mechanics and decreased adenosine triphosphate (ATP) production, metabolic acidosis and lactate accumulation occur. The initial pH of the patient's serum may be acidemic or alkalemic depending on the predominant acid-base disorder at the time of blood sampling. Ketonuria may also be noted. Hyper-thermia occurs due to the generation and release of heat secondary to uncoupling of oxidative phosphorylation. Coma and seizures indicate severe nervous system

toxicity and are associated with poor outcomes. Increased capillary permeability may result in NCPE and cerebral edema.

Management and Disposition

Fluid resuscitation to replace volume depletion is paramount early in the presentation. Since salicylate is a weak acid, alkalinizing the serum to a pH between 7.45 and 7.55 traps the salicylate in an ionized form, decreasing entry into the CNS. Similarly, urinary alkalinization prevents tubular reabsorption, thereby enhancing the renal elimination of the salicylic acid. Because of the underlying metabolic acidosis and bicarbonate-induced hypokalemia, potassium replacement is usually needed in order to alkalinize the urine. Hemodialysis should be considered for deterioration in the acid-base status of the patient, renal failure, NCPE, or cerebral edema. A rapidly rising serum salicylate concentration is another consideration for dialysis. Patients with chronic ingestions may meet clinical criteria for extracorporeal elimination even with serum salicylate levels in the 40 to 50 mg/dL range. Admission should be strongly considered for most of these ingestions.



FIGURE 17.37 ■ Aspirin Bezoar. Pill bezoar found in the gastrointestinal tract of a patient who ingested approximately 750 enteric-coated aspirin tablets. At the time of death, approximately 13 hours after ingestion, the serum salicylate level was 128 mg/dL. More than 300 partially digested pills remained in the

GI tract on postmortem. (Photo contributor: Jared M. Orrock, MD.)

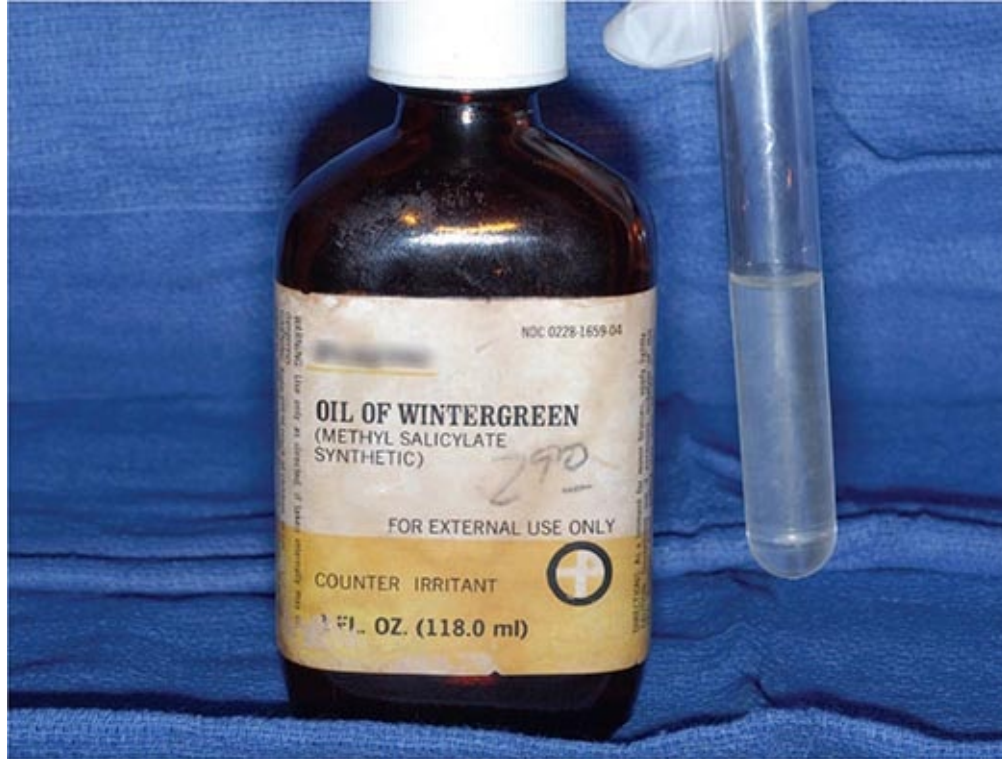


FIGURE 17.38 ■ Oil of Wintergreen. Severe salicylism may occur from ingestion of products that contain a high concentration of oil of wintergreen (methylsalicylate). This bottle of oil of wintergreen is a 98% solution, which contains the equivalent of 7000 mg of salicylate per teaspoon. (Photo contributor: R. Jason Thurman, MD.)

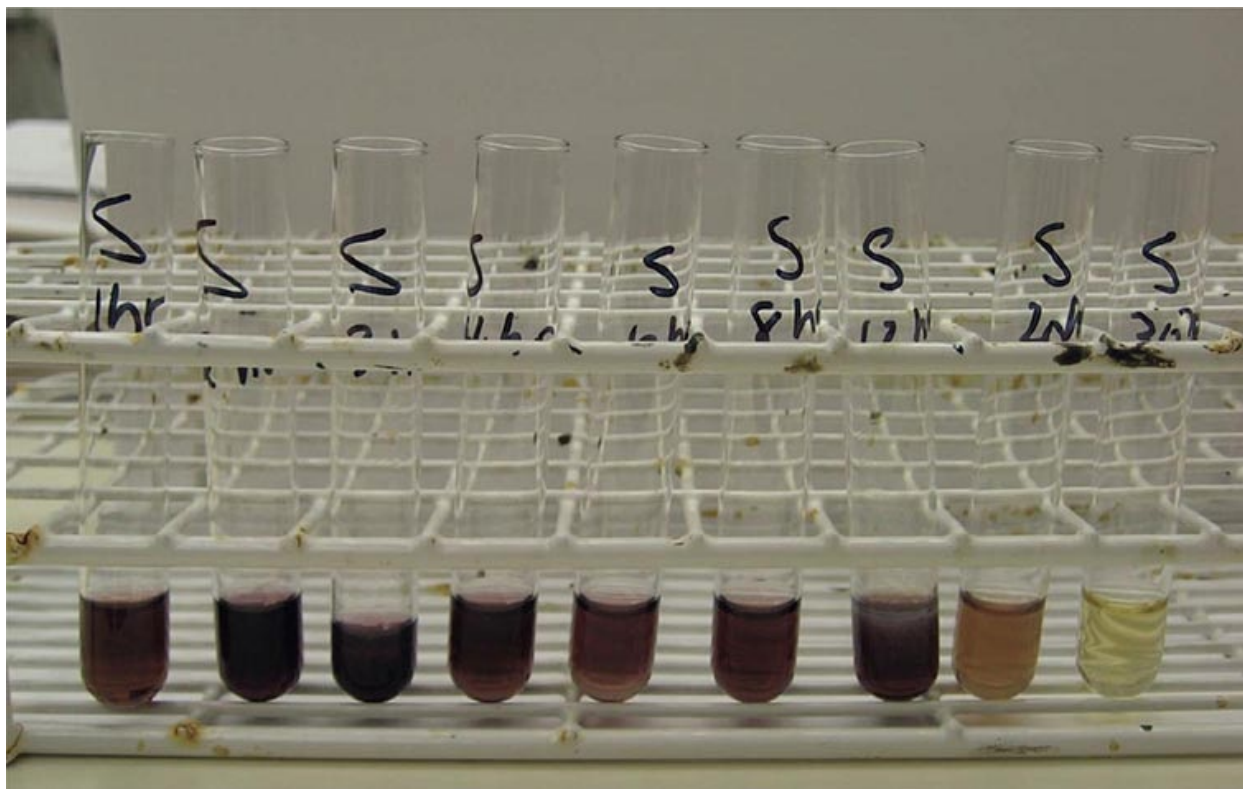


FIGURE 17.39 ■ Trinder Reagent. In the presence of salicylates, the addition of Trinder reagent to urine specimen will yield a purple color. This picture demonstrates the reaction to Trinder reagent from urine samples collected serially 1 hour to 30 hours after ingestion of 650 mg of aspirin. (Photo contributor: Sheila Dawling, PhD.)

Pearls

1. Oil of wintergreen is typically 98% methyl salicylate (1400 mg/mL). One teaspoon (5 mL) provides the equivalent 7000 mg of salicylic acid.
2. If a patient with severe salicylism must be intubated, careful attention should be made to mimic the minute ventilation of the nonparalyzed patient. If a respiratory acidosis occurs, the patient will become severely acidemic, which allows the salicylate to further distribute into the tissues and poison the mitochondria.
3. While acetazolamide administration results in alkalinization of the urine, the excretion of the bicarbonate into the urine comes at the expense of promoting acidemia, which could further drive the salicylate into the CNS and enhance toxicity.
4. The agent diflunisal will cause markedly elevated serum salicylate levels in the absence of other clinical or biochemical indicators of toxicity.

TOXIC ALCOHOL INGESTION

Clinical Summary

The commonly available toxic alcohols include ethylene glycol, methanol, and isopropanol. Ethylene glycol is a sweet-tasting liquid commonly found in antifreeze, as well as in brake fluid. Methanol is used in lock deicers, windshield wiper fluid, and industrial solvents. Isopropanol is commonly marketed as “rubbing” alcohol, although it is also found in nonstreaking glass and window cleaners, soaps, cosmetics, and antifreezes.

The parent toxic alcohols cause intoxication but are not otherwise toxic to end organs. Sequential metabolism via alcohol dehydrogenase and aldehyde dehydrogenase produces the organic acids responsible for end-organ toxicity and metabolic acidosis. Ethylene glycol is metabolized to glycolic and oxalic acids; the former is responsible for the acidosis, while the latter is responsible for calcium oxalate deposition in the renal tubules and delayed acute renal failure (24-72 hours after ingestion). Hypocalcemia may occur with severe intoxication. Methanol is less intoxicating than ethanol. Methanol is metabolized to formic acid, which is responsible for both acidosis and direct retinal toxicity. Patients often report blurred or dim vision (“snowstorm”) prior to development of objective signs, including optic disc hyperemia, pupillary dilation, and poor accommodation. Pancreatitis and delayed basal ganglia lesions may occur. Isopropanol metabolism is limited to ketone formation and does not result in significant acidosis.

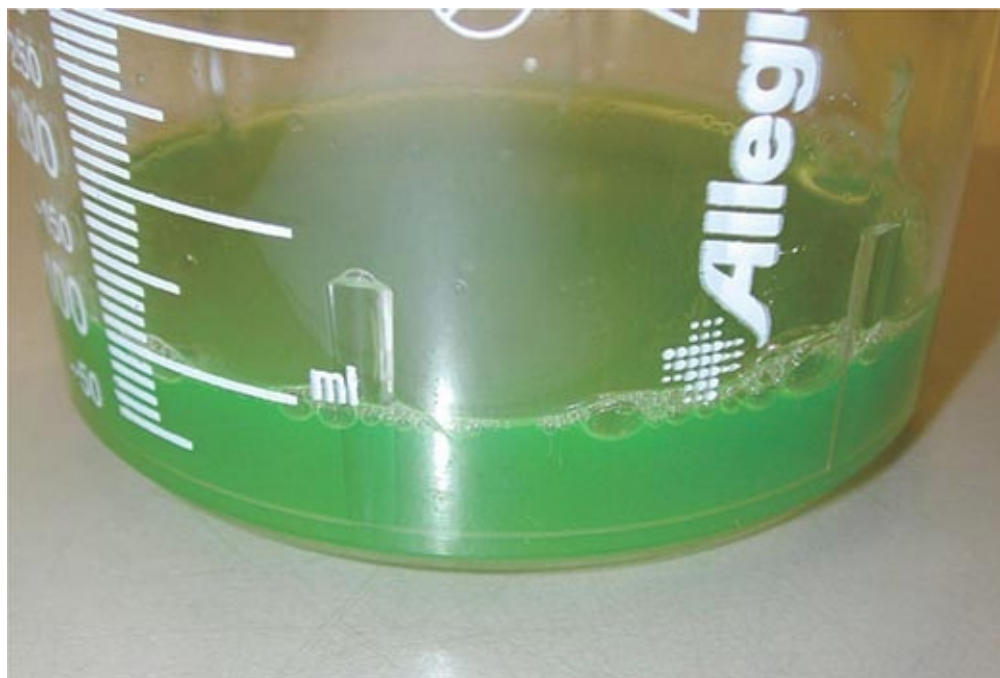


FIGURE 17.40 ■ Antifreeze. Addition of fluorescein to antifreeze gives colorless ethylene glycol its green appearance. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

Management and Disposition

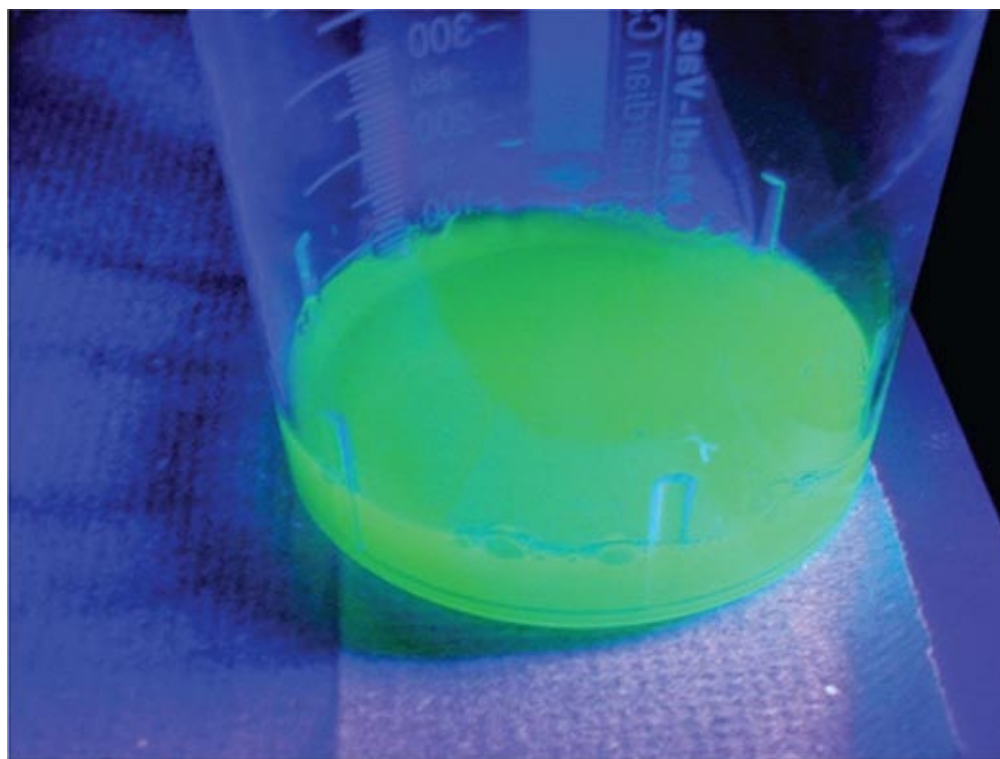


FIGURE 17.41 ■ Antifreeze Fluorescence. Application of a black light to antifreeze will demonstrate the

fluorescence in body fluids, provided fluorescein has been added. This sample was obtained from the emesis of an overdose patient. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

For ethylene glycol and methanol, emergency management is directed toward supportive care, diagnosis of the agent, and prevention of further metabolism. Methanol, ethylene glycol, and isopropanol levels may not be readily available. However, care should be taken in interpreting ancillary data, such as urinary fluorescence and the osmolar gap. Both ethanol and fomepizole competitively inhibit alcohol dehydrogenase. Both of these reduce metabolism of the parent compound but do not enhance elimination of the toxic metabolites. Administration of folate (methanol) or pyridoxine and thiamine (ethylene glycol) may inhibit organic acid production or increase degradation by shunting metabolism to alternate pathways. Hemodialysis is indicated for signs of end-organ toxicity (eg, anion gap acidosis, renal failure, mental status changes) and should be considered for elevated toxic alcohol levels. Isopropanol ingestions require primarily supportive care.

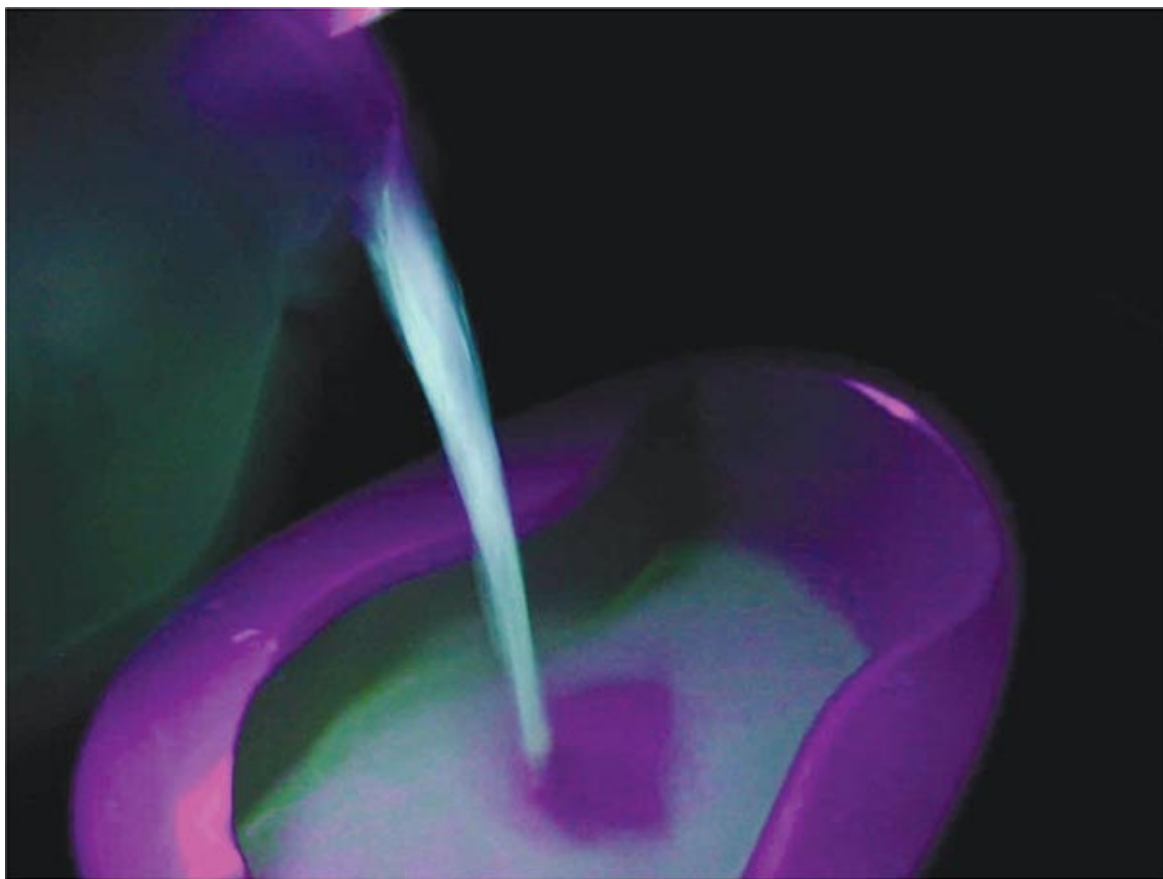


FIGURE 17.42 ■ Urine Fluorescence. Under black light, the urine of this ethylene glycol overdose patient shows a bright fluorescence. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

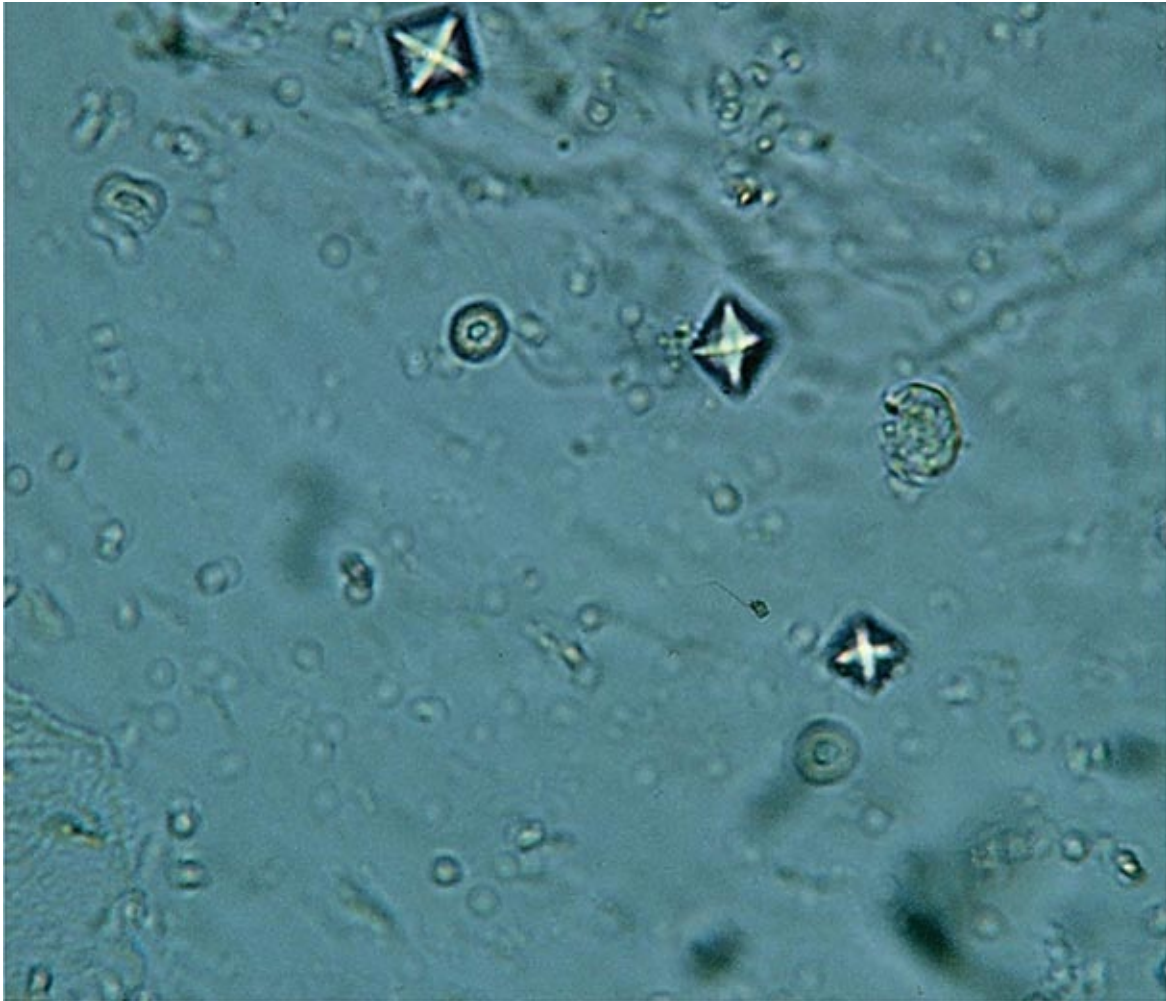


FIGURE 17.43 ■ Calcium Oxalate Crystals. Calcium oxalate crystals may be seen in the urine of the patient who ingested ethylene glycol and metabolized the parent compound to create oxalic acid. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. *Urinalysis and Body Fluids*. 4th ed. Philadelphia, PA: F.A. Davis Company; 2001.)

Pearls

1. Only a few sips of concentrated methanol or ethylene glycol are required to produce toxicity in a toddler; these ingestions should be viewed as a “one pill can kill” exposure.
2. Co-ingestion of ethanol may delay development of eventual toxicity due to preferential blockade of alcohol dehydrogenase.
3. Either fomepizole or ethanol may be used to reduce metabolism of the parent toxic alcohol. Do not administer both at the same time.
4. Due to the occurrence of hypocalcemia in ethylene glycol poisoning, careful monitoring of ionized calcium is critical when using sodium bicarbonate in

these patients.

TRICYCLIC ANTIDEPRESSANT POISONING

Clinical Summary

Tricyclic antidepressants (TCAs) are being used more frequently for a wide variety of clinical indications, and toxicity remains a significant cause of poisoning morbidity and mortality. TCA toxicity is related to pharmacologic effects on the myocardium, CNS, and vasculature. M_1 -muscarinic receptor blockade may result in an anticholinergic toxidrome. CNS toxicity may range from sedation to coma. Seizures, agitation, and delirium may occur. Inhibition of voltage-gated sodium channels results in characteristic widening of the QRS complex. A limb-lead QRS duration greater than 120 ms is associated with an increased incidence of seizures, whereas a limb-lead QRS duration greater than 160 ms is associated with an increased incidence of ventricular dysrhythmias. Similarly, in adults, a terminal R wave in lead aVR greater than or equal to 3 mm is associated with increased risk of seizure or dysrhythmias.

Management and Disposition

Signs and symptoms of significant overdose typically occur early. All patients presenting after TCA overdose should receive continuous cardiac monitoring and an ECG. Aggressive airway management may be indicated. No specific antidote exists for TCA poisoning. Benzodiazepines are the agent of choice for seizures. Management of QRS widening involves intravenous administration of sodium bicarbonate; controversy exists regarding optimal method of administration (intermittent dosing versus continuous infusion). Symptomatic patients should be admitted to the intensive care unit due to the potential for rapid deterioration.

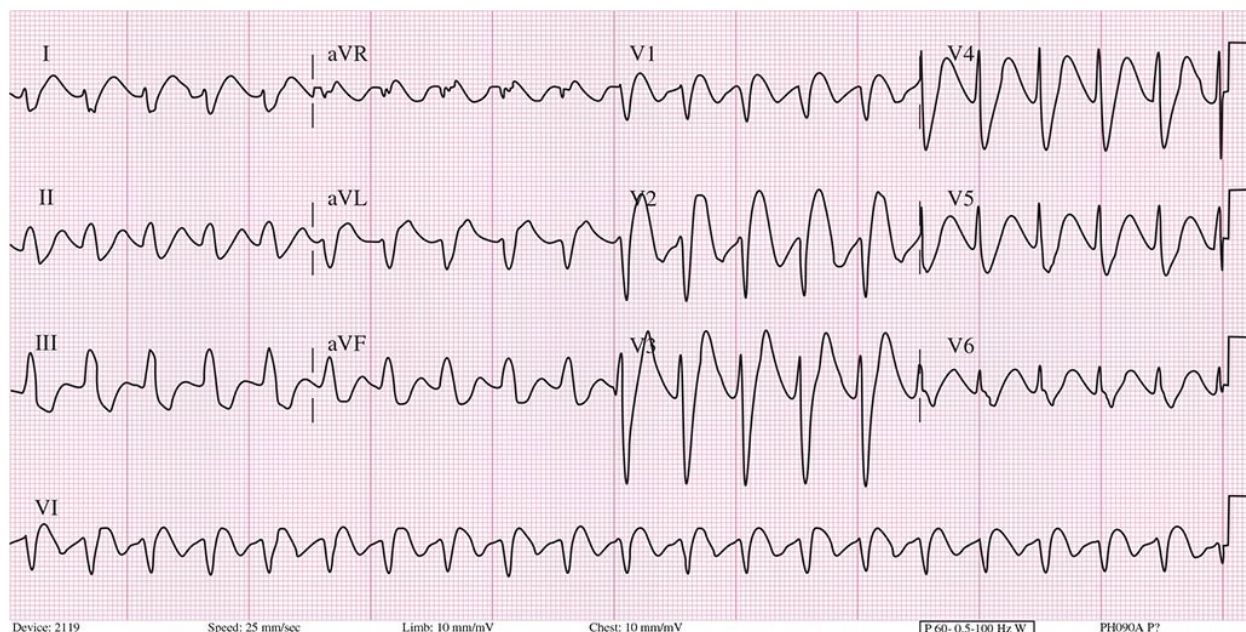


FIGURE 17.44 ■ TCA Cardiotoxicity. A 12-lead ECG of a patient who ingested a massive quantity of amitriptyline, demonstrating QRS widening. The patient presented awake and alert, but rapidly became obtunded. (Photo contributors: Thomas Babcock, MD, and Clay Smith, MD.)

Pearls

1. In one study, half of all patients presenting to emergency department with trivial signs of poisoning had catastrophic deterioration within 1 hour.
2. The use of flumazenil and physostigmine is contraindicated in the management of patients with ECG evidence of TCA poisoning.
3. Other xenobiotics that may cross-react with the TCA immunoassay on the urine drug screen include diphenhydramine, carbamazepine, cyclobenzaprine, and quetiapine.

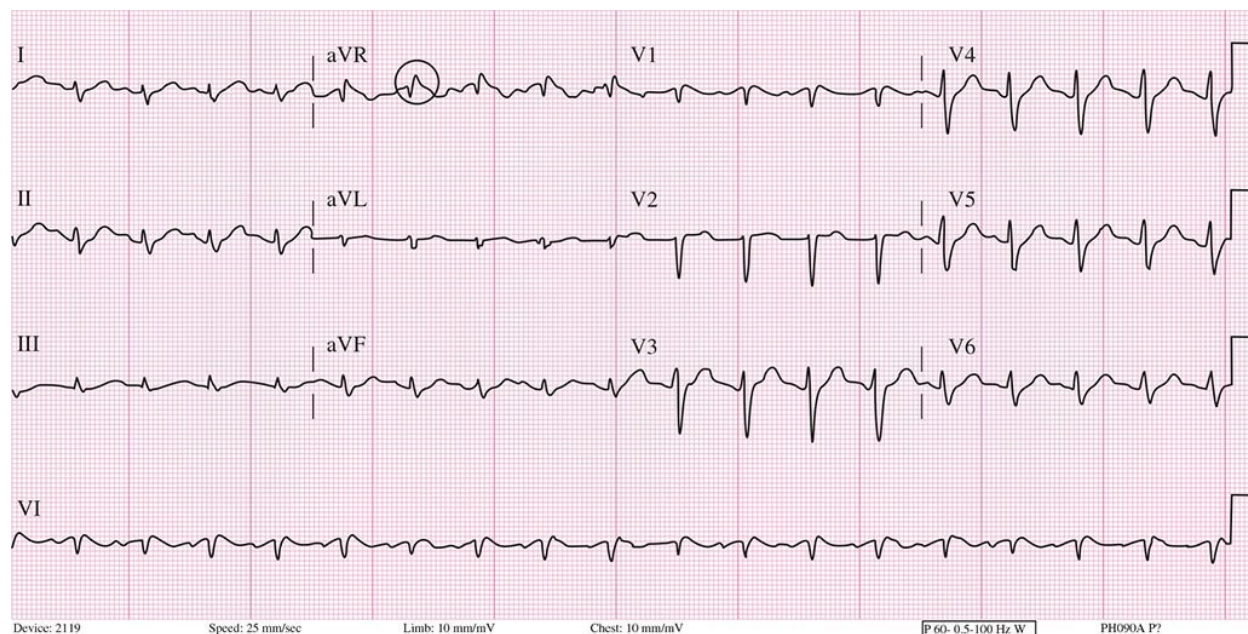


FIGURE 17.45 ■ TCA Cardiotoxicity—Treated. Repeat 12-lead ECG of the patient from Fig. 17.44 approximately 2 hours and 45 minutes after the first ECG. A total of 12 amperes of sodium bicarbonate had been administered intravenously. This ECG demonstrates the terminal R wave changes in aVR associated with sodium channel-blocking effects (circle). (Photo contributors: Thomas Babcock, MD, and Clay Smith, MD.)

POISONING BY β -BLOCKER AND CALCIUM CHANNEL BLOCKER AGENTS

Clinical Summary

β -Blockers and calcium channel blockers have various clinical indications, including the management of hypertension, myocardial infarction, and cardiac dysrhythmias, as well as the treatment of noncardiovascular conditions (eg, glaucoma, thyrotoxicosis, migraine headache prophylaxis). β -Blocking agents may be selective for B_1 -adrenergic receptors or nonselective. With therapeutic use, the commonly available calcium channel blockers are selective for the membrane-bound L-type calcium channel. Inhibition of this channel prevents influx of extracellular calcium.

Toxicity presents as an exaggeration of clinical effects, with significant toxicity manifesting predominantly as bradycardia and hypotension. β -Blocker toxicity may result in hypoglycemia, especially in children. Certain agents, such as propranolol, are associated with CNS toxicity (including seizures and CNS

depression) and fast sodium channel blockade (analogous to TCA toxicity). Calcium channel blocker toxicity is associated with hyperglycemia, believed to be secondary to impaired insulin release (a calcium-dependent process) and impaired peripheral utilization.

Management and Disposition

The use of aggressive GI decontamination (eg, whole-bowel irrigation) has been advocated for sustained-release preparations. Glucagon has been used as a specific antidote for β -blocker toxicity. No treatment has been universally successful in the management of severe calcium channel blocker toxicity. Calcium, glucagon, high-dose insulin euglycemia, and intravenous lipid emulsion have all been tried with variable success. Management of these patients should involve early consultation with a poison control center or toxicologist.

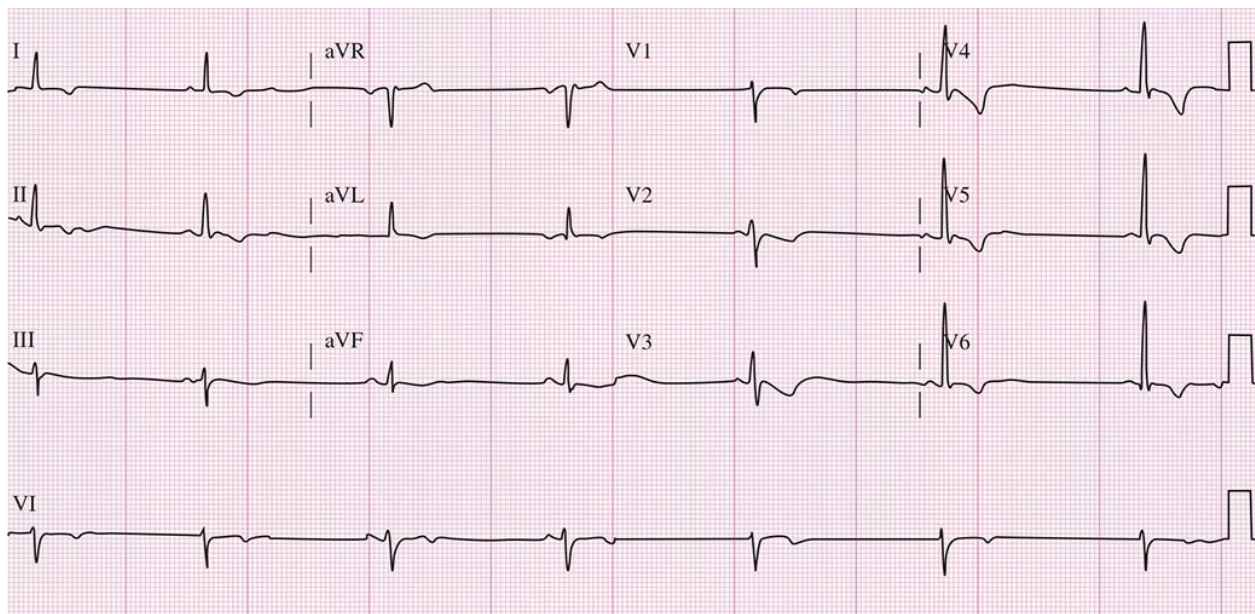


FIGURE 17.46 ■ β -Blocker Overdose. Atenolol poisoning resulting in severe bradycardia. (Photo contributor: Saralyn R. Williams, MD.)

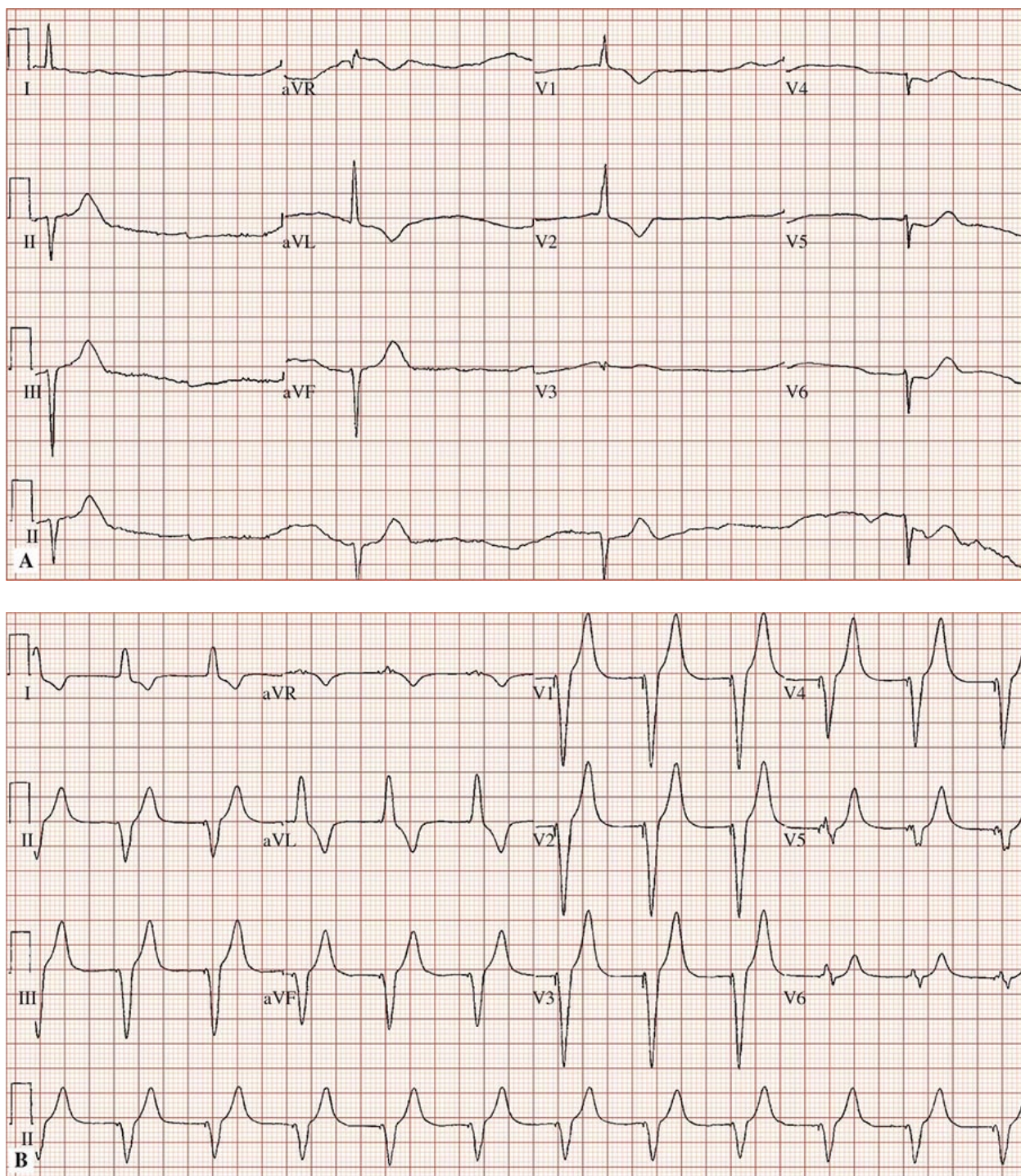


FIGURE 17.47 ■ Calcium Channel Blocker Overdose. (A) Verapamil poisoning causes profound negative inotropy and chronotropy. The 12-lead ECG demonstrates the bradycardia that may occur. (B) The same patient after transvenous pacing was initiated. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

Pearls

1. Topical β -adrenergic blocker administration (eg, for glaucoma) can result in significant systemic toxicity.
2. Development of toxicity may be appreciably delayed after ingestion of sustained-release formulations.
3. The presence of hyperglycemia versus hypoglycemia may help differentiate calcium channel blocker poisoning from β -blocker poisoning, respectively.
4. Due to the potential for significant local tissue toxicity with extravasation, calcium chloride therapy should optimally be administered through a central venous catheter.

INHALANT ABUSE

Clinical Summary

Inhalant abuse, the intentional inhalation of vapors for the purpose of becoming “high,” is more common among adolescents. Sniffing refers to the inhalation of the agent directly from a container, such as model airplane glue. Huffing involves placing solvent on some type of fabric and inhaling the vapors from the fabric. Bagging is the name given to the technique of spraying the solvent into a bag and then rebreathing from the bag. Occasionally the bag is placed over the head, potentially resulting in asphyxiant death. Inhalants are rapidly absorbed via the lungs and readily cross the blood-brain barrier. Initial effects include euphoria and occasional hallucinations. CNS depression may occur. Acute cardiotoxicity may also occur and is thought to be the cause of “sudden sniffing death.” The cause of death is thought to be due to increased myocardial sensitization that promotes dysrhythmogenesis in the setting of a catecholamine surge. A defatting dermatitis may be evident on the hands due to chronic exposure to solvents. Chronic effects from inhalant abuse include leukoencephalopathy, cardiomyopathy, cerebellar degeneration, and neuropathy.

Management and Disposition

Clues to the diagnosis of inhalant abuse are the presence of spray paint on the fingers or the face. Due to the increased solvent content in metallic-colored paints, gold and silver spray paint are particularly popular. Cardiac dysrhythmias are associated with a poor prognosis. Current recommendations suggest the use

of β -blockers to treat ventricular dysrhythmias. Consider electrolyte abnormalities and acid-base status, particularly with toluene-based products. Benzodiazepines may be used for treatment of agitation.

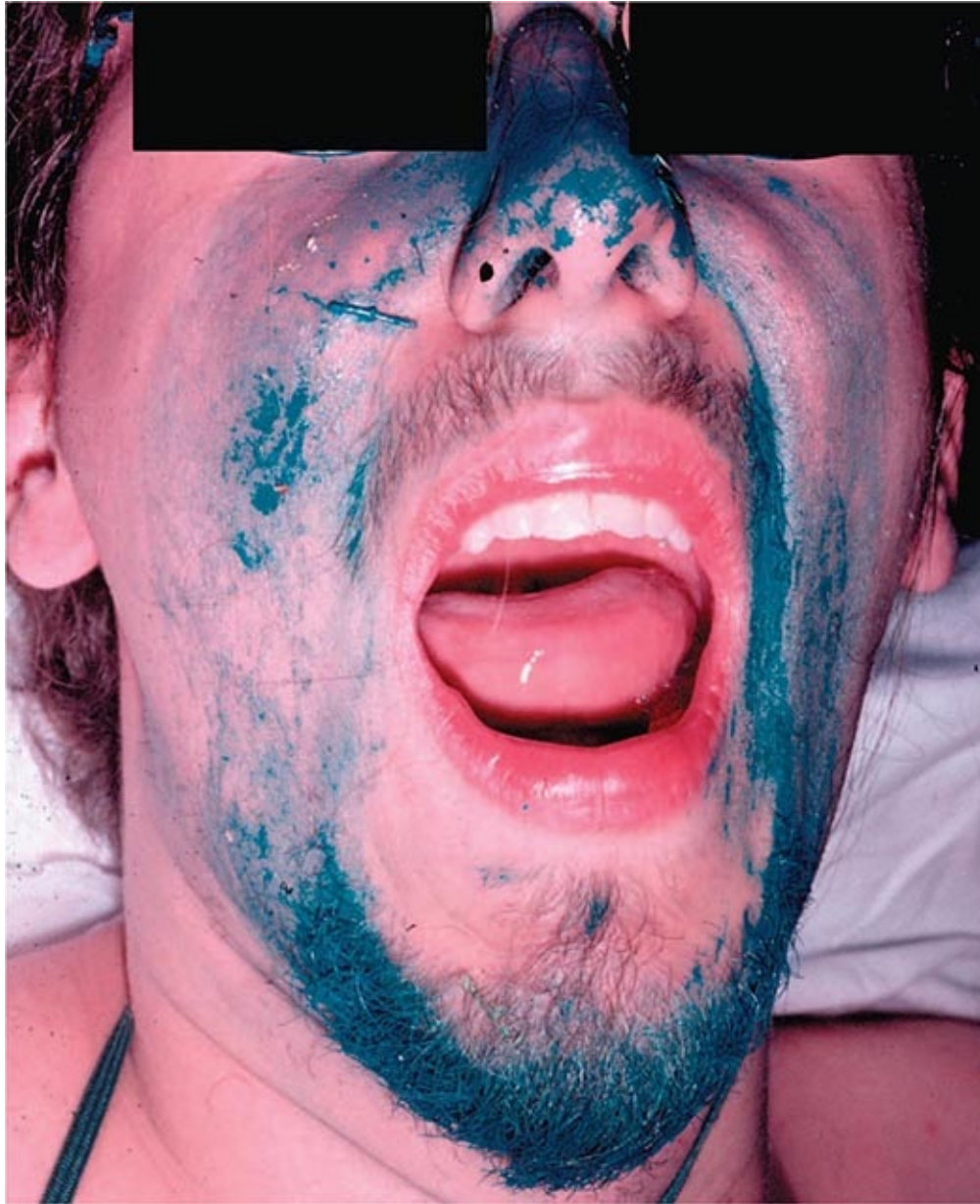


FIGURE 17.48 ■ “Huffing.” Patients who huff spray paints may present with the paint on their face and hands. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 17.49 ■ “Huffing.” The hand of the patient in Fig. 17.48. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 17.50 ■ “Bagging.” Silver paint lining is seen at the perioral area in a patient abusing the paint by “bagging.” (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.51 ■ “Bagging.” Gold and silver metallic paints are particularly popular for inhalant abuse due to the increased solvent content in the paint and a greater “high.” (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.52 ■ Motor Neuropathy of the Hand. The hands of a chronic huffer demonstrate the muscle wasting in the left hand in addition to a mild defatting dermatitis on the palm of the hand. (Photo contributor: Saralyn R. Williams, MD.)

Pearls

1. Chronic abuse of nitrous oxide (N_2O) may result in a megaloblastic anemia

and distal axonal sensorimotor neuropathy that may be a result of irreversible oxidation of cobalamin (vitamin B₁₂). In addition to being used medicinally as “laughing gas,” nitrous is abused from “whippets,” which are the cartridges of compressed air used for whipping cream canisters.

2. Chronic abuse of toluene may result in potassium-wasting renal tubular acidosis. Some of the carburetor cleaners that contain toluene also contain methanol.
3. Amyl, butyl, and isobutyl nitrites are strong oxidizers and may produce methemoglobinemia. These may be sold as “poppers.”
4. Carbon monoxide poisoning may occur after methylene chloride inhalation.

METHEMOGLOBINEMIA

Clinical Summary

Methemoglobin occurs when the iron in deoxyhemoglobin loses an electron, resulting in a ferric (Fe³⁺) ion instead of the usual ferrous (Fe²⁺) state. Ferric iron can no longer bind to oxygen, thereby reducing the oxygen-carrying capacity of hemoglobin. The presence of methemoglobin also shifts the oxygen hemoglobin dissociation curve to the left, resulting in decreased release of oxygen to tissues. Infants are more susceptible to the development of methemoglobinemia. Illnesses in infants such as diarrhea, dehydration, and acidosis may induce methemoglobin due to oxidant stress.



FIGURE 17.53 ■ Methemoglobinemia—Cyanosis. Methemoglobinemia resulted in the cyanotic appearance of this pediatric patient as noted on the hand on the left side of the image compared with the normal adult control on the right. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 17.54 ■ Methemoglobinemia—“Chocolate Blood.” “Chocolate blood” from an arterial sample of a patient with methemoglobinemia (left) compared to the normal bright red arterial blood (right). (Photo contributor: Kevin J. Knoop, MD, MS.)

Common pharmaceutical agents that cause methemoglobin include sulfonamides, dapsone, phenazopyridine, chloroquine, benzocaine, prilocaine, and more rarely lidocaine. Nitrites, which are used in the older cyanide antidote kit, induce methemoglobin.

Clues to the diagnosis include the patient who appears cyanotic and does not improve with the administration of oxygen. The pulse oximeter reading will drop to the mid 80% range but does not correlate with the percentage of methemoglobin. The blood may appear chocolate in color and does not become red with exposure to oxygen. The arterial blood gas will demonstrate a normal

partial pressure of oxygen with a resulting normal calculated arterial saturation. Methemoglobin may be measured via a co-oximeter using either arterial or venous heparinized blood.

Management and Disposition

Any patient who appears cyanotic should initially be treated with administration of supplemental oxygen and advanced airway management as appropriate. In general, any patient who is symptomatic from methemoglobinemia or has a level exceeding 25% to 30% should be treated with methylene blue. Methylene blue is available in a 1% solution and is administered as a 1 to 2 mg/kg dose intravenously over 5 minutes. This may be repeated if there is no initial response in 20 to 30 minutes. Patients who have methemoglobinemia from dapsone or aniline dyes may have recurrence and require additional dosing of methylene blue.

Pearls

1. High doses of methylene blue (5-7 mg/kg) may cause paradoxical methemoglobinemia and hemolysis.
2. The intravenous administration of methylene blue may interfere with the reading of the pulse oximeter and cause the reading to decrease transiently.
3. Methylene blue accelerates the ability of nicotinamide adenine dinucleotide phosphate (NADPH) methemoglobin reductase to reduce the ferric iron of methemoglobin back to a ferrous iron.

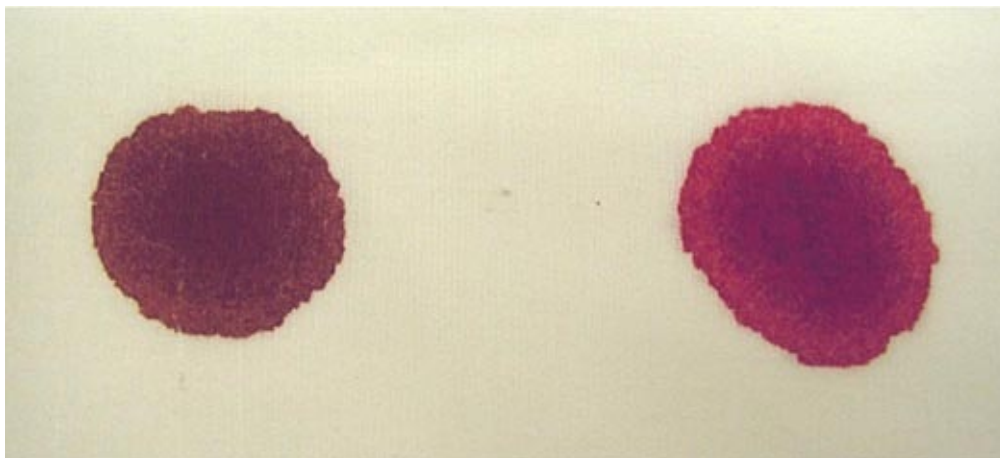


FIGURE 17.55 ■ Methemoglobinemia—Exposure to Air. The blood from a patient with methemoglobinemia on the left does not turn bright red upon exposure to air and will remain a “chocolate

color” compared to the normal blood sample on the right. (Photo contributor: Louise Kao, MD.)

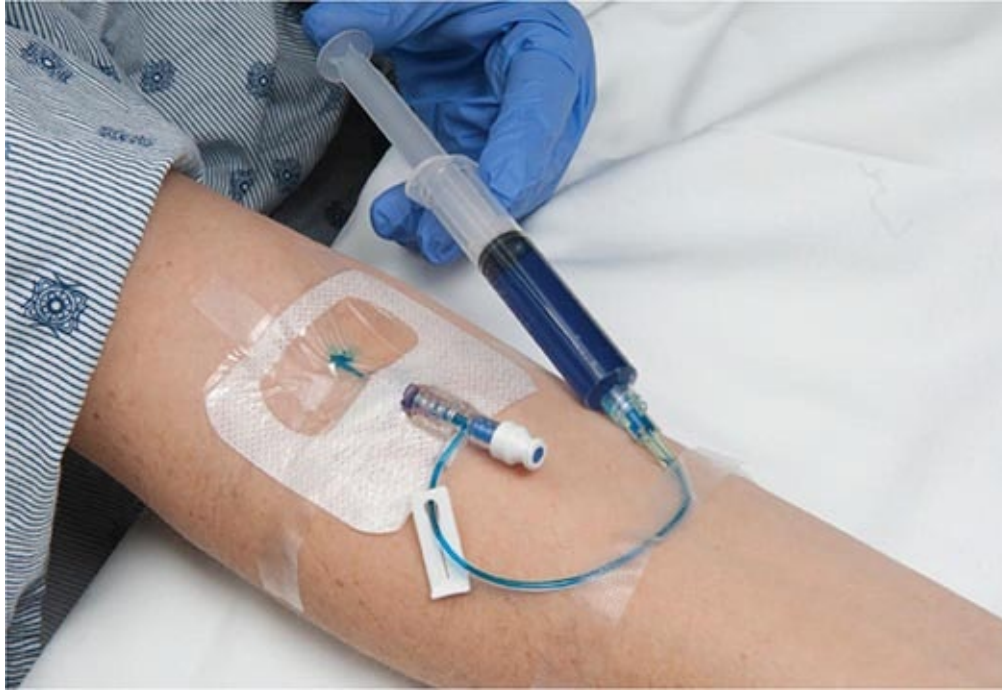


FIGURE 17.56 ■ Administration of Methylene Blue. The intravenous administration of methylene blue may be disconcerting to the patient who may not understand how a “blue dye” will help them. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.57 ■ Methylene Blue Urine. Methylene blue is excreted renally and gives a blue-green color to the urine. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)

4. Methylene blue may not reverse methemoglobin in a patient with glucose-6-phosphate dehydrogenase (G6PD) deficiency due to the absence of NADPH and may increase the risk of hemolysis.
5. Methylene blue has monoamine oxidase inhibitor activity; thus, use in patients who are on serotonin reuptake inhibitors may result in a serotonin syndrome.

CELLULAR ASPHYXIANTS

Clinical Summary

The cellular asphyxiants are a diverse group of substances including carbon monoxide (CO), cyanide, sodium azide, met-hemoglobin-producing oxidizing agents, and hydrogen sulfide, all of which interfere with the cellular utilization of oxygen. Depending on the substance, the interference may occur at the level of hemoglobin, the electron transport chain, or both. In contrast with the simple asphyxiants, ambient oxygen concentrations are not affected.

CO is a colorless and odorless gas generated from the incomplete combustion of carbonaceous compounds. The affinity of CO for hemoglobin is 250 times greater than that of oxygen. Binding of CO to hemoglobin shifts the oxyhemoglobin dissociation curve to the left, further impairing tissue oxygen delivery. Symptoms of acute poisoning may range from headache to ischemic chest pain, seizures, and CNS depression. Up to 40% of poisoned patients develop delayed neurologic sequelae (DNS); most cases of DNS are associated with an initial loss of consciousness.

Although a nonspecific enzyme inhibitor, cyanide interferes with oxidative phosphorylation. Sources of cyanide include industrial and household chemicals, plants, and structure fires. Clinical manifestations reflect dysfunction of oxygen-sensitive organs, including the CNS and cardiovascular systems. A cyanide toxidrome has been described, consisting of altered mental status, mydriasis, respiratory depression, hypotension, tachycardia, and metabolic (lactic) acidosis.

Management and Disposition

Immediate management focuses on airway stabilization and antidotal therapy. Carboxyhemoglobin levels should be obtained in patients with suspected CO poisoning. Pregnancy status should be determined in females presenting with suspected CO poisoning. Blood cyanide levels are not typically available in the immediate care setting. While 100% oxygen is the accepted antidote for acute CO poisoning, controversy persists regarding the mode of administration (normobaric oxygen versus hyperbaric oxygen). Potential indications for hyperbaric oxygen include syncope, altered mental status (especially with evidence of cerebellar dysfunction), acidosis, and pregnancy. Therapy for cyanide poisoning includes intravenous administration of hydroxocobalamin. Hydroxocobalamin has the advantage of raising blood pressure, but also causes intense red skin discoloration and chromaturia.

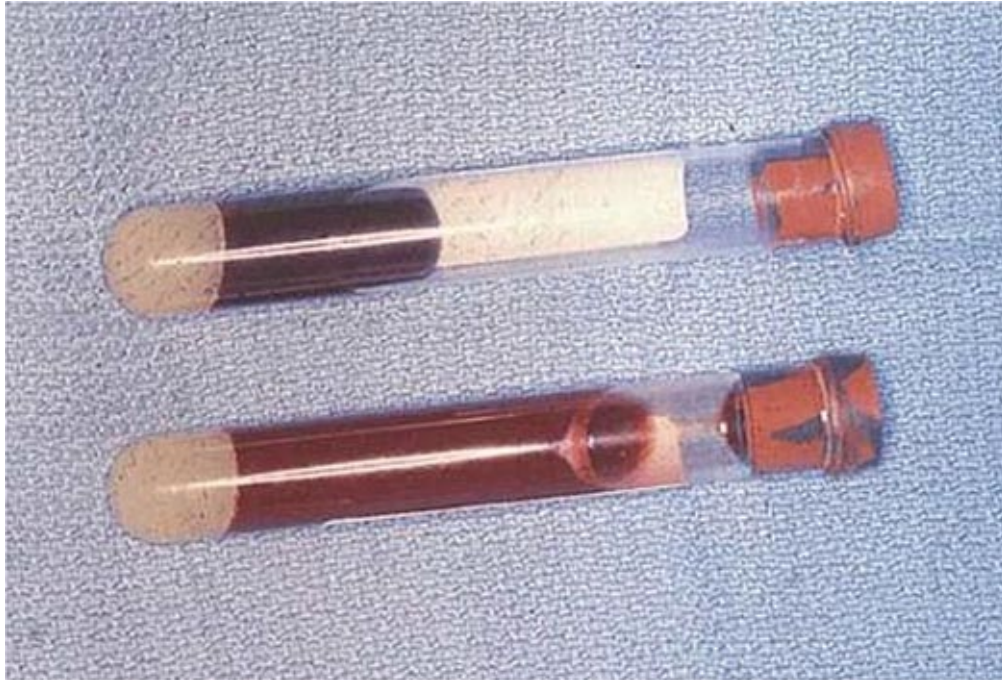


FIGURE 17.58 ■ Carbon Monoxide Poisoning. Venous blood samples with the bright red one (bottom sample) taken from a patient with acute carbon monoxide poisoning. The dark red venous blood (top sample) is a control sample from a patient with no carboxyhemoglobin. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 17.59 ■ Skin Changes after Hydroxocobalamin. After administration of therapeutic dosing of hydroxocobalamin, the skin of the patient may exhibit a bright red color. (Reproduced with permission from

Uhl W, et al. Safety of hydroxocobalamin in healthy volunteers in a randomized, placebo-controlled study. *Clin Toxicol*. 2006;44:17–28. Copyright © Informa Healthcare.)



FIGURE 17.60 ■ Chromaturia after Hydroxocobalamin. Shortly after the intravenous administration of hydroxocobalamin, chromaturia may be evident. (Photo contributor: Saralyn R. Williams, MD.)

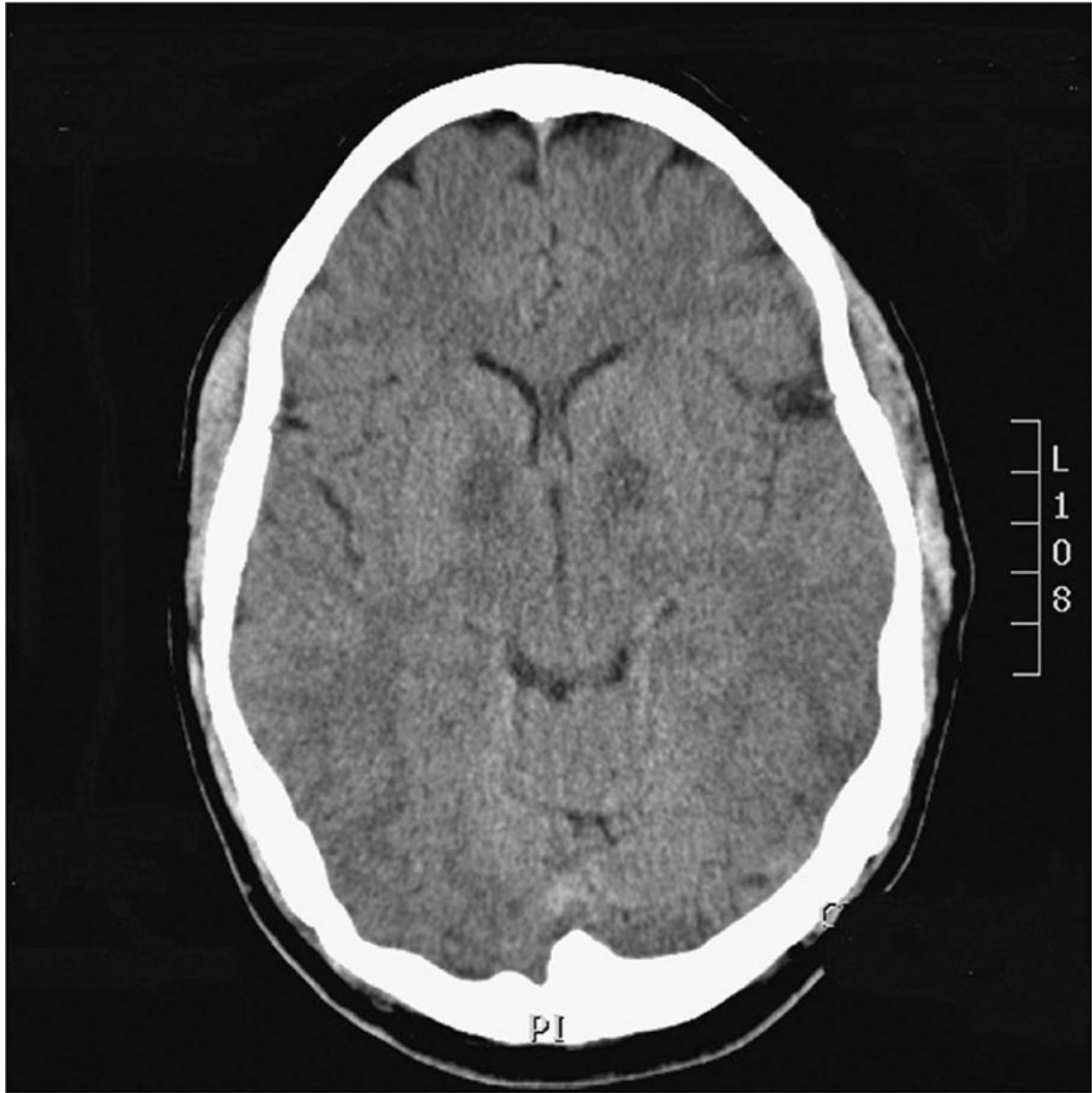


FIGURE 17.61 ■ Bilateral Globus Pallidus Lesions. Selective bilateral injury to the globus pallidus may be seen with cellular asphyxiants such as carbon monoxide or cyanide poisoning. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. The decision to treat acute CO poisoning is based on history and physical examination, and not solely on carboxyhemoglobin level.
2. Maternal carboxyhemoglobin levels fail to accurately reflect fetal carboxyhemoglobin levels.

3. In victims of structure fires who present without severe burns, a plasma lactate greater than 10 mmol/L correlates with cyanide level greater than 40 $\mu\text{mol/L}$.
4. Empiric therapy with hydroxocobalamin may be considered for victims of structure fires in whom suspicion exists for cyanide poisoning.
5. CO poisoning, as well as cyanide toxicity, may cause selective bilateral injury to the globus pallidus.

VANCOMYCIN-INDUCED RED MAN SYNDROME

Clinical Summary

Vancomycin has activity against gram-positive bacteria with little to no activity against gram-negative bacteria or mycobacteria. It is poorly absorbed after oral administration, although it may be used orally for treatment of pseudomembranous colitis. Intravenous administration is the most common route. This is well tolerated with minimal burning at the site of the intravenous line; however, rapid infusion may occasionally cause degranulation of mast cells and basophils. As a result, the patient experiences erythematous flushing, particularly of the face and neck, hence the name “red man syndrome.” Tachycardia and hypotension may occasionally be seen.

Management and Disposition

Slowing the intravenous infusion usually resolves the flushing. Increasing the dilution of vancomycin in solution may also assist with preventing the flushing. Diphenhydramine has been used for treatment and may be used as a pretreatment.

Pearls

1. The differential diagnosis for flushing includes scombroid poisoning, disulfiram reactions, niacin, and hydroxocobalamin infusions.
2. Concomitant administration of aminoglycoside with vancomycin may increase the risk of nephrotoxicity.



FIGURE 17.62 ■ Red Man Syndrome. Facial and neck flushing are manifestations that may be seen with red man syndrome from intravenous vancomycin infusion. (Photo contributor: R. Jason Thurman, MD.)

BOTULISM

Clinical Summary

Botulinum is a potent neurotoxin that is derived primarily from *Clostridium botulinum*. Botulinum toxin blocks release of acetylcholine, which results in decreased activation of muscarinic and nicotinic receptors. Initial effects may include nonspecific findings such as nausea and vomiting, constipation, and throat complaints. The classic neurologic findings are due to the lack of receptor activation of the nicotinic receptor at the neuromuscular junction. Dysarthria, dysphagia, diplopia, and mydriasis progress to a descending symmetric paralysis.

The common types of botulism include foodborne (ingestion of preformed toxin), infantile (in vivo production of toxin), and wound botulism (in vivo production of toxin). While food-borne botulism has the features classically described, infantile botulism manifests as the constipated, floppy baby. Wound botulism is most commonly associated with “skin popping,” a technique of subcutaneous injection of an illicit drug, usually black tar heroin. Diagnosis is initially a clinical one, with subsequent verification via a murine assay of the presence of botulinum toxin in a patient sample. This assay is performed through either the state department of health or the Centers for Disease Control and Prevention.

Management and Disposition

Any patient in whom botulism is suspected should be admitted to the hospital. Careful monitoring of the airway status is important to ensure that the patient has adequate ventilatory capacity. The local health department should be notified to assist with the procurement of botulinum antitoxin for foodborne and wound botulism patients. Human botulinum immune globulin is available through the California Department of Health for the treatment of infantile botulism.

Pearls

1. The initial chief complaint of a patient with wound botulism may be “sore throat” since the patient has dry mucous membranes and difficulty swallowing.
2. Since botulinum toxin does not cross the blood-brain barrier, the mental status should not be affected unless the patient has respiratory insufficiency.
3. Early signs of infantile botulism may be difficulty with feeding since feeding

for an infant requires use of the cranial nerves.



FIGURE 17.63 ■ Infantile Botulism. The floppy, constipated baby is a classic presentation of infantile botulism. (Photo contributor: Centers for Disease Control and Prevention.)



FIGURE 17.64 ■ Wound Botulism. Wound botulism occurs from the in vivo production of botulinum toxin. It manifests the same neurotoxicity, with the ptosis, bulbar paralysis, and respiratory compromise, as foodborne botulism. Note the profound ptosis in this patient. (Photo contributor: William H. Richardson, III, MD.)

ANTICOAGULANTS

Clinical Summary

Since the discovery of the cause of a hemorrhagic disorder in Wisconsin cattle in

the early 20th century, warfarin and its analogs have been used as rodenticides and pharmacologic agents. Warfarin is actually named after the Wisconsin Alumni Research Foundation. Warfarin-like xenobiotics inhibit the activity of vitamin K 2,3-epoxide reductase, thus affecting the production of the vitamin K–dependent factors of II, VII, IX, and X. While the effect on the production of the vitamin K–dependent factors occurs soon after absorption of warfarin, the clinical effect is delayed until the available activated factors are depleted.

Long-acting anticoagulants, known as “superwarfarins,” were developed as rodenticides due to the development of warfarin-resistant rodents. Their mechanism of action is the same as warfarin, but they are more potent and exhibit a much longer duration of effect. Poisoned patients may present with any sequelae of an anticoagulated state spanning the spectrum from easy bruising to life-threatening hemorrhage. In 2018, there was an outbreak of poisoning by long-acting anticoagulants that were presumed adulterants in synthetic cannabinoids (“K2/Spice”). Deaths occurred as a result of major bleeding events.

Management and Disposition

Patients who have a supratherapeutic international normalized ratio without bleeding may require reversal with the administration of vitamin K. Life-threatening hemorrhage may require immediate reversal with administration of 4-factor prothrombin complex concentrate (PCC) or fresh frozen plasma. Guidelines for correction of vitamin K antagonists are published by the American College of Chest Physicians. Patients with intentional poisonings from the long-acting anticoagulants (“superwarfarins”) may require high-dose vitamin K therapy for several weeks to months due to the prolonged anticoagulant effects.

Pearls

1. Warfarin is a racemic mixture of R and S enantiomers, which are metabolized by different CYP P450 pathways. The S enantiomer is more potent than the R enantiomer. Genetic polymorphisms influence the dose needed to achieve a therapeutic level.
2. Single unintentional ingestions of the “superwarfarins” by children under 6 years of age rarely result in an ingestion with clinically significant anticoagulant effect. Repetitive ingestions can result in coagulopathy. If a toddler has a vitamin K–dependent coagulopathy and the history indicates the

child ate a box of rat poison, one must have an index of suspicion for Munchausen by proxy.



FIGURE 17.65 ■ Rodenticide Package. Brodifacoum is a common long-acting anticoagulant used as a rodenticide for mice. Notice that the percentage of the active ingredient is only 0.005%. Thus, ingestion of a few pellets by a young child is unlikely to cause a coagulation disorder. (Photo contributor: R. Jason Thurman, MD.)

3. If a patient is on chronic warfarin therapy, be wary of the many xenobiotic interactions that can potentiate or antagonize the anticoagulant effect.



FIGURE 17.66 ■ Rodenticide Pellets. These are examples of the bluish-green pellets that are contained in the packaging for brodifacoum. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.67 ■ Warfarin Overdose. Generalized bruising as shown on the legs of this patient may be one of the presenting signs in a patient with a warfarin overdose. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.68 ■ Flank Bruising. This patient experienced spontaneous bleeding and bruising from use of a long-acting anticoagulant that was a contaminant of his synthetic cannabinoid. His international normalized ratio was above limits of detection. (Photo contributor: Saralyn R. Williams, MD.)



FIGURE 17.69 ■ Gross Hematuria. This is a urine sample from the patient in [Fig. 17.68](#). (Photo contributor: Saralyn R. Williams, MD.)

CAUSTIC INGESTION

Clinical Summary

Caustics are a diverse group of household and industrial products and

pharmaceutical agents that cause functional and histologic tissue damage through direct contact. They represent the 2nd most common toxic exposure for children 5 years of age or under. These agents are frequently described in terms of pH, with acids typically having a pH less than 3 and alkali (bases) typically having a pH greater than 11. Despite a near-physiologic pH, phenol may produce severe burns due to a high titratable acid reserve.

Alkali exposure results in a liquefactive necrosis, with deep and progressive tissue damage, predominantly to the esophagus. Endoscopic grading of esophageal burns is similar to thermal burns, ranging from mucosal hyperemia and edema (grade I) to full-thickness burns (grade III). Acid ingestion results in coagulative necrosis, which limits the depth of penetration. Damage is predominantly localized to the gastric mucosa, with pooling of the caustic agent in the antrum.

Management and Disposition

The primary goal of management is airway assessment and stabilization. Hypotension is a grave finding. A serum pH less than 7.20 may indicate the need for surgical exploration. Activated charcoal decontamination is relatively contraindicated. Endoscopy is recommended after large or deliberate caustic ingestion, presence of oral burns, or persistent refusal to take oral liquids. Steroids are occasionally used as an effort to prevent esophageal strictures in selected patients, but the decision to administer is made based on endoscopic grading in conjunction with the gastroenterologist.



FIGURE 17.70 ■ Caustic Oropharyngeal Burns. This patient suffered caustic burns by accidentally drinking a highly alkali solution that was stored in a soda can. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. The absence of oropharyngeal burns is a poor predictor of distal esophageal injury. The presence of vomiting, drooling, or stridor is more predictive of significant esophageal injury on endoscopy.
2. Analogous to thermal burns after smoke inhalation, upper airway edema and airway obstruction may occur abruptly.
3. Ingestion of muriatic acid (HCl) results in an initial non-anion gap metabolic acidosis.



FIGURE 17.71 ■ Caustic Esophageal Burns. Close-up image of the alkali burns on the tongue of the patient in [Fig. 17.70](#). (Photo contributor: Lawrence B. Stack, MD.)

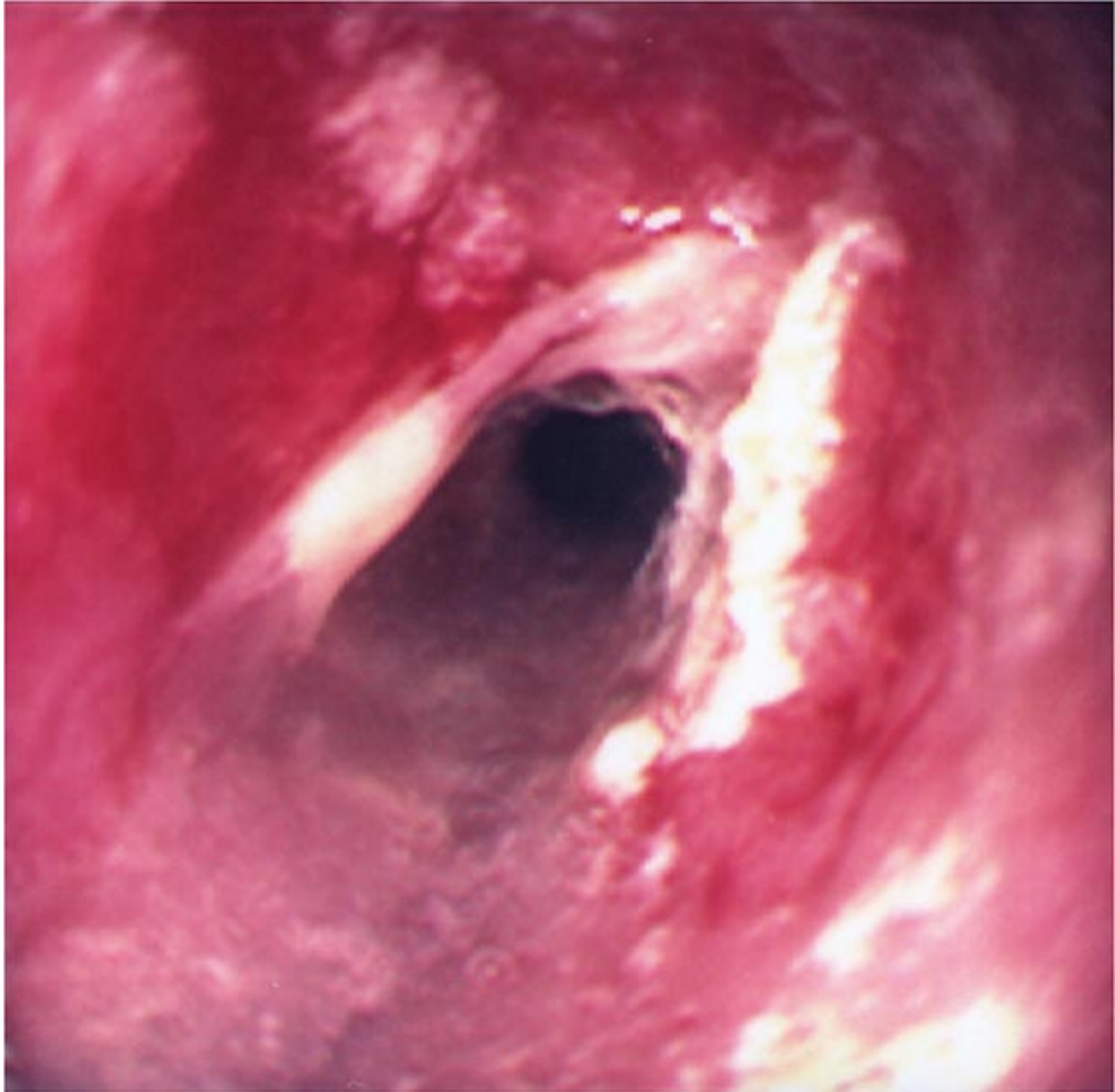


FIGURE 17.72 ■ Caustic Esophageal Burns. These esophageal burns were caused by an accidental ingestion of lye in a pediatric patient. (Photo contributor: Philip E. Stack, MD.)

HYDROFLUORIC ACID BURNS

Clinical Summary

Hydrofluoric acid (HF) is a colorless, corrosive liquid available in both commercial ($> 20\%$) and household ($< 20\%$, typically 6%-12%) formulations.

Commercially, HF is used in glass etching, electroplating, and semiconductor manufacturing. Consumer products are typically marketed as rust removers and chrome cleaners. Although toxic via the dermal, ocular, pulmonary, and GI routes, most patients present after dermal exposure, typically to the hands and fingers. The severity of local injury depends on the HF concentration and the extent of exposure.

Symptoms and tissue effects may be appreciably delayed, especially with household formulations. In the setting of hand exposure, pain is typically described as a progressive, severe, unremitting, deep burning sensation. Early local erythema is variable. Especially with higher HF concentrations, a pale blanching appearance may develop. HF causes coagulative necrosis similar to other inorganic acids, but may also result in toxicity by the binding and precipitation of calcium ions. In addition to local effects, significant and potentially life-threatening systemic effects may occur including hypocalcemia, hyperkalemia, and hypomagnesemia. Concentrations greater than 50% may cause rapid decompensation with even small dermal exposures (1% total body surface area [TBSA]).

Management and Disposition

Treatment varies depending on the route of exposure but is directed toward decontamination, neutralization of the fluoride ion, and pain control. In the setting of ingestion or significant dermal exposure (> 5% TBSA of a household HF formulation), serum electrolytes, including calcium and magnesium, should be obtained. In addition to pain control, management options for hand burns include commercially available calcium gluconate gel, subcutaneous calcium gluconate infiltration, and regional intravenous and intra-arterial calcium gluconate infusion.

Pearls

1. An expedient 2.5% calcium gluconate gel can be made by adding 3.5 g of calcium gluconate powder to 5 oz (150 mL) of water-based lubricant. Alternatively, 10 crushed 1-g calcium carbonate antacid tablets may be added to 20 mL of water-based lubricant. The resultant gel may be placed in a rubber glove and placed on the patient's hand for topical treatment (see [Fig. 17.76](#)).
2. With significant dermal exposure or ingestion, prolonged cardiac and serum electrolyte monitoring may be required; sudden delayed cardiac arrest has

occurred.

3. Replacement of calcium and magnesium may require substantially larger doses than typically required.



FIGURE 17.73 ■ Hydrofluoric Acid Burns. These hydrofluoric acid burns are due to application of a rust remover agent. The patient presented hours later after initial use with severe deep pain in the thumb and index finger. (Photo contributor: Karen Rogers, MD.)



FIGURE 17.74 ■ Hydrofluoric Acid Burn. This patient suffered severely painful hydrofluoric acid burns to the hands. The exposure resulted from the use of commercial chrome cleaner. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 17.75 ■ Hydrofluoric Acid Burn—Nailbeds. Hydrofluoric acid can seep underneath the nailbeds, resulting in blanched discoloration. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.76 ■ Hydrofluoric Acid Burn Treatment. Expedient topical therapy for hydrofluoric acid burns can be made using crushed calcium containing antacids mixed with surgical jelly. The jelly may be placed in a rubber glove, and then the glove put on the patient's hand. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

ARSENIC POISONING

Clinical Summary

Arsenic, a tasteless and odorless metalloid, is well absorbed by multiple routes of administration. Although arsenic exists in both inorganic and organic species, only the inorganic form is responsible for toxicity. Contaminated soil and water with inorganic arsenic are the primary sources of exposure to the general population. Chronic arsenic poisoning due to contaminated water continues to be a global health issue.

Arsenic inhibits multiple enzymes critical to the production of ATP. It inhibits

pyruvate dehydrogenase complex, decreases the citric acid cycle, and decreases gluconeogenesis. Acute arsenic poisoning typically starts with acute onset of nausea, vomiting, abdominal pain, and “rice water” diarrhea. Acute encephalopathy, acute renal failure, lung injury, and death may occur. Later findings in survivors include alopecia, rash, Mees lines, and neuropathy. Chronic toxicity results in dermatologic changes such as hyperpigmentation or hypopigmentation. Hyperkeratosis may occur on the skin, particularly the palms and soles. Peripheral vascular disease (blackfoot disease) may occur. Arsenic is a known carcinogen and is associated with skin and lung cancers.

Management and Disposition

After an acute ingestion, an abdominal radiograph may demonstrate radiopaque material in the GI tract. Supportive care with maintenance of electrolytes is important. A 24-hour urine collection in a metal-free container is the optimal method for determining arsenic burden. However, the acutely ill patient will likely require chelation before urine results are available. Chelation is usually initiated with intramuscular dimercaprol (British anti-Lewisite [BAL]). Succimer, an oral analog of BAL, may be useful in subacute poisoning. Patients with suspected acute arsenic poisoning should be admitted to an intensive care unit.

Pearls

1. When a nonselective heavy metal urine test is performed for possible arsenic exposure, the measured arsenic typically reflects the presence of nontoxic organic species from dietary sources such as seafood rather than toxic inorganic species. As such, urine should either be speciated, or the patient advised to refrain from seafood prior to urine collection.
2. Intravenous arsenic trioxide is approved by the US Food and Drug Administration for treatment of acute promyelocytic leukemia unresponsive to other therapies.
3. Arsenic is one of the few heavy metals that is directly cardiotoxic due to its blockade of delayed rectifier channels. Torsade de pointes has been described in the setting of acute arsenic poisoning.



FIGURE 17.77 ■ Chronic Arsenic Poisoning. Hyperpigmentation of the skin is a clinical finding from chronic arsenic poisoning. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.78 ■ Chronic Arsenic Poisoning. Hyperpigmentation and hypopigmentation along with hyperkeratosis are findings on the palms of patients with chronic arsenic poisoning. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.79 ■ Chronic Arsenic Poisoning. The patchy hair loss seen in this photograph is from chronic arsenic poisoning. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 17.80 ■ Mees Lines. Characteristic Mees lines of chronic arsenic poisoning. Note the transverse white lines on all the nails of both hands. Mees lines are often seen in conjunction with polyneuropathy of arsenic poisoning. (Photo contributor: Robert Hoffman, MD.)

IRON POISONING

Clinical Summary

Iron is a commonly used pharmaceutical agent and supplement found in prenatal vitamins and multivitamins. The toxic dose depends on the quantity of elemental iron in the preparation, which in turn depends on the iron formulation. While the minimum toxic dose remains controversial, ingestion of more than 40 mg/kg of elemental iron may result in toxicity, while ingestion of more than 60 mg/kg of elemental iron is associated with severe morbidity and possible mortality.

Iron toxicity is classically described as a four-stage process. Stage I develops within the first few hours of ingestion and reflects the direct caustic effects of

iron on the GI tract. Signs and symptoms may include abdominal pain and GI bleeding. Stage II, variably present, is a redistributive or quiescent phase; although the initial GI symptoms resolve during this phase, acidosis and end-organ toxicity may still develop. With significant toxicity, individuals may progress directly from stage I to stage III. Stage III is the phase of overt shock, metabolic acidosis, and end-organ dysfunction (including delayed hepatic failure). Individuals who survive stage III may rarely progress to stage IV—gastric outlet obstruction secondary to the initial caustic insult of stage I.

Management and Disposition

Activated charcoal does not bind iron. Whole-bowel irrigation has been advocated for substantial ingestions and for patients with evidence of iron tablets on abdominal radiographs. Serum iron levels peak between 2 and 6 hours after ingestion. Levels obtained more than 6 hours after ingestion are unreliable due to tissue redistribution. Patients with evidence of iron toxicity (eg, persistent vomiting, acidosis, altered mental status, and hypotension) or a 4- to 6-hour postingestion serum level more than 500 µg/dL should receive chelation with deferoxamine.

Pearls

1. Acute iron ingestion and the risk for toxicity must be assessed based on the quantity of elemental iron ingested, not the total amount of iron ingested.
2. The absence of radiopaque materials on abdominal radiographs is not a reliable indicator to exclude potential iron toxicity; liquid and pediatric (chewable) formulations are not typically radiopaque.
3. Despite the potential for anaphylactoid reactions and hypotension, patients requiring chelation therapy should receive deferoxamine via the intravenous route.



FIGURE 17.81 ■ Radiopaque Iron. KUB radiograph of a patient with an acute iron ingestion, demonstrating radiopaque foreign bodies as noted in the left mid-quadrant and right lower pelvis. (Photo contributor: Saralyn R. Williams, MD.)

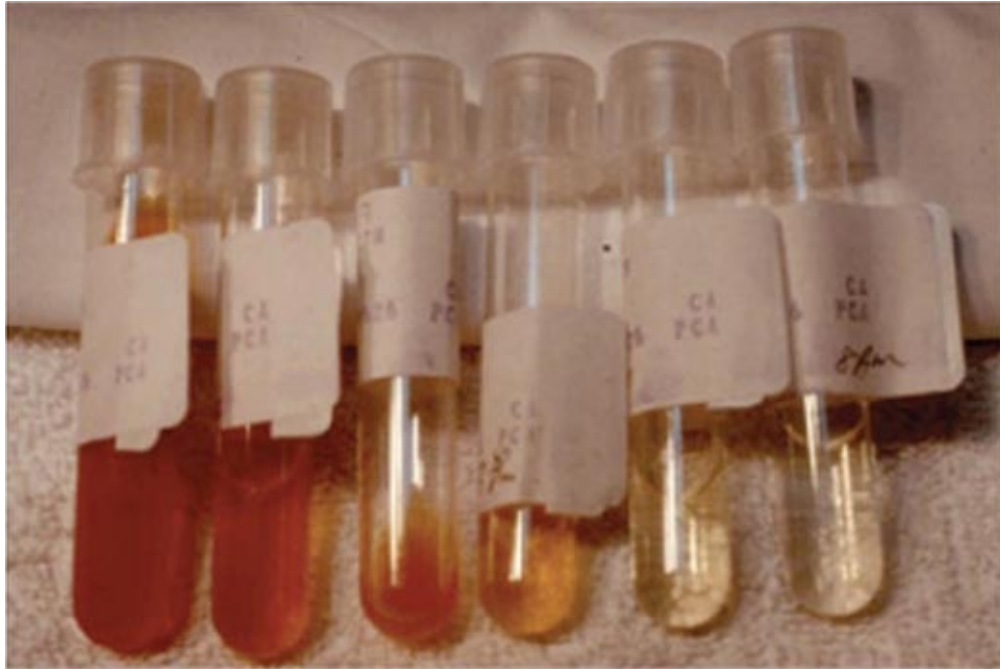


FIGURE 17.82 ■ Vin Rose Urine—Iron Poisoning. A rare example of the progression of coloration of vin rose urine (from the excreted ferrioxamine complex) over 15 hours of chelation with deferoxamine is shown. Deferoxamine chelates free iron (but not iron present in the transferrin, hemoglobin, hemosiderin, or ferritin). (Reproduced with permission from Strange GR, Ahrens WR, Schafermeyer RW, et al. *Pediatric Emergency Medicine*. 3rd ed. New York, NY: McGraw Hill; 2009: Fig. 127-1.)

LEAD POISONING

Clinical Summary

Although the prevalence of markedly elevated lead levels in the population has been declining, acute and chronic lead poisoning still occurs. Lead is well absorbed by the lungs and less well absorbed via the GI tract. Lead paint in older homes is a continued source of lead exposure. Other possible exposures may occur from occupational exposures, retained lead bullets in synovial fluid, jewelry, lead-painted toys, fishing weights, ceramic glazes, and cosmetics. Severe lead poisoning in adults has also been associated with ingestion of contaminated moonshine.



FIGURE 17.83 ■ Acute Lead Poisoning. This patient presented with acute encephalopathy after ingesting lead-based, tangerine-scented gloss glaze used for making pottery. The substance is present on his teeth, lips, and nose. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

Lead poisoning affects multiple organ systems. Neurotoxicity may range from subtle personality changes to encephalopathy and cerebral edema. At the societal level, even small lead burdens are associated with statistically significant decreases in intelligence quotient. Motor neuropathy such as foot drop and wrist drop may be seen in adult patients, especially after occupational exposure. Microcytic anemia may occur, and basophilic stippling of the red cells may be seen. Hypertension and an acute nephropathy may occur. Abdominal pain may be described by patients, but unlike other heavy metal poisonings, constipation is more likely than diarrhea. Radiographic “lead lines,” bands of increased density on long bone metaphyses, may be seen in young children. These densities are not due to deposition of lead but rather increased calcium deposition.

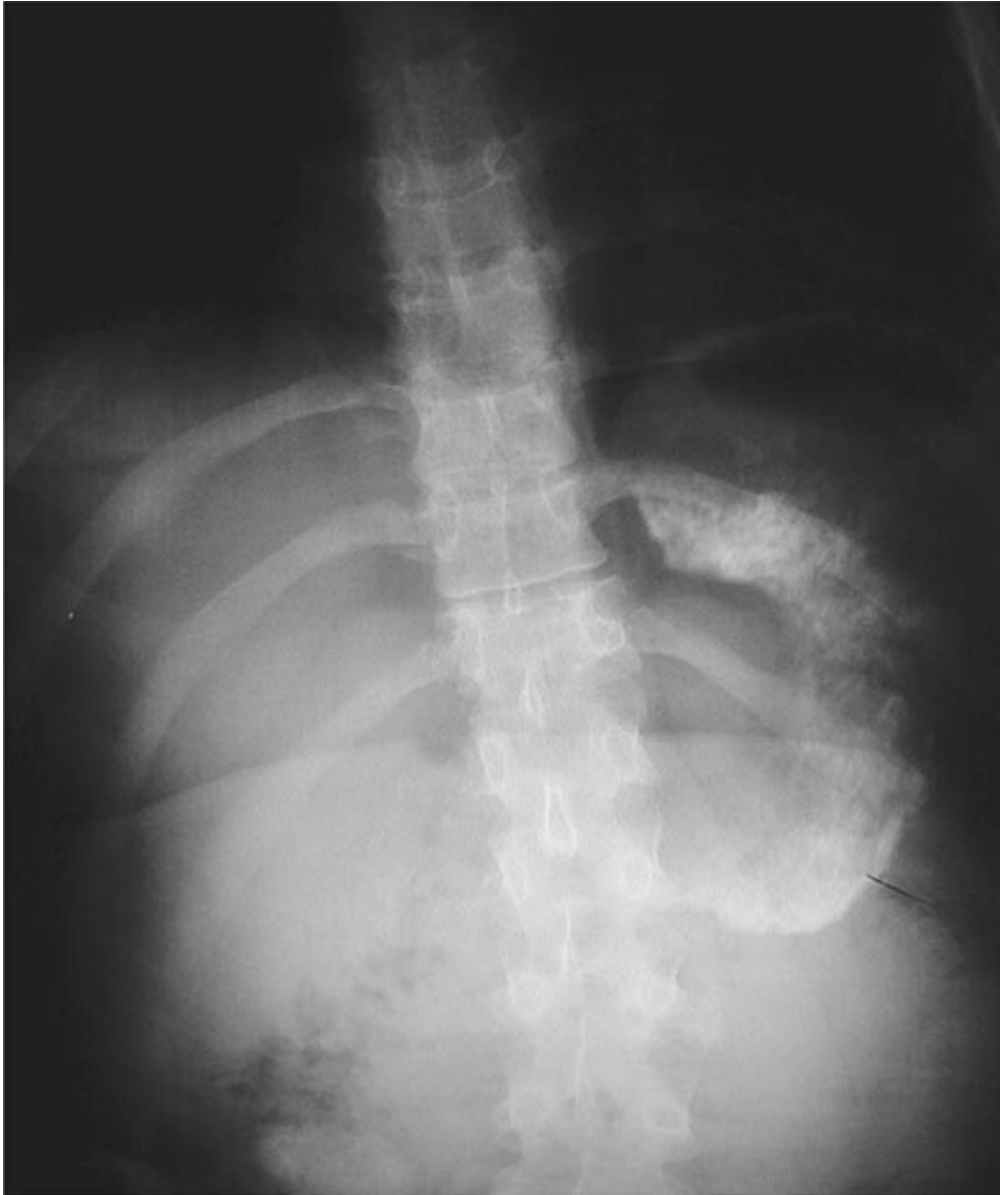


FIGURE 17.84 ■ Radiopaque Lead. Shown here is the abdominal x-ray of the patient in [Fig. 17.83](#). Lead is radiopaque when ingested. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)



FIGURE 17.85 ■ Lead Lines. Lead lines seen in a pediatric patient with chronic lead poisoning. The increased radiographic densities on the metaphyseal growth plates demonstrate radiologic growth retardation and increased calcium deposition. (Photo contributor: David Effron, MD.)

Management and Disposition

Whole blood lead level is the primary measure of lead exposure but is not usually available in real time. Radiographic studies may demonstrate radiopaque substances from ingested jewelry or paint chips in children. Lead encephalopathy must be aggressively managed. Dimercaprol (BAL) is administered intramuscularly and CaNa_2EDTA is later given intravenously. Chelation with oral succimer is currently recommended in asymptomatic children with levels between 45 and 70 $\mu\text{g}/\text{dL}$. Reducing the exposure in children is paramount to treatment, and the source of the lead may be elusive.

Pearls

1. One source of lead exposure in children is through the occupation of the parent. Workers in a lead dust environment will bring home the lead dust on their clothes and shoes.
2. Imported eye cosmetics with lead have been a source of pediatric exposures in certain ethnic groups.

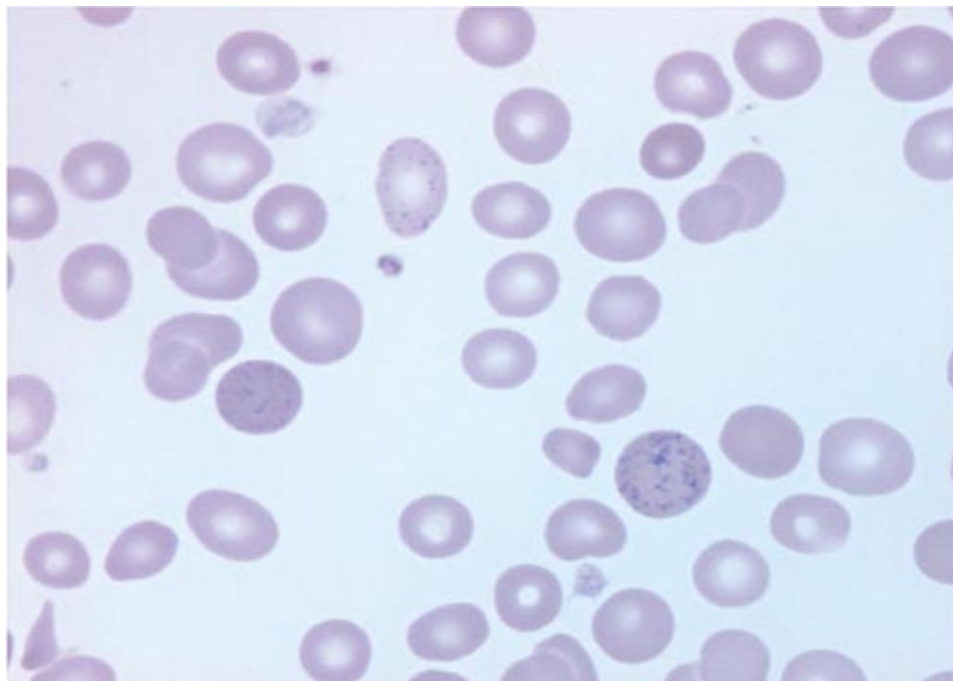


FIGURE 17.86 ■ Basophilic Stippling. Basophilic stippling along with a microcytic anemia may be seen in patients with chronic lead poisoning. (Photo contributor: Debbie Bennes, BS, MLT, ASCP.)

3. Azarcon and greta are lead-based remedies that are used to treat diarrhea

illnesses.

4. Adults usually require much higher blood lead levels than children before encephalopathy occurs.

MERCURY POISONING

Clinical Summary

Mercury occurs in three different forms (elemental, inorganic, and organic), each with its own clinical pattern of poisoning. Elemental mercury (“quicksilver”) is found in old thermometers and sphygmomanometers. Elemental mercury poisoning is associated with inhalation of volatilized mercurial ions, which may occur after vacuuming or heating. Manifestations include cough, fevers, chills, and dyspnea. Acute interstitial pneumonitis may occur and may progress to severe lung injury and death. Inorganic mercury poisoning usually occurs from the ingestion of the mercurial salts. Initial presentation is acute caustic gastroenteritis that may be hemorrhagic. Renal failure is a prominent finding in these patients. Organic mercury poisoning occurs from ingestion of short-chain alkyl mercurial compounds. Methylmercury distributes into brain tissue and causes neurologic disease such as ataxia, paresthesias, visual difficulties, movement disorders, and speech difficulties. Methylmercury is also a known teratogen.



FIGURE 17.87 ■ Subcutaneous Mercury. Lateral radiograph of an ankle demonstrating elemental mercury in the tissues. The patient had a barometer break into his skin. (Photo contributor: Saralyn R. Williams, MD.)

Management and Disposition



FIGURE 17.88 ■ Mercurial Emboli. Appearance of mercurial emboli in the pulmonary vascular tree on chest x-ray. Although this may be seen from intentional intravenous mercury injection, this patient absorbed the mercury intravenously following an accident involving multiple shattered thermometers. (Photo contributor: John Worrell, MD.)

After an ingestion of a mercurial substance, a radiograph may demonstrate radiopaque material in the GI tract. If elemental mercury was injected intravenously, mercurial emboli may be seen in the lungs. Local injection in the skin may demonstrate mercury deposition in the soft tissues. Ingestion of elemental mercury rarely results in significant absorption. For inhalational injury due to elemental mercury, respiratory support may be required. Ingestion of inorganic mercury may lead to early cardiovascular collapse as a result of the severe volume depletion. Fluid resuscitation and electrolyte management are critical. Chelation with dimercaprol (BAL) may be initiated early, and when the patient is able to take oral medications, the chelator may be switched to succimer. For organic mercury poisoning, oral succimer is the first-line agent.



FIGURE 17.89 ■ Mercury Salts. Mercury salts were used as topical antiseptics. Ingestion of inorganic mercury is caustic to the gastrointestinal tract and causes rapid renal failure. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)



FIGURE 17.90 ■ Mercury Tablets. These are tablets from the bottle in [Fig. 17.89](#). Although faded with age, the coffin-shaped tablets contain the word “poison” on one side and the “skull-and-crossbones” on the other. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

Pearls

1. Elemental mercury toxicity has occurred when it is heated and used to extract gold from jewelry.
2. “Mad as a hatter” is a phrase used to describe the delirium from anticholinergic poisoning; however, the phrase is derived from the erethism and hatter’s shakes from mercury exposure during the felt-hat manufacturing process in the late 19th and early 20th centuries.
3. Organic mercury is eliminated via the fecal route, so urine samples for methylmercury will not be reflective of the body burden.
4. BAL is suspended in peanut oil, and so can only be administered as an intramuscular injection and to non-peanut-sensitive individuals.

MUSHROOM INGESTION

Clinical Summary

While many species of mushrooms can cause toxicity when ingested, only a few contain amatoxins and account for most fatalities attributed to mushroom ingestion. Examples include *Amanita phalloides* (the “death cap”) and *Amanita ocreata* (the “destroying angel”) species. Amatoxin poisoning results in severe symptoms of gastroenteritis at least 6 to 24 hours after ingestion. Symptoms include nausea, vomiting, profuse watery diarrhea, and abdominal pain. GI symptoms may last 12 to 24 hours and are followed by a latent period of apparent improvement. This period is followed by a rise in liver enzymes and bilirubin. Fulminant hepatic failure and renal failure may become apparent.

The more common mushroom exposures include those with GI toxins. Ingestions result in acute nausea, vomiting, and diarrhea that occur within 2 hours of ingestion (eg, < 6 hours of ingestion). An example of this type of mushroom is the *Chlorophyllum molybdites*, which looks like a toasted marshmallow in the grass. Other types of mushroom poisonings include gyromitrin-containing mushrooms that are misidentified for the popular morels. Gyromitrin mushrooms cause status epilepticus that is responsive to high-dose pyridoxine. Coprine-containing mushrooms only cause toxicity when ethanol is co-ingested as they cause a disulfiram reaction.



FIGURE 17.91 ■ *Amanita phalloides*. The “death cap” produces amatoxins and accounts for most of the fatalities due to mushroom ingestion. (Photo contributor: Edward J. Otten, MD.)

Management and Disposition

For the GI toxin-containing mushrooms, management is supportive care. For the amatoxin-containing mushroom, administration of activated charcoal may be recommended depending on the time since ingestion. Specific interventions that may be helpful but are yet unproved include charcoal hemoperfusion, high-dose cimetidine, high-dose penicillin, high-dose ascorbic acid, silibinin, and *N*-acetylcysteine. Consultation with the local poison center is recommended.



FIGURE 17.92 ■ *Amanita ocreata*. These mushrooms were samples of *Amanita ocreata* provided by a family who had ingested them and subsequently developed significant hepatotoxicity. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)



FIGURE 17.93 ■ Morel Mushroom. Morels are considered a delicacy; however, inexperienced mushroom hunters may collect a “false morel,” which contains gyromitrin. Ingestion of gyromitrin results in status epilepticus that is responsive to intravenous pyridoxine. (Photo contributor: Saralyn R. Williams, MD.)

Pearls

1. A single “death cap” may contain enough amatoxin to kill an adult.
2. Cooking mushrooms does not substantially alter their toxicity.

3. Not all *Amanita* species of mushrooms cause hepatotoxicity when ingested. Some *Amanita* species are hallucinogens, and one causes renal failure.
4. Be wary of using the “6-hour rule” with mushroom scavengers who may have ingested multiple species of mushrooms.



FIGURE 17.94 ■ *Chlorophyllum molybdites*. This mushroom is ubiquitous and is a common culprit for mushroom toxicity. The patient typically presents with acute nausea and vomiting less than 2 hours after ingestion. (Photo contributor: Saralyn R. Williams, MD.)



FIGURE 17.95 ■ Coprinus Mushrooms. *Coprinus* mushroom cause toxicity when ethanol is co-ingested, resulting in a disulfiram-like reaction. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

CARDIAC GLYCOSIDE PLANT INGESTION

Clinical Summary

Cardiac glycosides (CGs) are found in the leaves, flowers, and seeds of *Nerium oleander* (common oleander), *Thevetia peruviana* (yellow oleander), *Digitalis purpurea* (foxglove), *Strophanthus gratus* (ouabain), *Convallaria majalis* (lily of the valley), *Apocynum cannabinum* (dogbane), *Urginea maritima* and *Urginea indica* (squill), and *Cheiranthus cheiri* (wallflower). If ingested, they produce

clinical findings similar to digoxin toxicity. The drinking of foxglove and oleander tea may be a cause of CG toxicity. Therapeutic effects occur from inhibition of the cardiac cell membrane sodium-potassium ATP pump, resulting in increased automaticity, decreased conduction through the atrioventricular node, and improved inotropy.

Toxic effects are an exaggeration of therapeutic effects. Bradydysrhythmias may result from impaired pacemaker function. Tachydysrhythmias may occur from increased automaticity.



FIGURE 17.96 ■ *Nerium oleander* (Common Oleander). A common decorative plant in subtropical climates often seen lining roads and highways. Flowers may be white, yellow, red, or purple. Drinking a tea brewed from the leaves of oleander results in severe cardiac glycoside poisoning. (Photo contributor: Saralyn R. Williams, MD.)



FIGURE 17.97 ■ *Digitalis purpurea* (Purple Foxglove). The flowers of foxglove are distinctive in their appearance. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.98 ■ *Convallaria majalis* (Lily of the Valley). While both beautiful and fragrant, lily of the valley contains multiple types of cardiac glycoside compounds. (Photo contributor: Saralyn R. Williams, MD.)

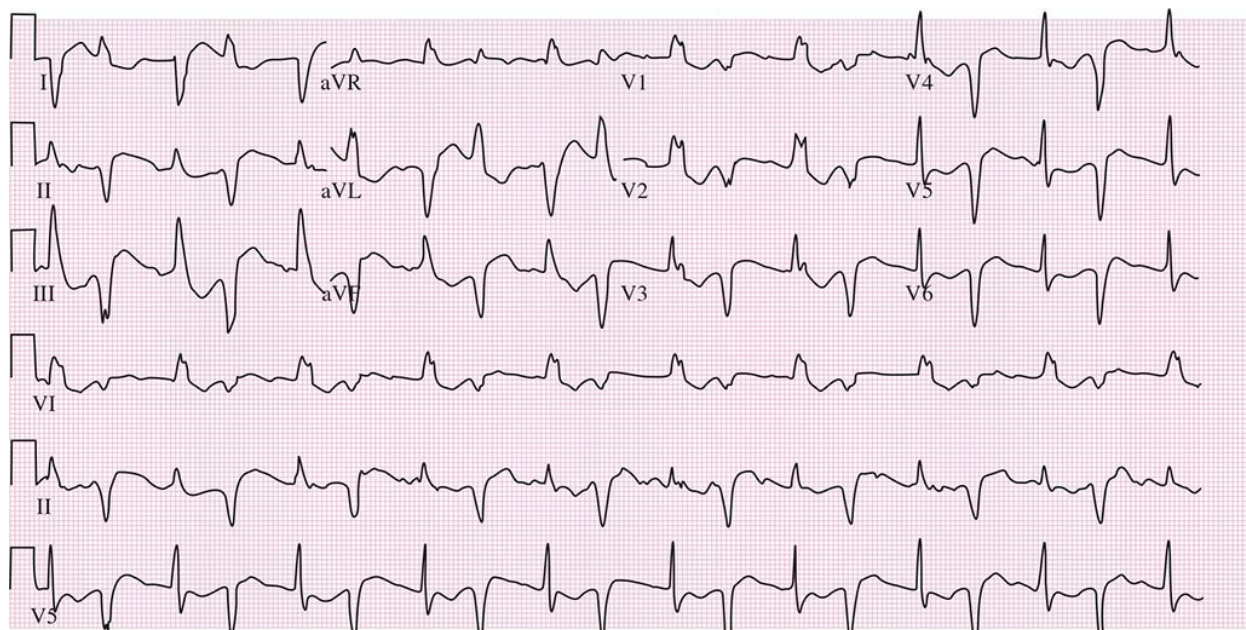


FIGURE 17.99 ■ Bidirectional Ventricular Tachycardia. An example of bidirectional ventricular tachycardia that occurred in a patient with digoxin toxicity. (Photo contributor: Binh Ly, MD.)

Nausea, vomiting, abdominal pain, confusion, depression, and fatigue may be present. Headaches, paresthesias, weakness, scotomas, and visual color disturbances (yellow halos around lights) may also occur.

Management and Disposition

Atropine may be initially given for bradydysrhythmias. Ventricular tachydysrhythmias have been treated with phenytoin or lidocaine when digoxin-specific Fab fragments are not available. Activated charcoal is the preferred method of decontamination. Cardioversion should be avoided in CG toxicity. Digoxin-specific Fab fragments are the treatment of choice for life-threatening dysrhythmias or CG-induced hyperkalemia.

Pearls

1. Treat CG overdose from plant exposure in the same way as an acute digoxin overdose. Higher doses of digoxin-specific Fab fragments may be required.
2. Calcium should be avoided in treating CG-associated hyperkalemia, as it may worsen ventricular dysrhythmias.
3. Dysrhythmias characterized by increased automaticity coupled with the presence of conduction disturbances are highly suggestive of cardiac

glycoside toxicity.

HOUSEPLANTS WITH CALCIUM OXALATE CRYSTALS

Clinical Summary

Many common houseplants such as dieffenbachia (dumb cane) and peace lily cause irritant effects when ingested owing to large amounts of insoluble oxalate crystals in its leaves. The oxalate crystals are highly irritating, and those who ingest the leaves experience painful burning of the lips, tongue, mouth, and esophagus. Marked swelling of the tongue, lips, and oropharynx can occur, and airway patency may become a major issue in managing these patients. Ocular exposures may occur as well, resulting in painful burning, erythema, and eyelid swelling. Fortunately, these calcium oxalate crystals are not absorbed, and hypocalcemia is not an issue.

Management and Disposition

Topical anesthetics are helpful in controlling severe pain from burning mucous membranes. Management is largely supportive, as the painful oral burns experienced with these exposures usually limit ingestion. As with any oropharyngeal burn, airway issues must be addressed. A period of observation is appropriate to make sure that airway compromise does not occur with continued swelling. If leaves are swallowed and the patients are symptomatic, GI consultation should be considered to assess the extent of esophageal injury. Gastrointestinal decontamination is usually not necessary.



FIGURE 17.100 ■ *Dieffenbachia*. *Dieffenbachia* is a common houseplant because of its colorful appearance and ease of indoor growth. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Performance of nasopharyngoscopy may be helpful in assessment of airway patency for more posterior burns.
2. Patient should be instructed not to swallow topical anesthetics, as toxicity may result with extensive use.



FIGURE 17.101 ■ Peace Lily. The attractive foliage and blooms make this a popular house plant; however, it is not a true lily. (Photo contributor: Saralyn R. Williams, MD.)

PLANTS WITH BELLADONNA ALKALOIDS

Clinical Summary

There are several species of plants that contain tropane alkaloids consisting of atropine, scopolamine, and hyoscyamine compounds. Given the effect on the pupils with mydriasis, these are known as the belladonna (beautiful woman) alkaloids. Jimson weed species (*Datura* spp.) are the most well-known plants that contain these alkaloids. Ingestion may occur through the drinking of tea made from the leaves or flowers of the plant or from eating the plant's seeds or leaves. Poisoned victims demonstrate an anticholinergic toxidrome resulting from the antimuscarinic receptor antagonistic effects of atropine and scopolamine. Patients may exhibit altered mental status, xerostomia, xeroderma, xerophthalmia, blurred vision, mydriasis, tachycardia, decreased bowel and bladder motility, and hyperthermia. Occasional presentations of unilateral mydriasis occur from topical exposure of the alkaloids (usually by touching the plant and then the eye).

Management and Disposition

Treatment initially consists of assessing the ABCs (airway, breathing, circulation) and stabilization measures. Hypotension resulting from tropane alkaloid ingestion usually responds to fluid boluses. Vasopressor agents are rarely necessary. Whole-bowel irrigation is contraindicated with intestinal ileus and must be considered with great caution due to decreased bowel motility. Physostigmine is a reversal agent, but may be better used as a diagnostic agent after consultation with the poison center. Severe agitation and psychosis may be treated with benzodiazepines and carefully administered, properly dosed physostigmine.



FIGURE 17.102 ■ **Jimsonweed.** Jimsonweed seed pod with dried seeds. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)



FIGURE 17.103 ■ Jimsonweed-Induced Xerostomia. A severe case of xerostomia from the antimuscarinic effects of Jimsonweed ingestion. Note the associated flushing of the patient's cheek. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.104 ■ Angel's Trumpet. Plants from the genus *Brugmansia* are cultivated for their beautiful pendulous flowers. Occasionally the leaves are brewed as a tea for intentional ingestion of the belladonna alkaloids. (Photo contributor: Saralyn R. Williams, MD.)

Pearls

1. Administering 1% pilocarpine eye drops does not reverse anticholinergic mydriasis.
2. Jimsonweed toxicity should be considered in the differential diagnosis of children and adolescents presenting with acute altered mental status, especially when accompanied by signs of anticholinergic toxicity after eating seeds.

PEYOTE INGESTION

Clinical Summary

Peyote (*Lophophora williamsii*) is a cactus plant found primarily in the southwestern United States. The cactus contains a significant amount of mescaline, a potent hallucinogen with structural similarities to norepinephrine. Peyote buttons and seeds are frequently ingested for recreational use but are also used in the religious ceremonies of some Native American groups. Toxicity of peyote is generally mild and self-limited, but hypotension and respiratory depression can occur. Mescaline induces some sympathomimetic effects due to its similarity to norepinephrine; marked visual hallucinations and a sense of depersonalization follow. These effects are often accompanied by unpleasant GI symptoms such as severe nausea and vomiting. Full recovery from these symptoms usually occurs within a few hours.

Management and Disposition

Treatment of peyote ingestion is largely supportive; severe toxic effects are uncommon. Marked agitation may be managed with benzodiazepines.



FIGURE 17.105 ■ Peyote Cactus Patch. Patch of peyote crowns growing in the desert. (Photo contributor: Martin Terry, PhD.)

Pearls

1. An individual peyote button contains about 45 mg of mescaline; a mescaline dose of 5 mg/kg usually produces psycho-tropic effects.
2. Botulism poisoning has been reported from the ingestion of dried peyote buttons.



FIGURE 17.106 ■ Peyote Cactus. An individual crown of peyote. (Photo contributor: Martin Terry, PhD.)



FIGURE 17.107 ■ Peyote Button. This desiccated button of peyote is the form that is ingested for recreational or religious purposes. (Photo contributor: Martin Terry, PhD.)

TOXALBUMIN INGESTION

Clinical Summary

The jequirity pea (*Abrus precatorius*) and castor bean (*Ricinus communis*) belong to a family of poisonous plants that contain toxalbumins. The chief toxin of the jequirity pea is abrin, which is structurally very similar to the toxin ricin of the castor bean. Ingestion of jequirity peas and castor beans rarely results in toxicity, as most of the plant toxin is concentrated within the hard shell of the seeds. However, when these seeds are chewed or the shell is digested, symptoms of severe gastroenteritis follow within 1 to 3 days. Nausea, vomiting, abdominal pain, and diarrhea are common but, in severe cases, may be accompanied by hemorrhagic gastritis and hematemesis, seizures, arrhythmias, marked dehydration, CNS depression, and even death. Unfortunately, because of the colorful attractive nature of jequirity peas and castor beans, most cases of ingestion occur in the pediatric age group. Because of the very high potency of these toxins, they are occasionally used for homicidal purposes, and growing concern exists for their potential utilization as an agent of bioterrorism.

Management and Disposition

Treatment of jequirity pea and castor bean ingestions is largely supportive, as there is no specific antidote for abrin or ricin. Gastric decontamination may be considered and may include activated charcoal and whole-bowel irrigation. In asymptomatic patients, decontamination, careful observation, and close follow-up are adequate. With symptoms of toxicity, however, admission is recommended, as the potential for marked clinical worsening is present.



FIGURE 17.108 ■ Castor Bean Plant. The castor bean plant is large and leafy; it may reach a height of 10 to 12 ft. (Photo contributor: Alex Wilson.)

Pearls

1. Most jequirity pea and castor bean ingestions are benign, as the vast majority of toxin resides within the undigested shell of the plant.
2. The toxalbumins abrin and ricin are structurally similar to botulinum toxin, cholera toxin, diphtheria toxin, and insulin.
3. Severe allergic reactions with anaphylaxis have been reported with handling of the seeds of castor bean and are also seen among workers in factories where castor oil is produced.
4. The castor bean plant is commercially cultivated as a source of castor oil. Such oil has been used for centuries as a purgative and as a lubricant for machines.



FIGURE 17.109 ■ Castor Bean. Typical appearance of the castor bean. (Photo contributor: Alex Wilson.)



FIGURE 17.110 ■ Jequirity Pea. Jequirity peas are also known as rosary peas, Indian beans, Buddhist's beads, crab's eyes, and prayer beads. They are about 5 mm in diameter and have a colorful glossy shell, usually red with a black center, although black and white may also be seen. (Photo contributor: Kevin J. Knoop, MD, MS.)

POKEWEED

Clinical Summary

Pokeweed (*Phytolacca americana*) is one of the most common ingested plants and contains a toxin called phytolaccotoxin. Pokeweed leaves are sometimes occasionally eaten as a vegetable (poke salad) after parboiling the leaves. If the leaves or unripe berries are eaten raw, acute onset of gastroenteritis occurs within a few hours of ingestion. Bradycardia, heart blocks, and hypotension have been reported as well.

Accidental ingestion of the ripe berries is a common exposure in young children who may mistake the berries for blueberries. Typical purple staining of the hands and the mouth commonly occurs.

Management and Disposition

Care is primarily supportive for symptomatic patients. Intravenous fluids should be administered, and antiemetics may be given. Patients who have no symptoms within a few hours of ingestion may be discharged to home. Children who ingest the berries usually demonstrate a limited amount of toxicity and may be discharged with reassurance.

Pearls

1. Phytolaccotoxin may cause a lymphocytosis a few days after ingestion. There is little clinical significance to this laboratory effect.
2. Purple staining of the hands, perioral area, or clothing may be a physical examination clue that pokeweed ingestion has occurred when history is lacking.



FIGURE 17.111 ■ Pokeweed Plant. This is the typical appearance of a tall pokeweed plant with immature white berries and mature purple berries. (Photo contributor: Saralyn R. Williams, MD.)



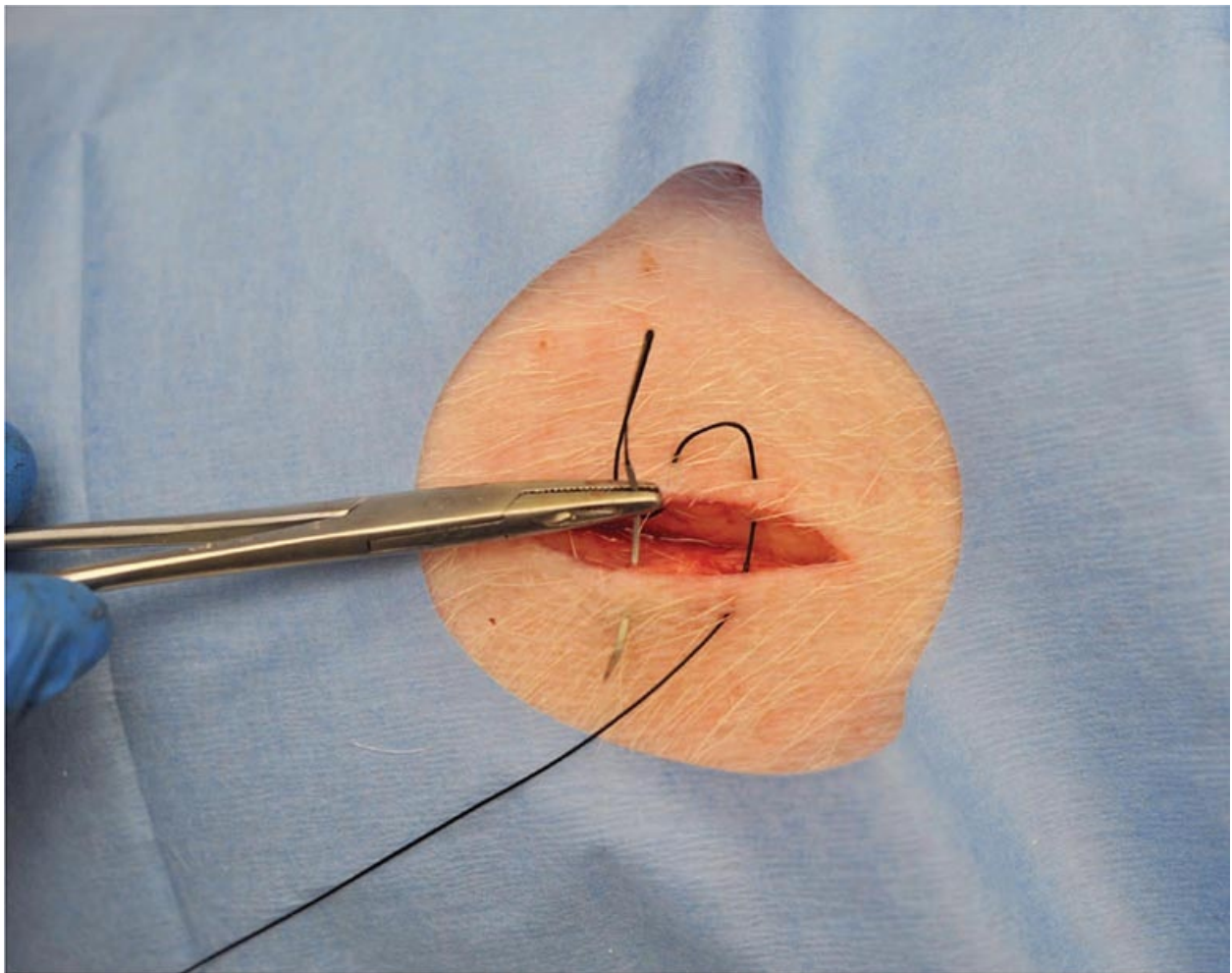
FIGURE 17.112 ■ Pokeweed Berries. This is a close-up of the mature purple pokeweed berries; these may be easily mistaken for blueberries given their similar appearance. (Photo contributor: Saralyn R. Williams, MD.)

The authors thank and acknowledge Matthew D. Sztajnkrzyer, MD, PhD, FACEP, for his contributions to prior editions of this chapter.

Chapter 18

WOUNDS AND SOFT-TISSUE INJURIES

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Michael L. Juliano
Dana Woodhall



Horizontal Mattress Suture. (Photo contributor: Michael L. Juliano, MD.)

WOUND CLEANING AND IRRIGATION

Clinical Summary

The goals of minor wound care are to achieve optimal wound aesthetics and infection prevention. For most uncomplicated wounds, irrigation is the most effective means of reducing bacterial count. However, debridement may be necessary in contaminated wounds since devitalized tissue may impair the wound's ability to resist infection.

Management and Disposition

Preliminary wound management begins with assessment, adequate hemostasis, foreign-body removal, and irrigation. Provide adequate analgesia prior to wound cleansing. In simple well-vascularized wounds, tap water is as effective as normal saline or sterile water. In contaminated wounds, povidone-iodine diluted 1:10 with normal saline may help with disinfection. Bacteriostatic solutions, such as nonionic surfactant cleaner, may also reduce bacterial inoculum. Solutions containing ionic detergents (eg, Betadine surgical scrub) should not be used because they are toxic to wound tissue. If necessary, wound scrubbing should be done gently to avoid damaging viable tissue.

Irrigation is the most effective means of reducing bacterial inoculum; 500 to 1000 mL of fluid or 60 mL/cm of wound length is adequate for most uncomplicated wounds. The recommended irrigation pressure of 5 to 8 lb per square inch (PSI) can be accomplished by attaching an 18- or 19-gauge intravenous (IV) catheter sheath, or a commercially available splash shield, to a 20- or 30-mL syringe. A bulb system is suboptimal; it generates only 0.5 to 1 PSI. Debris that cannot be irrigated can be scrubbed or sharply debrided. The tissue should appear pink and viable; a scant amount of fresh bleeding indicates good vascular supply. High-pressure irrigation (≥ 25 PSI) may be necessary in highly contaminated or complicated wounds requiring operating room washout. However, it offers no advantage for routine wounds.



FIGURE 18.1 ■ High-Pressure Irrigation Devices. Ideal pressure for routine wound irrigation is 5 to 8 PSI. This can be achieved with a 30-mL syringe attached to an 18- or 19-gauge IV catheter sheath (top) or splash shield (bottom) (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

Pearls

1. Universal precautions, including gloves and face shield, should always be observed.
2. Antibiotics are no substitute for thorough wound cleansing and irrigation.
3. Shaving the eyebrow for wound repair is contraindicated due to the unpredictable pattern of hair regeneration.
4. Soaking may loosen debris and coagulated blood but is not a substitute for irrigation.



FIGURE 18.2 ■ Wound Irrigation. After adequate anesthesia, a wound is thoroughly irrigated. Even with the shield, there can be significant splatter and potential for body fluid exposure. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

WOUND ASSESSMENT

Clinical Summary

Proper wound assessment and preparation are essential to management. Consider the age and mechanism of injury; contamination or foreign-body risk; risk to the nerve, blood vessel, and tendon; and tetanus status. Identify comorbid conditions

that may alter healing.

Management and Disposition

Patient compliance is integral to achieving adequate wound exploration. The use of local or regional anesthesia is usually sufficient; however, procedural sedation may be required. Document neurovascular status prior to any anesthesia. Direct pressure is often the easiest way to achieve hemostasis. Other methods include the use of blood pressure cuff or tourniquet to achieve temporary hemostasis. Anesthetic solution containing epinephrine (1:100,000 dilution) may help constrict small vessels; however, caution should be exercised when using in areas of end arterial circulation (eg, fingers, nose, toes, ears, and penis).

While hemostats or retractors may help achieve exposure, care must be exercised to avoid damaging the dermis and vascular integrity. If exposure is not adequate despite hemostasis and separation, wound margins may be slightly extended with fine iris scissors or a scalpel. The wound is extended from one end, through the epidermis and dermis only, to avoid further injury to underlying structures. Once the superficial fascia has been exposed, it may be carefully and bluntly dissected using forceps or scissors.

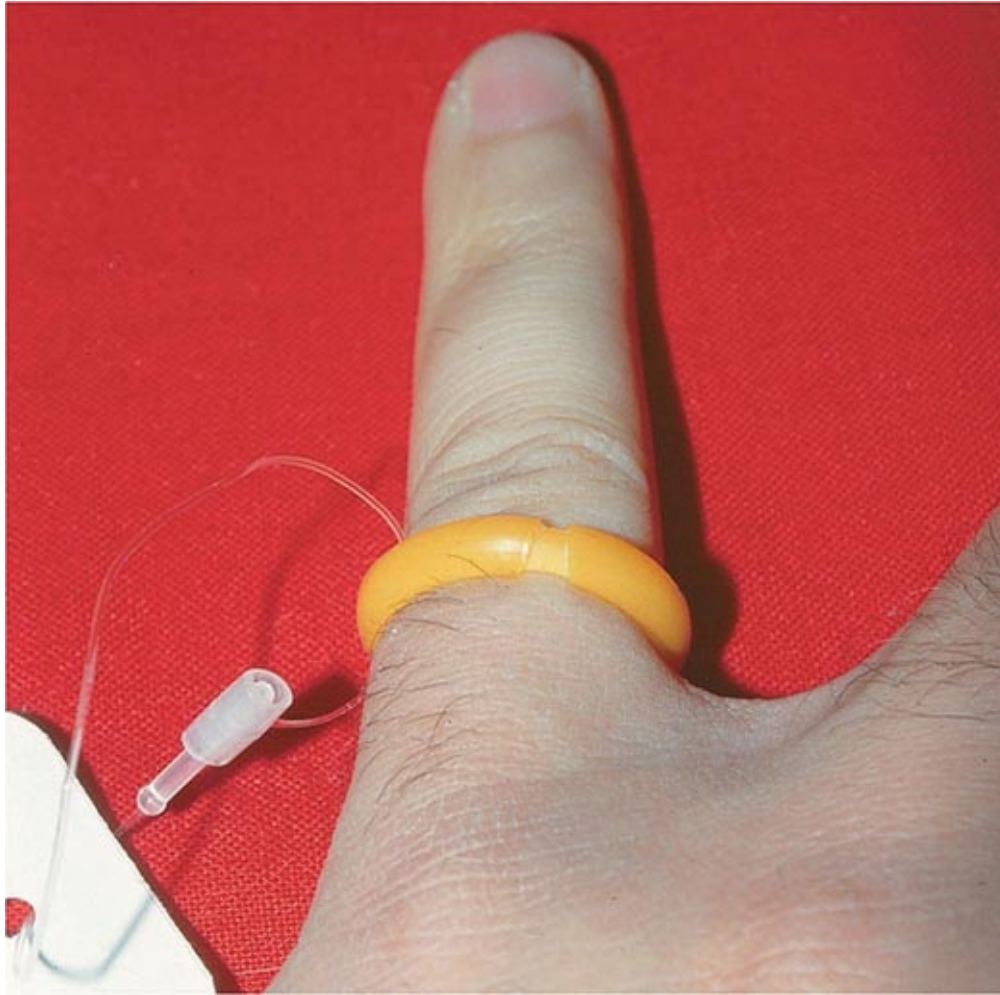


FIGURE 18.3 ■ Finger Ring Tourniquet. The ring tourniquet is an effective means of hemostasis. Removal after the procedure is important to prevent finger ischemia. Another effective method of hemostasis involves using a Penrose drain tightened with hemostats. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

Pearls

1. Never probe a wound blindly or blindly attempt to control bleeding with hemostats.
2. If epinephrine-induced tissue ischemia occurs, injection of phentolamine around the area may help restore flow.



FIGURE 18.4 ■ Epinephrine Injection. Although epinephrine can be used to help achieve hemostasis, it should be used with caution on areas with poor collateral blood supply. Lidocaine with epinephrine injected near the thenar eminence shows the pallor of finger and thenar eminence due to the vasoconstriction. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 18.5 ■ Wound Exploration. A blood pressure cuff is an alternative means to obtain hemostasis

before wound assessment. (Photo contributor: Alan B. Storrow, MD.)

WOUND FOREIGN BODIES

Clinical Summary

All foreign bodies can become a nidus for infection; reasonable attempts should be made to remove them. Radiographic evaluation or ultrasound (see related item and chapter) may assist in locating foreign bodies not directly visualized. Foreign bodies are characterized as being either *reactive* (eg, organic materials such as wood, bone, and soil) or *nonreactive* (eg, glass and metal).

Management and Disposition

Patients are often unaware that a foreign body is present. A high level of clinical suspicion should accompany any injury pattern at risk for foreign-body penetration, such as lacerations caused by broken glass, perioral injuries with loss of dentition, and injuries involving needles, nails, or splinters.

Suspicion of a retained foreign body mandates local wound exploration and the consideration of radiographic or ultra-sound evaluation. Most objects can be identified on plain radiographs. More specifically, approximately 90% of glass fragments greater than 2 mm in size can be identified using plain radiographs; fragments as small as 0.5 mm can be identified in 50% to 60% of cases. In situation where plain radio-graphs are poor, ultrasound may be considered.

Due to their increased risk for delayed infection and poor wound healing, reactive material must be removed. Nonreactive objects, however, may be left in place if reasonable effort to remove them has been unsuccessful and no potential for harm to a vital structure exists. Glass, however, has the potential for significant irritation and a removal attempt should be pursued.

Pearls

1. The base of the wound must be visualized as many foreign bodies hide there.
2. Common foreign bodies retained in hand wounds are wood splinters, glass fragments, metallic objects, and needles.
3. Missed retained foreign bodies are a common source of litigation.

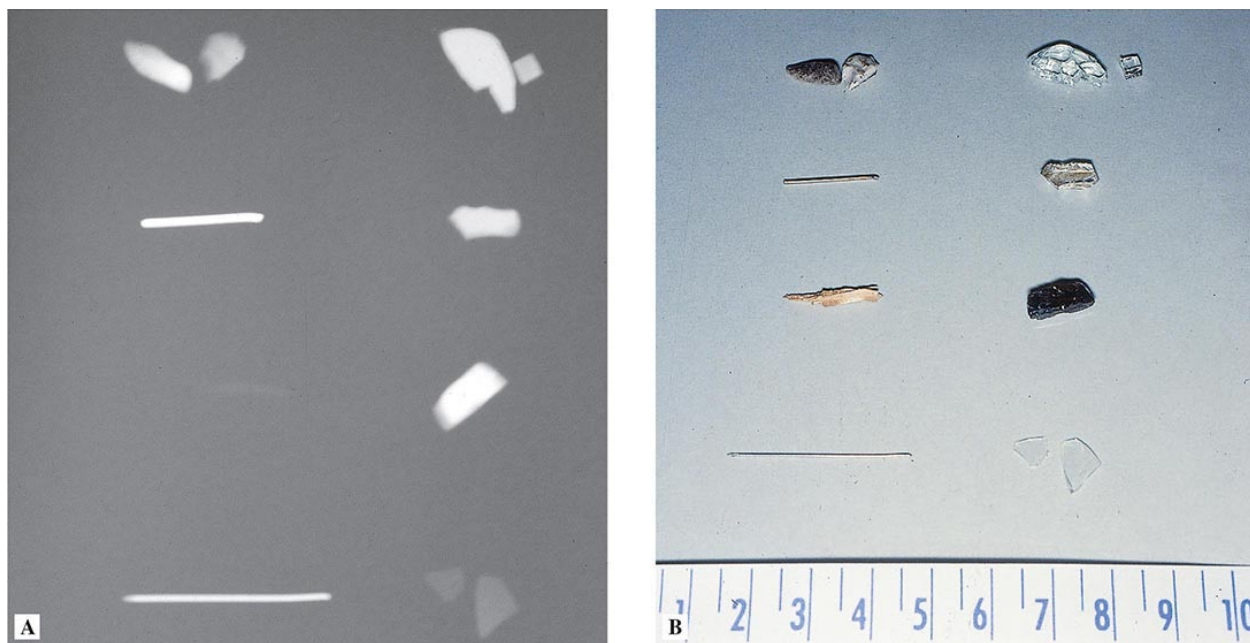


FIGURE 18.6 ■ Radiodensity of Common Foreign Bodies. The plain radiograph (A) demonstrates the radiodensity of common foreign bodies. Counterclockwise from top left: pebbles, paper clip fragment, wood splinter, hollow needle, light bulb glass, dark (“beer bottle”) glass, transparent glass, and automobile windshield glass. Note that, although faint, the wood splinter is visible on plain radiography. Ruler markings (B) for the photograph of the corresponding objects are in centimeters. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

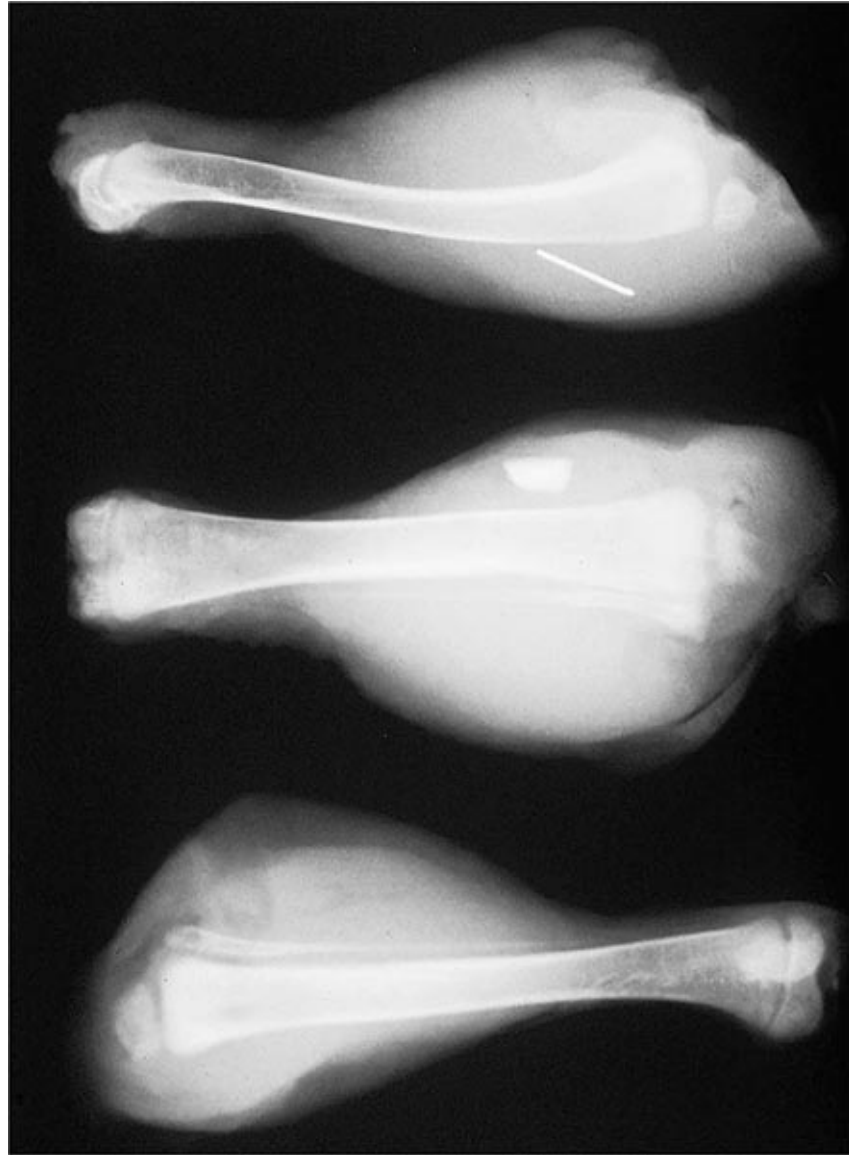


FIGURE 18.7 ■ Radiodensity of Common Foreign Bodies in Tissues. The paper clip, dark glass, and wood splinter (*top to bottom*) imaged in [Fig. 18.6](#) were inserted into chicken legs and radiographs taken. The wood splinter is no longer clearly visible within the soft tissue. For purposes of foreign-body localization, a minimum of two radiographic views at 90 degrees to one another are obtained, and the site of the foreign-body entry clearly marked. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

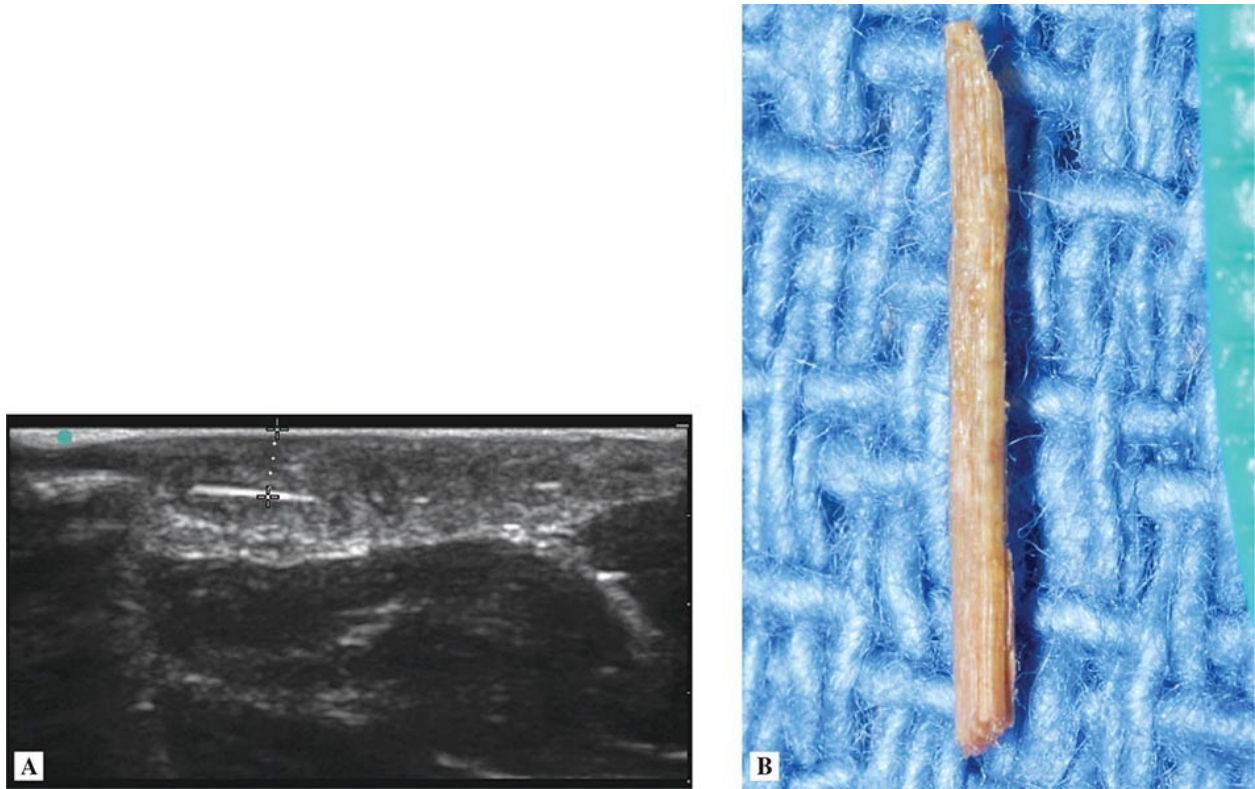


FIGURE 18.8 ■ Ultrasound Detection of Wood Foreign Body. Ultrasound is a good method to detect suspected wood foreign bodies (A). After removal (B). (Photo contributor: R. Jason Thurman, MD.)

TRAUMATIC WOUNDS

Clinical Summary

Traumatic surface wounds are caused by one of three mechanisms: shearing, tension, or compression. Such a division helps to guide management decisions involving infection risk and scar formation.

Shearing injuries are caused by sharp objects, such as glass shards or knives, which impart low-energy injury and minimal tissue destruction. Most uncomplicated shearing injuries (ie, those not involving neurovascular or anatomically important structures) are repaired primarily in the emergency department (ED). The risk of infection is low, and scar formation is typically cosmetically acceptable. Puncture wounds occur from sharp objects that pierce the skin and penetrate deeper tissues. Such wounds are at a higher risk for infection, foreign-body retention, and underlying structural injury.



FIGURE 18.9 ■ Uncomplicated Linear Laceration. A linear leg laceration. Given the depth and gaping, it can be sutured using a layered closure to remove surface tension at the wound edges and promote a more cosmetically acceptable outcome. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 18.10 ■ Complicated Linear Laceration. High-energy circular saw laceration with significant depth, tissue destruction, and likely neurovascular involvement. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 18.11 ■ Puncture Wound. A puncture wound to the foot with a contaminated garden instrument. Tetanus status must be carefully addressed in such an injury. A radiograph of the foot demonstrated no associated bony injuries. (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)

Tension or partial avulsion injuries occur when an object strikes the skin at a sharp angle creating a triangular flap. This results in potential vascular disruption, greater tissue destruction, and a higher risk for infection and tissue ischemia. During the repair, vascular supply to the flap must be meticulously preserved; otherwise, the flap may become ischemic.



FIGURE 18.12 ■ Partial Avulsion Injury. A typical partial avulsion laceration from a fall onto the edge of a staircase. Note the triangular “flap” in the upper left wound quadrant. Closure of partial avulsion injuries must be particularly meticulous to reduce any further compromise of the flap tip’s vascular supply (see Complex Wound Closures and Animal Bite Wounds). (Photo contributor: Alan B. Storrow, MD.)

Crush or compression injuries occur when a blunt object strikes tissue at a right angle, imparting a high degree of kinetic energy. This results in significant tissue destruction of the skin and its underlying supportive fascial layers. Crush injuries are typically ragged, with irregular wound edges and a complex laceration pattern. Despite meticulous wound care and careful primary closure, the resulting scars may be cosmetically poor.

Management and Disposition

Update the tetanus status of all patients requiring wound management ([Table 18.1](#)). Carefully document the time and functional/neurovascular status at initial evaluation, as this may change. Upon optimizing wound preparation, appropriate

closure tension must be achieved. If open fracture is suspected or confirmed, IV antibiotics are administered, and orthopedic consultation obtained. Repair of traumatic wounds depends on the depth, complexity, and location. Deep wounds are closed in layers or by using a vertical mattress technique to remove dead space and relieve tension. Superficial wounds may be repaired with staples, simple interrupted sutures, or running sutures. In certain circumstances, the use of adhesive skin closures or adhesive glues may be adequate.



FIGURE 18.13 ■ Lower Extremity Degloving Avulsion. A complex degloving injury after entanglement of the lower extremity in a rope while water-skiing. (Photo contributor: Alan B. Storrow, MD.)

TABLE 18.1 ■ GUIDE TO TETANUS PROPHYLAXIS IN ROUTINE WOUND MANAGEMENT

History of Adsorbed Tetanus Toxoid (Doses)	Clean Minor Wounds		All Other Wounds ^a	
	Tdap or Td ^b	TIG ^c	Tdap or Td ^b	TIG ^c
<3 or unknown	Yes	No	Yes	Yes
≥3 doses	No ^d	No	No ^e	No

^aSuch as (but not limited to) wounds contaminated with dirt, feces, soil, and saliva; puncture wounds; avulsions; and wounds resulting from missiles, crushing, burns, and frostbite.

^bFor children younger than 7 years of age, DTaP is recommended; if pertussis vaccine is contraindicated, DT is given. For persons 7 to 9 years of age, Td is recommended. For persons >10 years, Tdap is preferred to Td if the patient has never received Tdap and has no contraindication to pertussis vaccine. For persons 7 years of age or older, if Tdap is not available or not indicated because of age, Td is preferred to TT.

^cTIG is human tetanus immune globulin. Equine tetanus antitoxin should be used when TIG is not available. If only three doses of fluid toxoid have been received, a fourth dose of toxoid, preferably an adsorbed toxoid, should be given. Although licensed, fluid tetanus toxoid is rarely used.

^dYes, if it has been 10 years or longer since the last dose.

^eYes, if it has been 5 years or longer since the last dose. More frequent boosters are not needed and can accentuate side effects.

Source: Table courtesy of the Centers for Disease Control and Prevention. Please see www.cdc.gov for the most current information.



FIGURE 18.14 ■ Ring Injuries. Rings being caught and forced proximally are a common cause of finger degloving (A). Soft-tissue injury due to tourniquet effect of a ring in an elderly woman with diabetic neuropathy (B). (Photo contributors: Selim Suner, MD, MS [A]; Lawrence B. Stack, MD [B].)

Pearls

1. Simple, uncontaminated, and uncomplicated wounds caused by clean, sharp objects in otherwise healthy patients may undergo primary closure up to 18 hours from the time of injury with good cosmetic result.



FIGURE 18.15 ■ Hand Degloving Avulsion. This extensive degloving (**A**) was caused by an industrial accident and resulted in near-complete removal of the skin of the hand (**B**). (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 18.16 ■ Crush (Compression) Injury. Extensive crush injury, with degloving, resulting from an industrial accident. (Photo contributor: David Effron, MD.)

2. The vascular supply to a flap is often tenuous; improper closure may further compromise the tissue, especially at the tip. A repair using a corner stitch will help minimize further ischemia (see Complex Wound Closures and Animal Bite Wounds).
3. Crush injuries have an increased susceptibility to infection. Thorough cleansing, copious irrigation, and judicious debridement are required.

EAR LACERATIONS

Clinical Summary

The ear is composed of a poorly vascularized cartilaginous skeleton covered by tightly adherent skin. Given the paucity of subcutaneous tissue, injury that

results in hematoma formation can cause cartilage pressure necrosis. Repair involves completely covering the exposed cartilage and preventing hematoma formation.

Management and Disposition

Prior to repair, the area is examined for signs of acute hematoma formation or other associated traumatic injuries. Hemotympanum or Battle sign suggests the presence of a more serious closed head injury, especially basilar skull fracture. Blunt trauma may result in barotrauma resulting in tympanic membrane perforation. Exam can be facilitated by local anesthesia infiltration or, in the case of larger or more complex lacerations, a regional nerve block.

Simple lacerations through the earlobe or the helix can be repaired with interrupted 6-0 nonabsorbable monofilament sutures if the cartilage is not exposed. Simple lacerations that involve the cartilage are primarily repaired by ensuring complete coverage of the exposed cartilage by careful apposition of the overlying skin. The skin generally provides enough support, so sutures are not required for the cartilage itself. If the wound is sufficiently irregular and cartilage debridement becomes necessary to avoid undue wound tension, the debridement should be kept to a minimum.

A perichondral hematoma must be drained within 72 hours to prevent potential pressure necrosis, which can result in a “cauliflower” ear. Ear wounds are best dressed with a mastoid pressure dressing either primarily or after later hematoma drainage. Such a dressing reduces the chances for future hematoma formation and its complications. Ear sutures are removed in 4 to 5 days.

Pearls

1. Hematoma evacuation needs to be rechecked in 24 hours to evaluate for reaccumulation.
2. If cartilage has been exposed or a hematoma drained, anti-staphylococcal antibiotic coverage is recommended.
3. Complex lacerations and hematomas of the ear are best cared for in conjunction with a consultant from otorhinolaryngology (ENT) or plastic surgery.



FIGURE 18.17 ■ Ear Laceration. This patient sustained an uncomplicated, linear pinna laceration. Closure must cover all exposed cartilage. (Photo contributor: Alan B. Storrow, MD.)

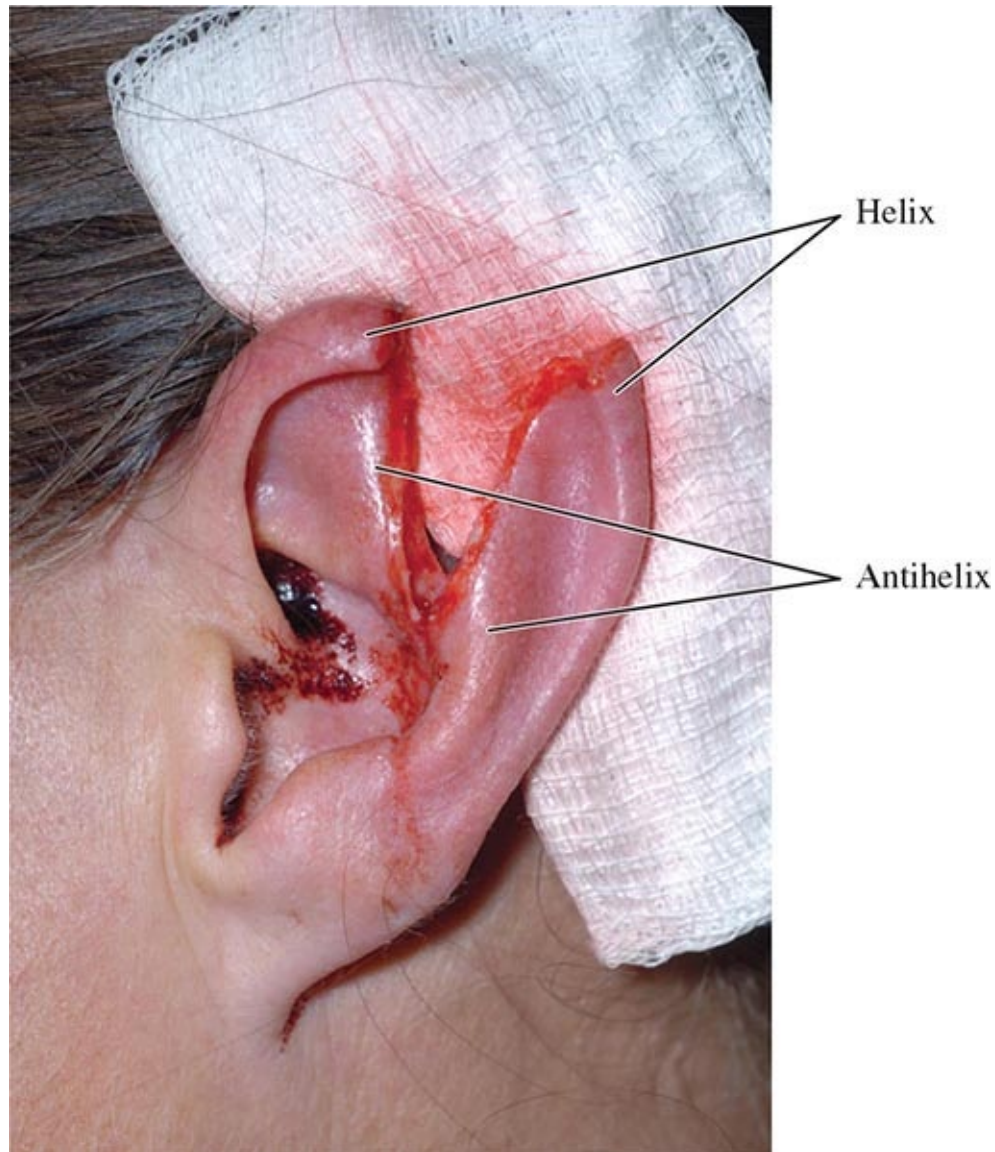


FIGURE 18.18 ■ Ear Laceration. Complex ear laceration through the helix down through the antihelix. (Photo contributor: Lawrence B. Stack, MD.)

LIP LACERATIONS

Clinical Summary

Lip lacerations may result in significant cosmetic defects if not properly repaired. The lip has two significant anatomic landmarks: the mucosal border (divides intraoral and external portions) and the vermilion border (separates the lip mucosa from facial skin). Meticulous alignment of the vermilion border and

its associated “white line” is the cornerstone of cosmetic repair. Lip anatomy may be distorted by the kinetic energy of the impact as well as the resultant edema surrounding the wound. Lacerations of the lip’s vermilion border may be partial or full thickness, compromising the underlying orbicularis oris.

Management and Disposition

Given the high bacterial content of the oral cavity, lip lacerations will not remain clean during repair. The goal of irrigation is to remove clotted blood and gross contaminants such as tooth fragments or dirt. If a fractured tooth is noted, the wound must be explored for fragments. If the tooth or fragment is unaccounted for, then a Panorex or soft-tissue radiograph of the face and a chest radiograph should be obtained. Anesthesia for laceration repair is best performed using either an infraorbital (upper lip) or mental (lower lip) nerve block since local infiltration often distorts the tissue and impairs proper alignment of the vermilion border.

If the vermilion border is violated by a superficial laceration, then the 1st suture, typically 6-0 in size, is placed at the border to reestablish anatomic relationships. Once alignment is judged adequate, simple interrupted sutures are used for completion. If the laceration extends within the oral cavity, absorbable 5-0 sutures are used to close the intraoral component.

With deep or “through and through” lacerations involving the orbicularis oris, the muscle layers are initially approximated with deep, usually 5-0, absorbable sutures. Once the muscle is approximated, the 1st skin suture is again placed at the vermilion border.

Sutures are removed in 3 to 5 days in children and 4 to 5 days in adults. The patient is advised to eat soft foods, not to apply excessive force to the suture line, and to rinse after eating to prevent the accumulation of food particles.

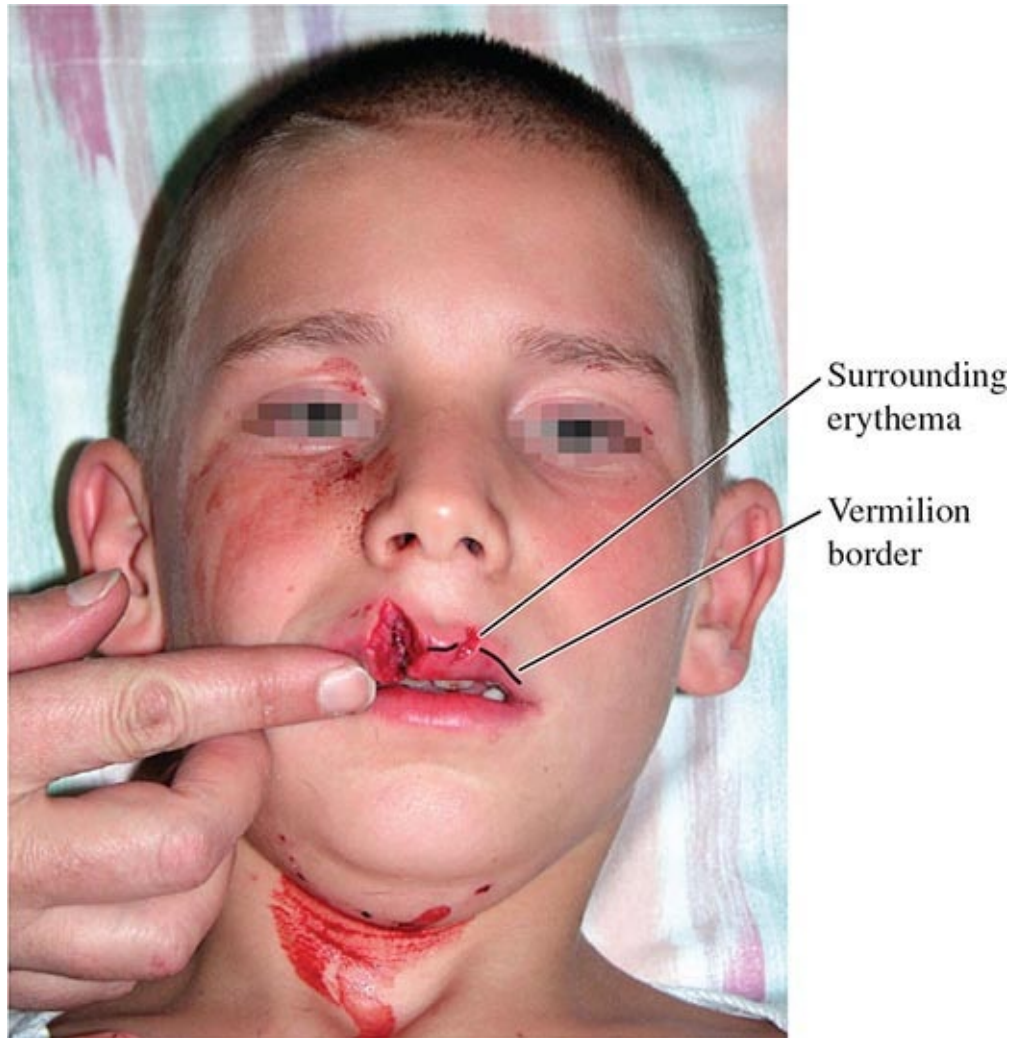


FIGURE 18.19 ■ Vermilion Border Laceration. A complex and a simple vermilion border laceration. (Photo contributor: David Efron, MD.)

Pearls

1. Misalignment of the vermilion border by as little as 1 mm may result in a cosmetically noticeable defect.
2. A marking pen may be used to identify landmarks prior to placing the sutures, as suturing itself causes some tissue edema, bleeding, and distortion.
3. Any patient with a lip laceration requires a thorough inspection of the oral cavity for associated trauma, including dental fractures, oral lacerations, and mandibular injuries.



FIGURE 18.20 ■ Angle Grinder Injury. Facial laceration through the vermilion boarder from a shattered angle grinder disk. (Photo contributor: Lawrence B. Stack, MD.)

TENDON LACERATIONS

Clinical Summary

Tendon injuries are often associated with hand or wrist lacerations. Accurate assessment requires documentation of both motor function and strength. Partial tendon ruptures, including near complete, may still result in normal function.

Management and Disposition

Prior to wound exploration, a thorough exam is performed to assess neurovascular and motor function. All individual flexor and extensor tendons are assessed, including deep and superficial. Abnormal resting posture of the involved extremity can also indicate tendon injury. Tendons are taken through a full range of motion, including re-creation of limb position at the time of insult, to detect injuries along the tendon length. Adequate exploration requires excellent hemostasis achieved through direct pressure or use of a blood pressure cuff or other tourniquet. Initial wound care should include irrigation, exploration

for foreign bodies, debridement, antibiotics, and tetanus prophylaxis if indicated.

Partial tendon lacerations are treated conservatively, with splinting in neutral position and follow-up. Isolated extensor tendon lacerations may be repaired in the ED with subsequent specialist follow-up. Flexor tendon lacerations generally require consultation with a hand surgeon or orthopedic surgeon.

Pearls

1. While extensor tendon repair may be accomplished by an emergency care physician with appropriate training and experience, flexor tendon lacerations are a challenging orthopedic problem and require referral.
2. Flexor tendons are weakest approximately 3 weeks after repair.
3. Inability to flex the distal phalanx with intact proximal phalanx extension suggests a flexor digitorum profundus disruption.

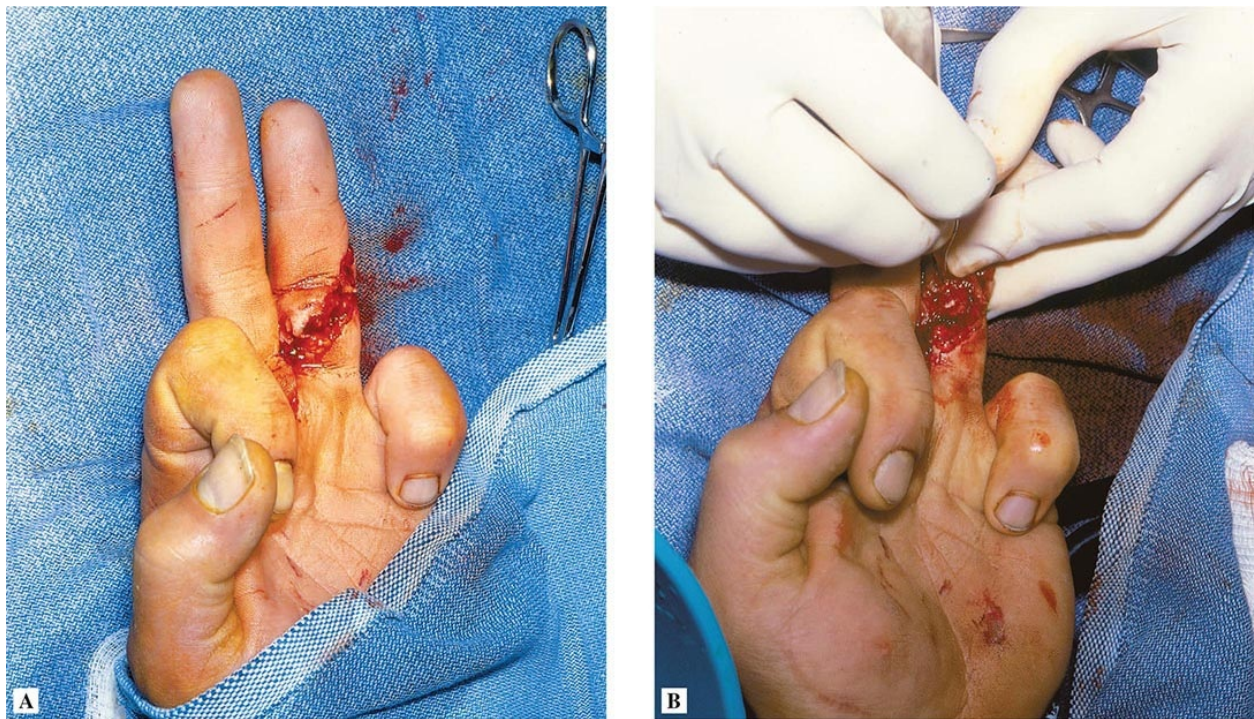


FIGURE 18.21 ■ Flexor Tendon Laceration. This patient presented after sustaining a laceration to his third and fourth digits (**A**). The injury was associated with an inability to flex these two digits. Wound exploration revealed the distal segment of the transected flexor tendon apparatus (**B**). (Photo contributor: Matthew D. Sztajnkrzyer, MD, PhD.)



FIGURE 18.22 ■ Extensor Tendon Lacerations. Note the laceration over the third and fourth metacarpals (**A**). Inability to extend the long fingers is evidence of complete extensor tendon disruption. Another extensor tendon laceration associated with inability to extend the fifth digit. (**B**). (Photo contributors: Selim Suner, MD, MS [A]; David Effron, MD [B].)



FIGURE 18.23 ■ Flexor Tendon Laceration. Wrist laceration and associated complete tendon rupture. (Photo contributor: David Effron, MD.)

ANIMAL BITE WOUNDS

Clinical Summary

Bite wounds (abrasions, lacerations, and punctures) are a frequent emergency care challenge, although most are minor. Dog bites account for the majority, followed by cats, humans, and, rarely, other animals (eg, raccoons, foxes, livestock, zoo animals).

Bite wounds are frequently contaminated with mixed aerobic and anaerobic bacteria. *Pasteurella multocida* (a gram-negative anaerobe) has been cultured from up to 80% of cat bites and 25% of dog bites. *Eikenella corrodens* has been recovered from human bites. *Capnocytophaga canimorsus* (formerly known as DF-2), a virulent organism that can be normal oral flora in dogs (16%) and cats (18%), can lead to sepsis, disseminated intravascular coagulation, gangrene, or death in susceptible individuals (eg, immunocompromised, asplenic, or diabetic). The hand is at highest risk for developing infection, while the face is the most infection resistant. Puncture wounds, especially from cats, are also prone to infection. Simple abrasions, regardless of animal, are unlikely to become infected with proper wound care.



FIGURE 18.24 ■ Dog Bite. Extensive facial wounds secondary to a dog bite. (Photo contributor: David Effron, MD.)

Management and Disposition

All wounds should be thoroughly cleaned and debrided. Radio-graphs should be obtained to exclude bony injury or retained dentition. Contusions and superficial abrasions can be treated with local wound care.



FIGURE 18.25 ■ Dog Bite. This patient sustained multiple avulsion injuries to her hand and forearm from a pit bull attack. (Photo contributor: Selim Suner, MD, MS.)

Recommendations vary regarding timing of wound closure. Closure of facial and head wounds can be performed up to 12 hours (and in some reports up to 24 hours). Puncture wounds (especially if caused by a cat), hand lacerations, or high-risk wounds (wounds > 12 hours or clinically infected) should be considered for delayed primary closure. While a linear incision over a puncture wound may facilitate cleaning and exploration, efficacy of this is questioned. Human bites to areas other than the face and head should generally be left open and considered for delayed primary closure. Hand injuries caused by human bites are left open and managed in consultation with a hand specialist (see [Chapter 11](#)). One should maintain a high index of suspicion for human bite in any laceration near the metacarpals.

All sutured bite wounds should be reevaluated by a health-care provider within 24 hours. Closed wounds that appear infected on reevaluation should be opened, irrigated, and allowed to close by secondary intention. Cyanoacrylate adhesives should never be used to close a bite wound.



FIGURE 18.26 ■ Cat Bite. Note the swelling and erythema to the index finger indicating infection, possibly with *P multocida* (a prominent and fastidious organism seen commonly with feline bites). (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 18.27 ■ Dog Bite and Repair. A thigh flap laceration (**A**) from a dog bite and repair (**B**). (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 18.28 ■ Crocodile Bite. Foot and leg tissue destruction and ankle dislocation from a crocodile bite. (Photo contributor: Michael Parsa, MD.)

Antibiotic recommendations for bite wounds differ widely. Antibiotics are not recommended for minor wounds. They are recommended for cat bites, hand wounds, and in persons with chronic diseases (eg, diabetes). Empiric therapy is started with broad-spectrum ampicillin-sulbactam, cefoxitin, or ceftriaxone. Alternatively, ciprofloxacin (or trimethoprim-sulfamethoxazole in children) and clindamycin can be used. Infection by *P multocida* classically becomes apparent within 24 hours and is marked by prominent pain, erythema, and swelling. Amoxicillin-clavulanate is the suggested regimen to cover polymicrobial infections and infections caused by *P multocida* or *C caninmorsus*. Cultures are not recommended for initial treatment but should be obtained in any wound worsening during antibiotic therapy. Tetanus immunization should be appropriately updated.

Rabies infection is rare in the United States; worldwide, dogs are the number one reservoir (see [Chapter 21](#)). Carnivorous animals (eg, raccoons, skunks, foxes, dogs, or bats) are more likely to be infected. If an animal is suspected as rabid, it should be detained and observed; occasionally this requires euthanasia and necropsy. Bats require special attention as many people do not recall being bitten. Healthcare professionals should have a low threshold for instituting rabies prophylaxis in persons with a bat exposure or in whom the animal is not available. Local animal control officers can be contacted for recommendations on appropriate rabies prophylaxis measures.

Pearls

1. Given cosmetic concerns and low infection risk, dog bites to the head and face

- can generally be sutured up to 24 hours after appropriate irrigation.
2. Bites that become infected within 24 hours, especially from cats, should be assumed to be caused by *P multocida*.

FISHHOOK INJURIES

Clinical Summary

Fishhook barbs often prevent backing the hook out of the puncture site. Several different methods have been described for removal.

Management and Disposition

Adequate anesthesia, usually local, is essential for removal. Procedural sedation may be needed if a child has a fishhook embedded in a sensitive area (eg, eyelid).

The method used to remove the hook depends primarily on the location of the barb relative to the skin surface and the body part. The most common removal technique is the “push-through and cut.” This is recommended when the tip of the fishhook is close to breaking through the skin surface after being embedded (see figures and videos). Care should be taken when performing this maneuver in the hand or face as pushing the fishhook forward may damage nearby structures.

Superficially embedded hooks or hooks with small barbs may be removed in a retrograde fashion, by exerting pressure on the fishhook shaft toward the barb and backing the hook out through the original site of penetration. This technique can be performed manually or with the use of a string (see videos).

Once fishhooks are removed, the wound should be cleaned, irrigated, and left open. Antibiotics are usually not necessary; however, treatment (doxycycline) for *Vibrio* species (especially *Vibrio parahaemolyticus*) should be considered in wounds contaminated with saltwater.

Pearls

1. Hooks embedded in cartilaginous structures, such as the ear or nose, are best managed with the push-through methods.

2. Hooks that penetrate joint spaces or bone should be managed in consultation with orthopedics.
3. Fishhooks that penetrate the globe of the eye are left in place, and emergent ophthalmologic consultation is obtained. The patient is placed in the semirecumbent position, and the globe is protected with an eye shield. Pressure patches are contraindicated, as they may extrude intraocular contents.

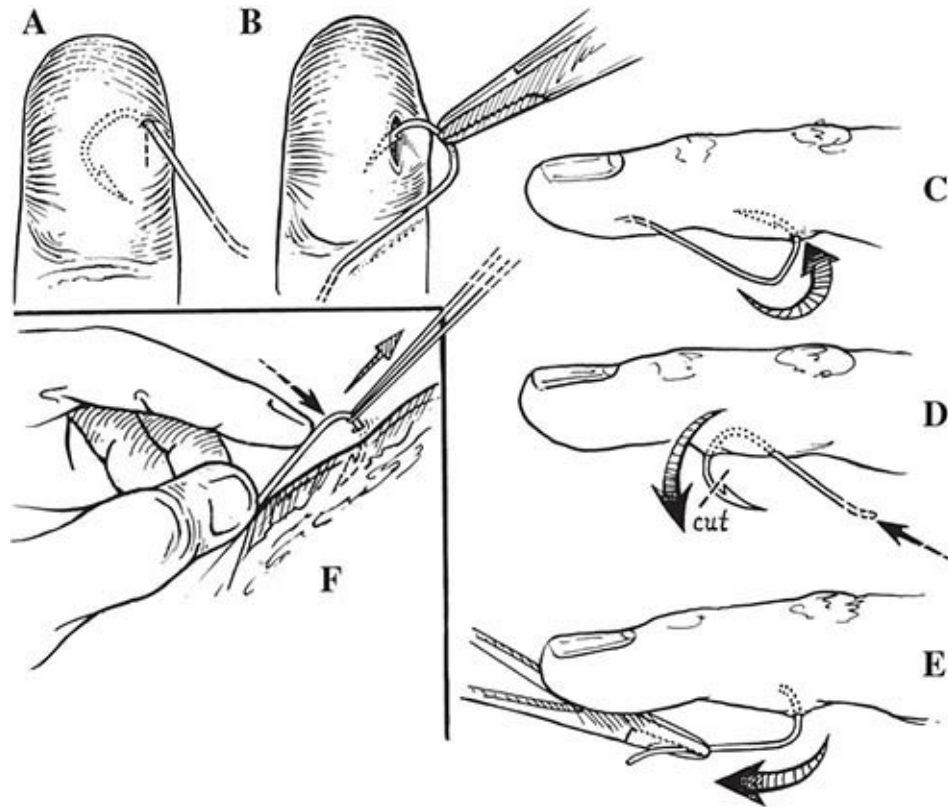


FIGURE 18.29 ■ Fishhook Removal. Hooks with small superficially embedded barbs may be carefully backed out through the original puncture site (**A** and **B**). This may require a small incision, made in line with the concavity of the hook curve. The push-through technique is useful for large barbs or those more deeply embedded. The hook is pushed out through the skin, the barb removed, and the remainder of the hook subsequently removed through the original penetration site (**C**, **D**, and **E**). The traction (string) technique provides an alternative for removing hooks with small barbs. While pressing down on the shaft of the hook, traction is applied with 0 silk or umbilical tape. A swift yank of the cord in the direction opposite the barb will dislodge the hook (**F**). Care is taken to warn bystanders of the potential for the fishhook to fly across the room.



FIGURE 18.30 ■ Multiple Pronged Fishhook. Care must be taken when removing this three-pronged hook due to the multiple sharp tips and potential for damaging surrounding structures. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 18.31 ■ Fishhook Injury and Removal. The push-through technique was used to remove this

hook. Use of a ring cutter proved unsuccessful; a bolt cutter was eventually required to remove the distal portion of this large hook. (Photo contributor: Alan B. Storrow, MD.)

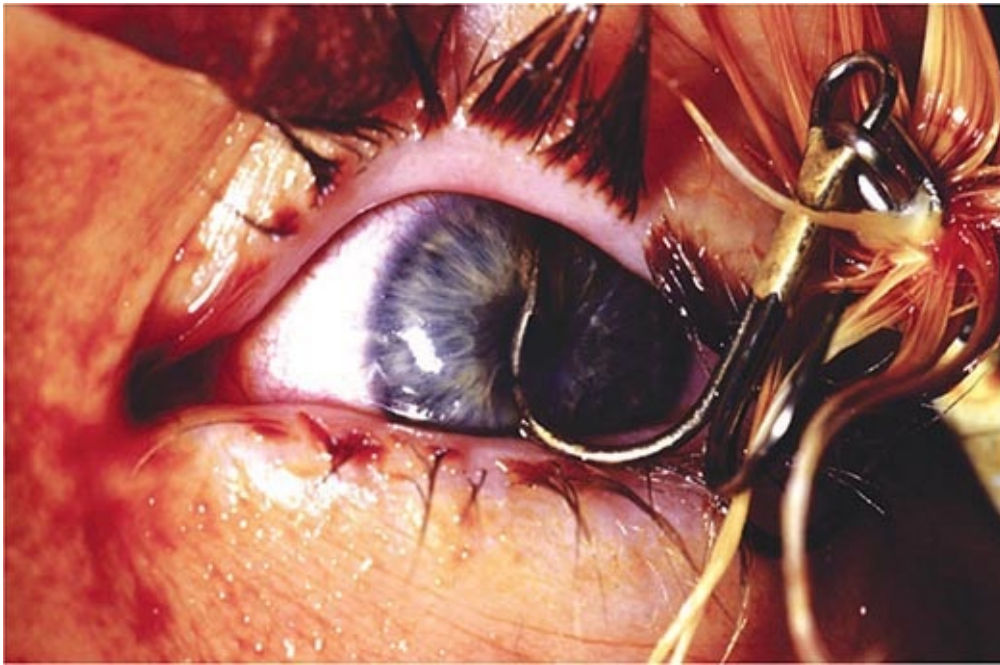


FIGURE 18.32 ■ Fishhook Penetrating Globe Injury. Intraoperative image of a treble hook from a fishing lure penetrating the cornea and barb seen in the anterior chamber. Operatively removed without complication. (Photo contributor: Lawrence B. Stack, MD.)

SIMPLE WOUND CLOSURES

Clinical Summary

Most lacerations are uncomplicated and can be repaired primarily. Complications (dehiscence, infection, improper healing, scarring) can be minimized by proper suturing techniques, including good wound edge approximation and decreasing wound tension. Wound edge eversion is also essential for proper healing as the edge will flatten with time.

Management and Disposition

Suture techniques must be individualized depending on physician's skill, laceration type, and location. Suture material, size, and duration before removal are determined by anatomic site ([Table 18.2](#)).

Deep interrupted absorbable sutures are used, if necessary, to reduce wound tension before superficial repair. Start the deep suture from the bottom of the wound, continue across the top (subdermally), and return to the bottom in finishing. This will leave the knot at the bottom of the wound, decreasing the chance suture material will exit at the surface. **Simple interrupted closures** involve single nonabsorbable sutures, each independently tied. The needle should penetrate the skin at 90 degrees and exit the other side of the wound at 90 degrees. **Staples** are a rapid means of closing linear wounds. They should not be used on the hands, feet, face, or over joints. Wound edge eversion is also critical to obtain the best outcome.

Running closure is a rapid technique using several bites along the length of a wound without tying individual knots. Knots are tied only at the beginning and end. **Liquid wound adhesives** (eg, cyanoacrylate) have advantages due to speed and lack of a repeat visit for suture removal. Anesthesia may not be required. Small linear lacerations are ideal. The wound must be dry and free of active bleeding for proper adhesion. Antibiotic ointment should not be applied as this will dissolve the adhesive, although elastic wound strips may be used on top for added closure tension.

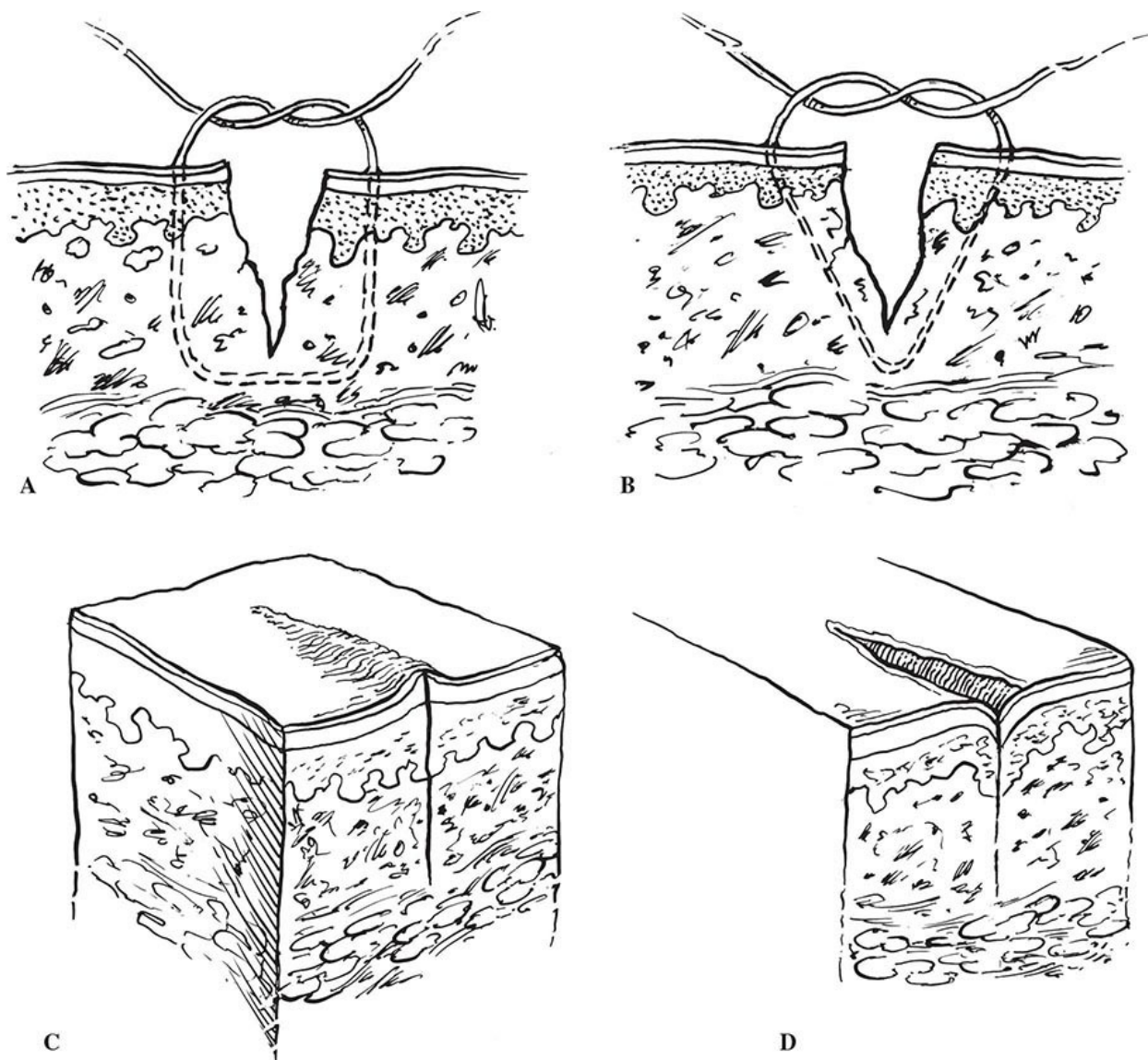


FIGURE 18.33 ■ Wound Edge Eversion. Eversion of wound edges is critical for optimal healing. For proper eversion, the needle point should enter the epidermis at a 90-degree angle, generating a square or bottle-shaped suture configuration (A). This results in a slight rise of the skin edges above the skin plane (B). Such eversion will flatten at the level of the skin plane during healing. Entry at a shallower angle (C) often leads to wound edge inversion, eventual contraction of the wound edges below the skin plane, and subsequent scar formation (D).

Elastic wound closure strips have the same advantages and indications as wound adhesives. They can be used alone or in conjunction with a tissue adhesive (benzoin resin) applied to the skin on either side of the wound. The edges will curl up over time and can be trimmed by the patient as needed, until they fall off in 2 to 3 weeks. They can also be used after suture removal to give the wound more time to gain tensile strength. Wound closure strips are not recommended in children as they tend to remove them prematurely.

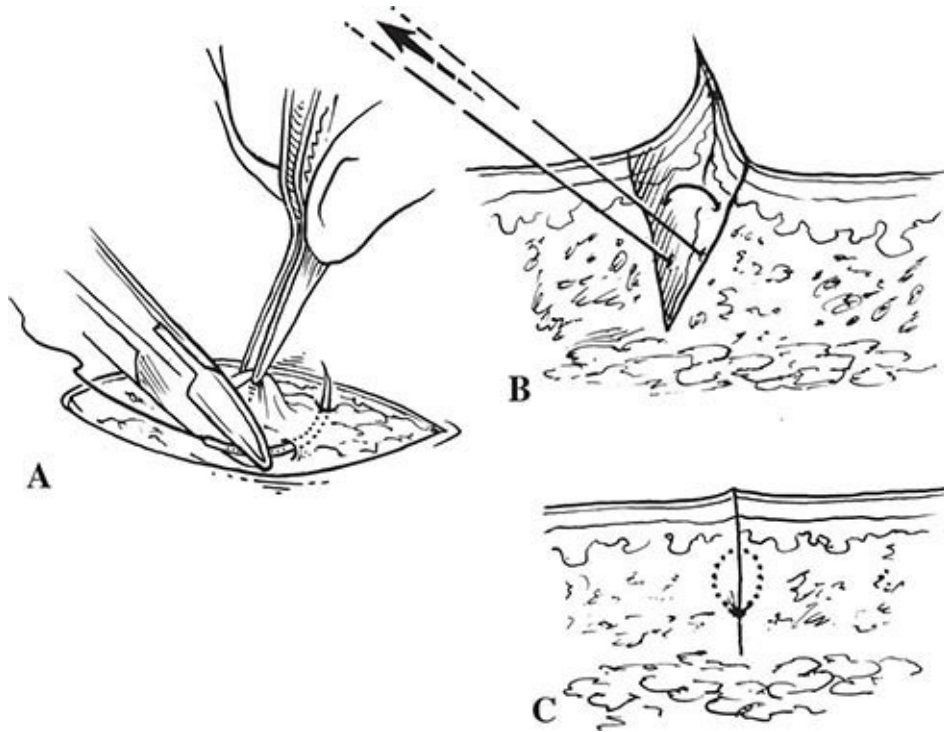


FIGURE 18.34 ■ Deep Sutures. Judicious placement of deep sutures allows approximation of the dermis, reduces tension on the wound edges, and may facilitate final superficial closure. The needle is driven from deep within the wound to a superficial level (**A**). On the opposite side of the wound, the needle is driven from superficial to deep (**B**). By having the leading and trailing suture come out on the deep and same side of the superficial cross suture, the knot is buried within the wound (**C**).

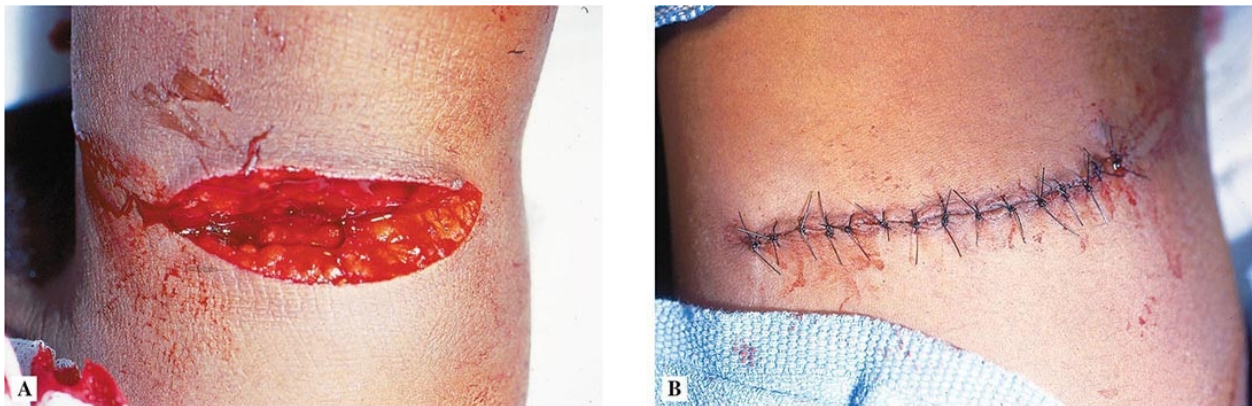


FIGURE 18.35 ■ Simple Interrupted Wound Closure. An uncomplicated linear laceration generated by a sharp object (**A**). For anesthesia and hemostasis, the wound edges are infiltrated with lidocaine-containing epinephrine. The wound is subsequently closed with simple interrupted sutures (**B**). Attention is paid to obtaining a degree of wound edge eversion. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 18.36 ■ Staple Closure. Meticulous care must be taken when using staples to properly approximate and evert wound edges. This can be facilitated using forceps during staple closure.

Pearls

1. The bites on both sides of a wound should be equidistant for both optimum wound healing and cosmetic outcome.
2. In gaping wounds, surface tension should be reduced with deep sutures.
3. Antibiotic ointment may be used to remove the adhesive from areas unintentionally glued together by cyanoacrylate.
4. Wound closure tapes (Steri-Strips) may be used over liquid wound adhesives to reduce tension.

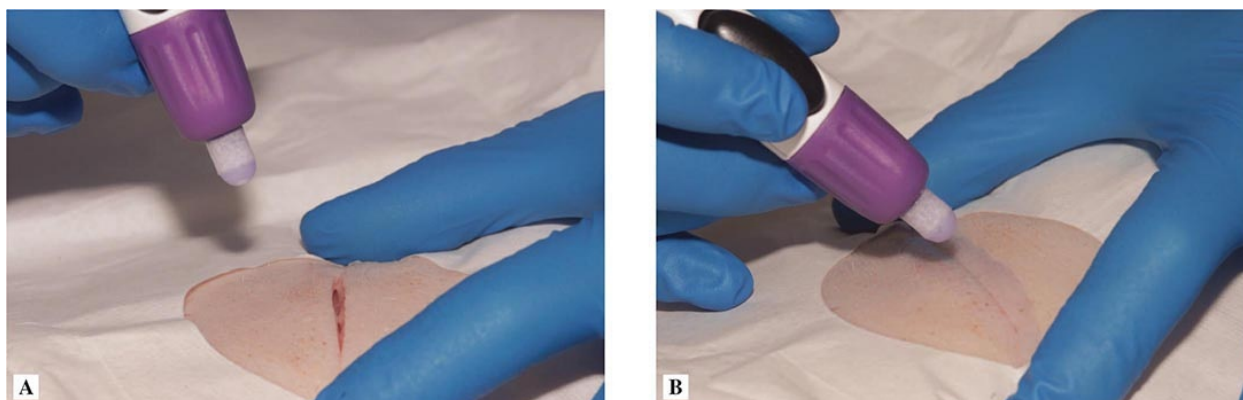


FIGURE 18.37 ■ Liquid Wound Adhesive Closure. Wounds that are ideal for this type of closure are small, linear, and hemostatic (A). Care must be taken to ensure the wound edges are correctly approximated before applying the adhesive (B). (Photo contributor: Michael L. Juliano, MD.)

TABLE 18.2 ■ SUTURE MATERIALS, SIZE, AND DURATION BY ANATOMIC SITE

Anatomic Site	Skin	Deep	Duration
Scalp	5-0, 4-0 monofilament	4-0 absorbable	6-8 days
Ear	6-0 monofilament	N/A	4-5 days
Eyelid	7-0, 6-0 monofilament	N/A	4-5 days
Eyebrow	6-0, 5-0 monofilament	5-0 absorbable	4-5 days
Nose	6-0 monofilament	5-0 absorbable	4-5 days
Lip	6-0 monofilament	5-0 absorbable	4-5 days
Oral mucosa	N/A ^a	5-0 absorbable	N/A
Face/forehead	6-0 monofilament	5-0 absorbable	4-5 days
Chest/abdomen	5-0, 4-0 monofilament	3-0 absorbable	8-10 days
Back	5-0, 4-0 monofilament	3-0 absorbable	12-14 days
Arm/leg	5-0, 4-0 monofilament	4-0 absorbable	8-10 days
Hand	5-0 monofilament	5-0 absorbable	8-10 days ^b
Extensor tendon	4-0 monofilament	N/A	N/A
Foot/sole	4-0, 3-0 monofilament	4-0 absorbable	12-14 days

^aNot applicable.

^bAdd 2 to 3 days for joint extensor surfaces.

Source: Adapted with permission from Trott AT. *Wounds and Lacerations: Emergency Care and Closure*. 2nd ed. St. Louis, MO: Mosby-Year Book; 1997.

COMPLEX WOUND CLOSURES

Clinical Summary

Horizontal and vertical mattress sutures, as well as the corner stitch (see figures for technique), may be used to manage wounds unable to be closed with simple techniques.

Management and Disposition

Vertical mattress suture is a useful technique for deep wounds. It reduces wound tension by acting as both a deep and superficial suture. **Horizontal mattress suture** is best used for wide, gaping wounds with a risk of increased wound tension after closure (eg, lacerations overlying a joint). **Corner stitch** is used to close triangular wounds or flaps. A simple interrupted suture cannot be placed to approximate the point of the flap due to a tenuous blood supply and increased chance of dehiscence. Once the corner is secured (see figures), simple sutures are used to repair the rest of the wound, with care taken to place the sutures far enough from the tip to optimize circulation.

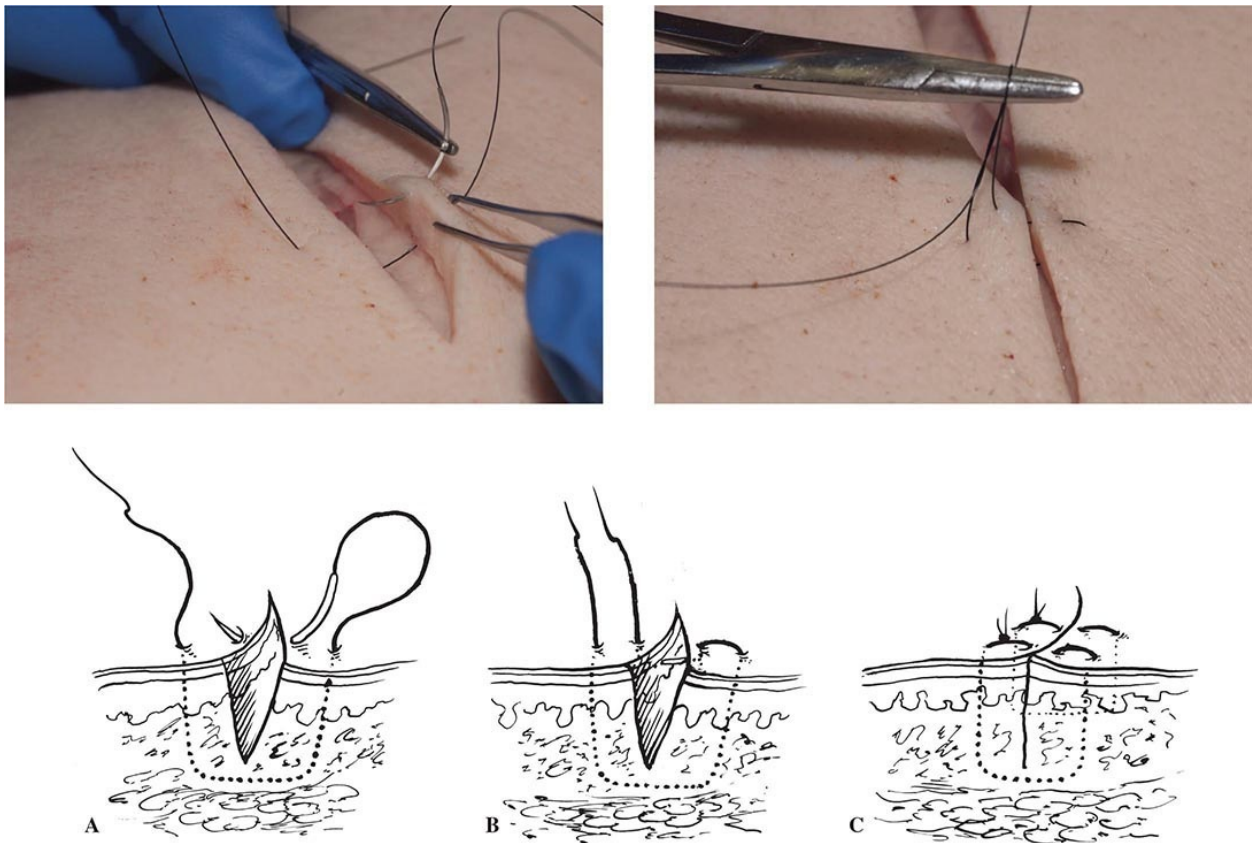


FIGURE 18.38 ■ Vertical Mattress Suture. The suture is placed by first taking a large deep bite of tissue approximately 1 cm away from the wound edge and exiting at the same location on the other side of the wound. A second small superficial bite is then performed in the reverse direction (A). When the bites are complete (B), tying results in nice apposition of the wound edges (C). This technique is especially useful in areas of lax skin, such as the elbow or dorsum of the hand. (Photo contributor: Michael L. Juliano, MD.)

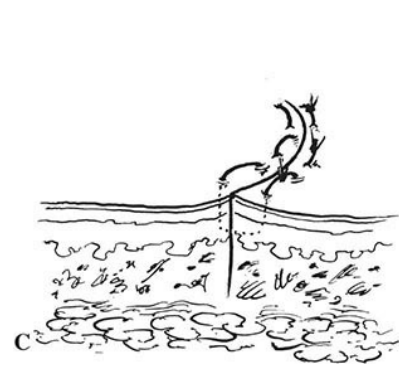
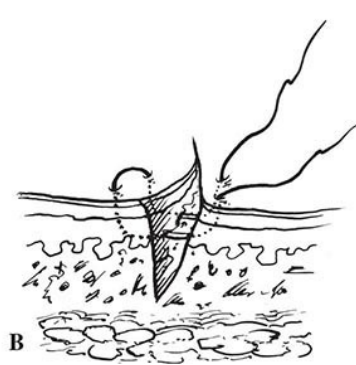
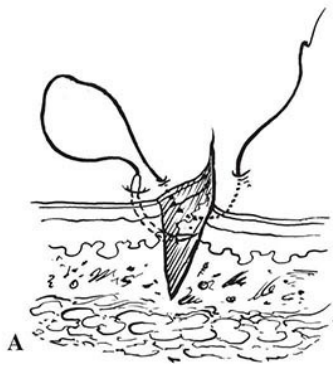
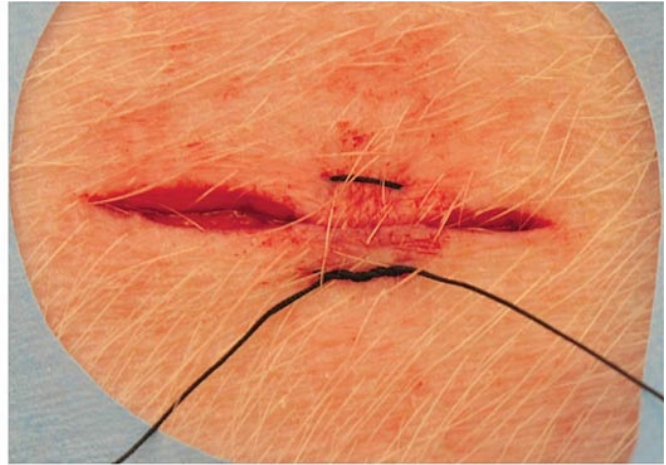
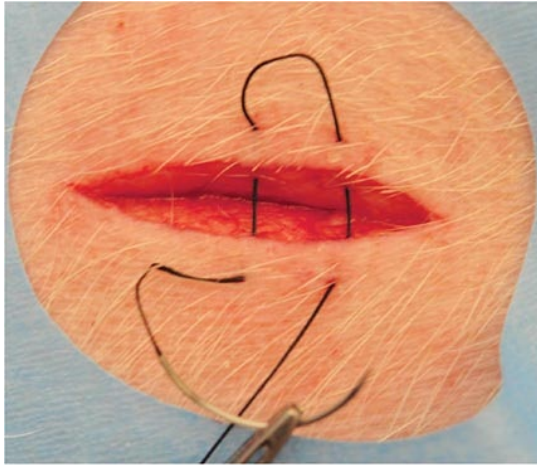


FIGURE 18.39 ■ Horizontal Mattress Suture. Useful in achieving wound edge eversion, the horizontal mattress suture begins with a standard suture throw. A second bite is taken approximately half a centimeter from the first exit (A) and brought through at the original starting edge, half a centimeter from the original entry point (B and C). (Photo contributor: Michael L. Juliano, MD.)

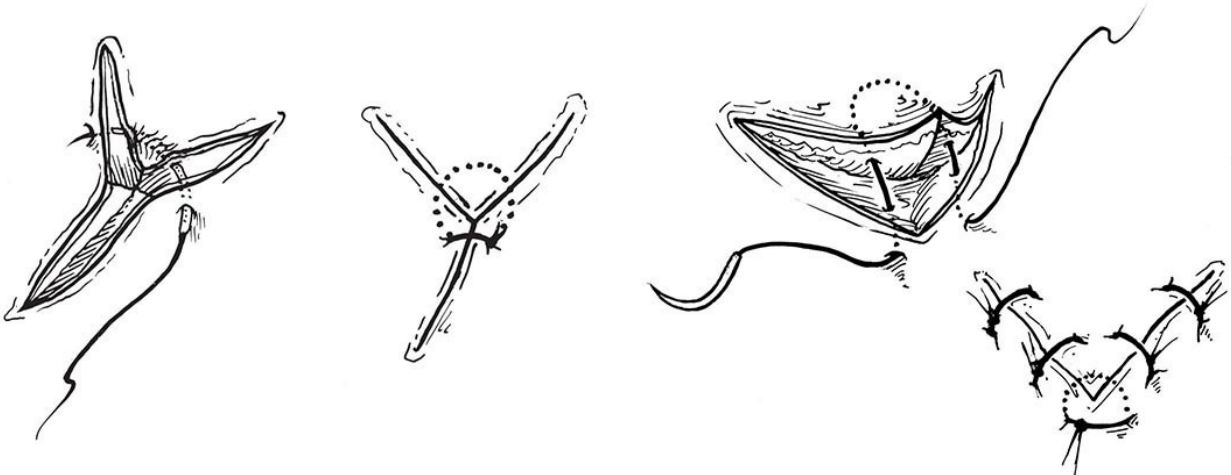


FIGURE 18.40 ■ Corner Stitch for Stellate Wounds. The corner stitch may also be used to close stellate lacerations.

Pearls

1. Utilization of a mattress suture can aid in wound edge ever-sion and tension reduction. These sutures are particularly useful in areas where the deep subcutaneous tissues are too fragile for deep sutures (eg, over a joint or shin).
2. A single corner stitch may be used to close several corners of a stellate wound.

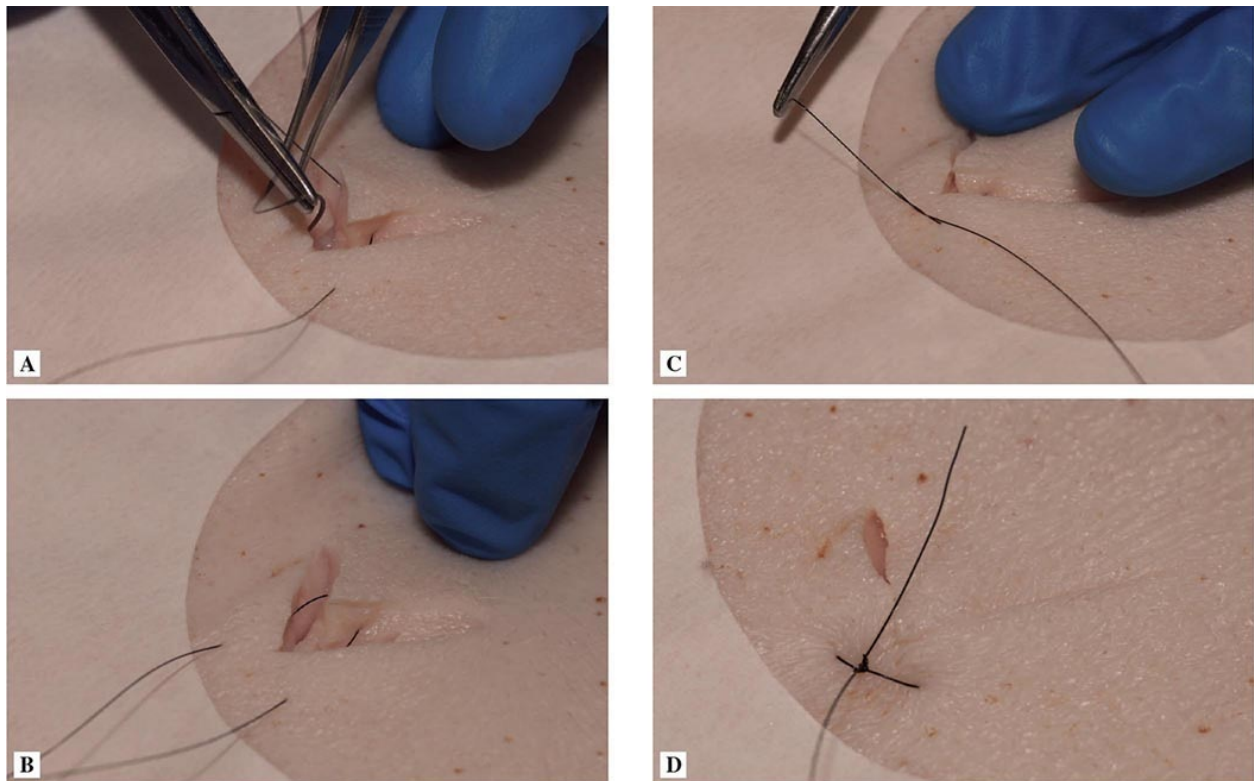


FIGURE 18.41 ■ Corner Stitch. Flaps generated by partial avulsion injuries must be repaired with care to avoid compromising the tenuous blood supply of the flap. The corner stitch is performed using a half-buried horizontal mattress suture. The suture begins percutaneously away from the wound corner. The suture needle is then brought horizontally through the corner at the level of the dermis and back out through the epidermis at the opposite noncorner portion of the wound (**A**). This technique avoids placing suture material near the apex of the flap (**B**). Tying (**C**) results in approximation of the corner (**D**). (Photo contributor: Michael L. Juliano, MD.)

WOUND CARE COMPLICATIONS

Clinical Summary

Infection is suggested by pain, warmth, erythema, edema, and purulent drainage.

While dehiscence can occur at any time, 7 to 10 days after repair, a wound is at its weakest (this also closely coincides with suture removal). Impaired wound healing, primarily from infection, medications (especially corticosteroids), foreign bodies, advanced age, poor nutritional status, diabetes mellitus, and peripheral vascular disease, contributes to dehiscence. Wound closure strips can be applied after suture removal and may reduce dehiscence. Some degree of scarring is inevitable, but not considered a complication of wound repair. This should be discussed with the patient or caregiver. Wound myiasis is infestation by fly larvae (see also [Chapter 21](#)) that invade necrotic tissue.

Management and Disposition

Infections are treated with suture removal, thorough irrigation, and low threshold to explore for missed foreign bodies. A 7-day course of a first-generation cephalosporin or antistaphylococcal penicillin is appropriate; however, if methicillin-resistant *Staphylococcus aureus* (MRSA) is suspected, the antibiotic choice should be adjusted (eg, trimethoprim-sulfamethoxazole or other current recommendations for your area). For animal bites, other antibiotics (eg, amoxicillin-clavulanate) may be more appropriate. Sepsis, advanced infections, or infections in persons with chronic medical problems (eg, diabetes, immunocompromised) should be managed with parenteral antibiotics and possible inpatient admission.



FIGURE 18.42 ■ Wound Infection Versus Inflammation. Intense erythema that extends from the wound margins peripherally (**A**) and puru-lent drainage (**B**) in postoperative wound infections. Compare this erythema to the pink inflammatory changes from the staples (**C**). This mild erythema extends from staple entrance into the skin; wound margins appear normal and are healing well. (Photo contributors: Lawrence B. Stack, MD [A and C]; Matthew D. Sztajnkrycer, MD, PhD [B].)



FIGURE 18.43 ■ Wound Dehiscence. After suture removal, the patient returned with dehiscence. The wound had a clean base of granulation tissue and was allowed to close by secondary intention. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 18.44 ■ Keloid. The degree of excessive scar bulk extending beyond the original wound margins may be dramatic and cosmetically significant. (Photo contributor: Thea James, MD.)



FIGURE 18.45 ■ Wound Myiasis. Necrotic tissue can be invaded by fly larvae (maggots). (Photo contributor: Lawrence B. Stack, MD.)

Wound dehiscence is managed conservatively by treating the underlying causes and allowing healing via secondary intention. Dehiscence of wounds in cosmetically sensitive areas is best managed in conjunction with a consultant. Myiasis is treated with wound cleaning and irrigation.

Pearls

1. All accidental wounds are considered contaminated and treated as such. Thorough irrigation and cleansing are of paramount importance in preventing wound infection.
2. Expedient ED wound care is important since bacterial contamination increases over time.

BURNS

Clinical Summary

Burns can be caused by heat, electricity, chemicals, friction, or radiation. Skin barrier damage can lead to infection, fluid loss, and electrolyte abnormalities. Long-term consequences include permanent scarring, loss of sensation, and in severe cases loss of extremities due to inadequate circulation. Burns are assessed by determining the percentage of body surface area (BSA) involved, the depth, and the area of the body involved. A common system used to estimate BSA is following the “rule of nines.” This system breaks up the body into zones that each equate to 9% of BSA (see figure). Some clinicians use the palm of the patient’s hand as an approximate equivalent to 1% BSA.

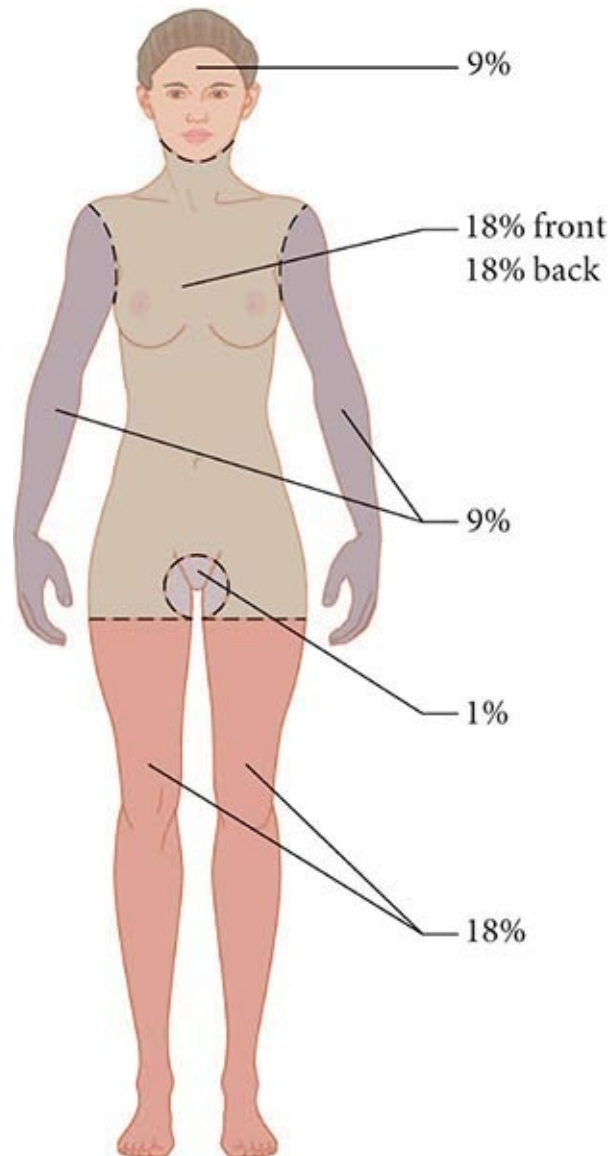


FIGURE 18.46 ■ Rule of Nines. Rule of nines is used to estimate the percentage of burn. (Reproduced with permission from Tintinalli JE, Stapczynski JS, Ma OJ, Cline DM, Cydulka RK, Meckler GD. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. 7th ed. McGraw Hill; 2011: Fig. 210-2, p. 1375.)

First-degree burns only involve the epidermal layer. They are red, painful, and heal in approximately 1 week. **Second-degree** burns are subdivided into superficial or deep partial thickness. Superficial second-degree burns extend from the epidermis to the superficial dermis. Pain, skin blistering, and intact capillary refill are characteristic. Deep partial-thickness burns extend into the deep (reticular) layer of the dermis and damage hair follicles, sweat glands, and sebaceous glands. Blisters may occur, the exposed dermis is pale white to yellow in color, and capillary refill is absent. The entire thickness of the skin is compromised in **third-degree** burns. They appear pale (waxy), feel leathery, and

are painless. **Fourth-degree** burns extend through the layers of the skin and involve muscle or bone.



FIGURE 18.47 ■ Second-Degree Burn. This patient has sustained a second-degree burn to his hand from hot oil. (Photo contributor: Suzanne Dooley-Hash, MD.)

Management and Disposition

After resuscitation and stabilization, refer to a burn unit for partial-thickness burns that involve greater than 10% BSA, third-degree burns, or involvement of the hands, feet, face, or perineum. Electrical burns, chemical burns, inhalation injuries, and patients with significant comorbidities should also be considered for a burn unit. Clinicians should cover the burned areas with a clean, dry sheet, administer aggressive pain control, and address fluid resuscitation. The Parkland formula is commonly used to estimate fluid requirements. The patient's weight in kilograms is multiplied by the percent BSA involved; this number is multiplied by 4 mL of lactated Ringer solution. Half of this amount is given during the first 8 hours from time of initial injury and the remaining amount is given over the next 16 hours. It is recommended to keep urine output approximately 0.5 to 1.0 mL/kg/h. To monitor for effects of cell breakdown, urinalysis, creatine kinase, and an electrocardiogram (ECG) should be obtained. Circumferential burns of the extremities may compromise circulation. If compartment syndrome is suspected, an escharotomy should be considered.

For minor burns, provide pain control, cleansing of the area, and application of topical antimicrobials (eg, commonly bacitracin, or triple-antibiotic ointment). Dressing changes should occur daily, and patients must be instructed to watch for signs of infection. Follow-up with a burn care expert needs to be arranged within a few days of discharge for deep partial-thickness or third-degree burns.

Pearls

1. Minor burns can be managed in the ED. Pain control, irrigation, antimicrobial ointments, and dressing changes are the mainstays of therapy.
2. The extent of the burn is often overestimated and the depth underestimated.



FIGURE 18.48 ■ Facial Burn, Second Degree. Ensuring that the patient has a protected airway in this type

of injury is crucial. (Photo contributor: David Effron, MD.)



FIGURE 18.49 ■ Extensive Burn Injury. Burns can be of varying depths; this patient has both second- and third-degree burns. Distal pulses should be closely monitored to guarantee that there is adequate circulation. (Photo contributor: David Effron, MD.)



FIGURE 18.50 ■ Abdominal Burn. Mostly second-degree abdominal burn. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 18.51 ■ Third-degree Burn. Note pale, waxy appearance after a dry cleaner press burn. (Photo contributor: R. Jason Thurman, MD.)

ELECTRICAL INJURY

Clinical Summary

Electricity generates heat through tissue resistance or directly by the current on cells. Many factors affect injury severity: type of current (DC or AC), intensity, duration, tissue resistance, and pathway through the body. When electricity traverses the tissues, it may cause contact burns, thermal injury, muscular tetany, or severe contraction. Sudden death (asystole, respiratory arrest, ventricular fibrillation), myocardial damage, myoglobinuria, compartment syndrome, and various metabolic disorders have been described.



FIGURE 18.52 ■ Electrical Burn. This patient grabbed a high-voltage power line with his hand. Exit wounds occurred where the patient was grounded, through his feet. This patient with transthoracic injury should have cardiac monitoring. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 18.53 ■ Electrical Flashover Burn. A patient struck by flashover electricity while repairing urban outdoor electric lines. (Photo contributor: Selim Suner, MD, MS.)

High-voltage DC or AC current typically causes a single violent muscular contraction and throws the victim from the source; blunt trauma and blast injuries may occur. Low-voltage AC currents (as from a household outlet) may cause muscular tetany, forcing the victim to continue contact with the source.

Management and Disposition

After initial stabilization, consider cervical spine immobilization, oxygen administration, cardiac monitoring, and IV crystalloid infusion. A Foley catheter will help monitor urine output and is especially important if rhabdomyolysis is suspected. Diagnostic testing to consider includes ECG, complete blood count, urinalysis, creatine phosphokinase (CPK), CPKMB, electrolytes, blood urea nitrogen, creatinine, and coagulation profile.

Severe or high-risk injuries should be admitted to a burn unit or a trauma center with burn consultation. Patients with minor, brief, low-intensity exposures; a normal ECG; normal urinalysis; and no significant burns or trauma may be considered for discharge after a period of observation.



FIGURE 18.54 ■ Electrical Burn. Extensive electrical hand burn from a high-voltage power line. (Photo contributor: David Effron, MD.)

Pearls

1. Toddlers are at increased risk of labial injury from chewing on electrical cords.
2. High-risk features include high-voltage exposure (>600 V), deep burns, neurologic injury, dysrhythmias, abnormal ECG, evidence of rhabdomyolysis, suicidal intent, or significant associated trauma.



FIGURE 18.55 ■ DC Thermal Ring Injury. This middle-aged man sustained a circumferential thermal finger burn. His ring was heated when a wrench he was holding simultaneously contacted both terminals on a car battery. (Photo contributor: Aubrey Mowery, MSN, MPH, CPNP.)

DECUBITUS ULCERS

Clinical Summary

Decubitus ulcers develop when soft tissue is compressed between a bony prominence and a hard external surface. Tissue compression results in decreased blood flow, tissue ischemia, and cell death. Pressure, shearing forces, friction, and excessive moisture contribute to formation. Areas commonly affected include the sacrum and heels. Immunocompromised, nursing home, neurologically impaired, and immobilized trauma patients are at high risk.

Stage I ulcers are characterized by an area of nonblanchable erythema over intact skin. *Stage II* appears as a shallow, open sore with a pink wound base. When the wound is full thickness with no muscle, tendon, or bone exposed, it is defined as *stage III*. If muscle, tendon, or bone is exposed, it is *stage IV*. Some wounds may have an area of black eschar over them; these wounds cannot be categorized since injury depth cannot be determined.

Management and Disposition

Prevention is key. All patients who have decreased mobility, such as trauma and nursing home patients, should have their entire skin surface regularly checked for skin breakdown and should be repositioned frequently. Backboards, c-collars, and other immobilizing devices should be removed as soon as possible.

Treatment includes pain relief, keeping the affected area clean, and keeping pressure off the area to prevent further tissue destruction. There are multiple commercial products available (hydrocolloid dressings) to use for the treatment of pressure ulcers. Frequent monitoring is required, and consultation with wound care specialists or a surgeon may be necessary for advanced ulcers. The main complications of decubitus ulcer formation include infection and skin dehiscence.



FIGURE 18.56 ■ Stage II Decubitus Ulcer. Partial thickness loss of dermis presenting as a shallow open ulcer with a red-pink wound bed. (Photo contributor: Suzanne Dooley-Hash, MD.)

Pearls

1. Decubitus ulcers are divided into four stages. If an eschar is present, the ulcer cannot be categorized.
2. Remove hard external surfaces and immobilizing devices as soon as possible.



FIGURE 18.57 ■ Stage III Decubitus Ulcer. Stage three decubitus ulcers extend into the subcutaneous tissue. (Photo contributor: David Effron, MD.)



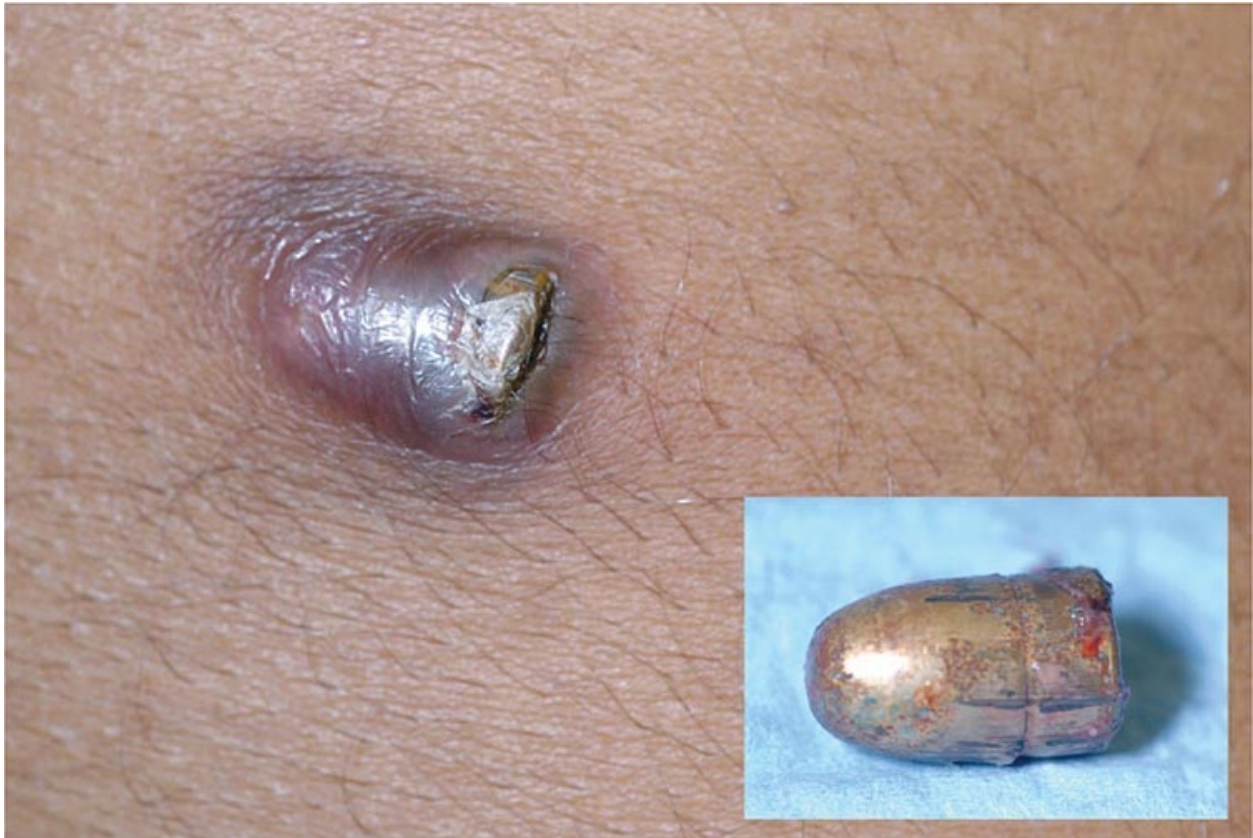
FIGURE 18.58 ■ Stage IV Decubitus Ulcer. Full-thickness tissue loss with exposed muscle. (Photo contributor: Lawrence B. Stack, MD.)

The authors acknowledge Matthew D. Sztajnkrzyer and Alexander T. Trott for portions of this chapter written for the second edition.

Chapter 19

CLINICAL FORENSIC MEDICINE

William S. Smock
Lawrence B. Stack



Retained Projectile. A bullet from a remote gunshot wound to the right chest migrated to the skin surface over time and was removed at the patient's request. The bullet (insert) was sent to pathology. (Photo contributor: Lawrence B. Stack, MD.)

GUNSHOT WOUNDS

Clinical Summary

Gunshot injuries can be accurately identified and classified as entrance, atypical entrance, exit, or atypical (grazing) wounds based on their physical characteristics. **Wounds are not classified based on their size.** Physical findings in and around these wounds may offer evidence as to the actual mechanism of injury, supporting or refuting the initial history given to the provider. As most physical findings are transient in nature (cleaned, debrided, or eventually healed), the emergency physician must be diligent in recognizing and documenting them at the time of presentation. The physician's failure to accurately document the physical characteristics of the wounds or correctly interpret the physical findings can compromise the legal process and obstruct justice.

Entrance Wounds

Gunshot wounds of entrance are divided into four categories based on their range of fire: *distant*, *intermediate*, *close*, and *contact*. Range-of-fire is the distance from the gun's muzzle to the victim's skin or clothing.

The size of the entrance wound bears no relation to the caliber of the inflicting bullet. Entrance wounds over elastic tissue will contract around the tissue defect and have a diameter much less than the caliber of the bullet.

Distant Wounds: The distant wound is inflicted from a range sufficiently distant that the bullet is the only component expelled from the muzzle that reaches the skin. There is no visible tattooing or soot deposition associated with a distant entrance wound. As the bullet penetrates the skin, friction between it and the epithelium results in the creation of an "abrasion collar" (Fig. 19.1). The width of the abrasion collar will vary with the angle of impact. Elongated abrasion collars from projectiles that enter on an angle may produce a collar with a "comet tail" (Fig. 19.2). Most entrance wounds will have an abrasion collar; however, gunshot wounds to the palms and soles are exceptions—their entrance wounds appear slit-like. Bullets that pass through an intermediate object, a door or windshield for example, will become deformed or misshapen (Fig. 19.3). A misshapen bullet creates an irregular abrasion collar (Fig. 19.4) as compared to the smooth abrasion collar created by a nondeformed bullet (Fig. 19.1).

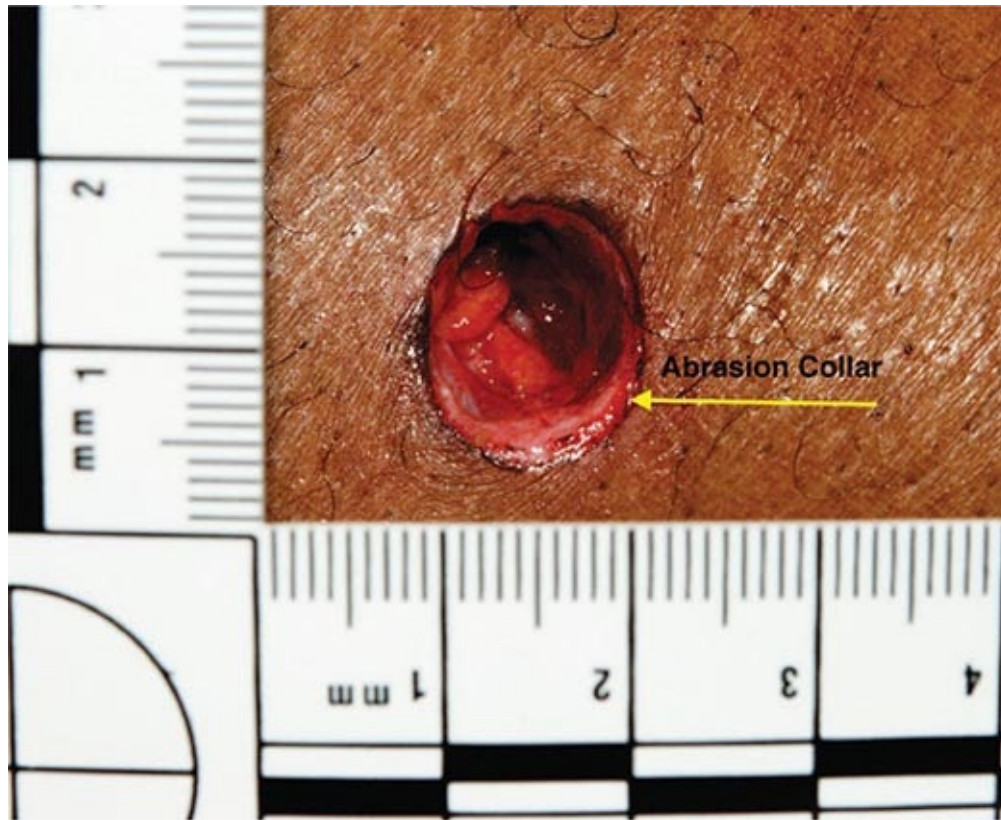


FIGURE 19.1 ■ Distant Gunshot Wound. An abrasion collar surrounds the wound defect and is created from the friction of a bullet passing through the skin. All entrance wounds will have an abrasion collar with the exception of entrance wounds to the palms and soles. The lack of soot, seared skin, or gunpowder tattooing confirms this is a “distant” range of fire. (Photo contributor: William S. Smock, MD.)

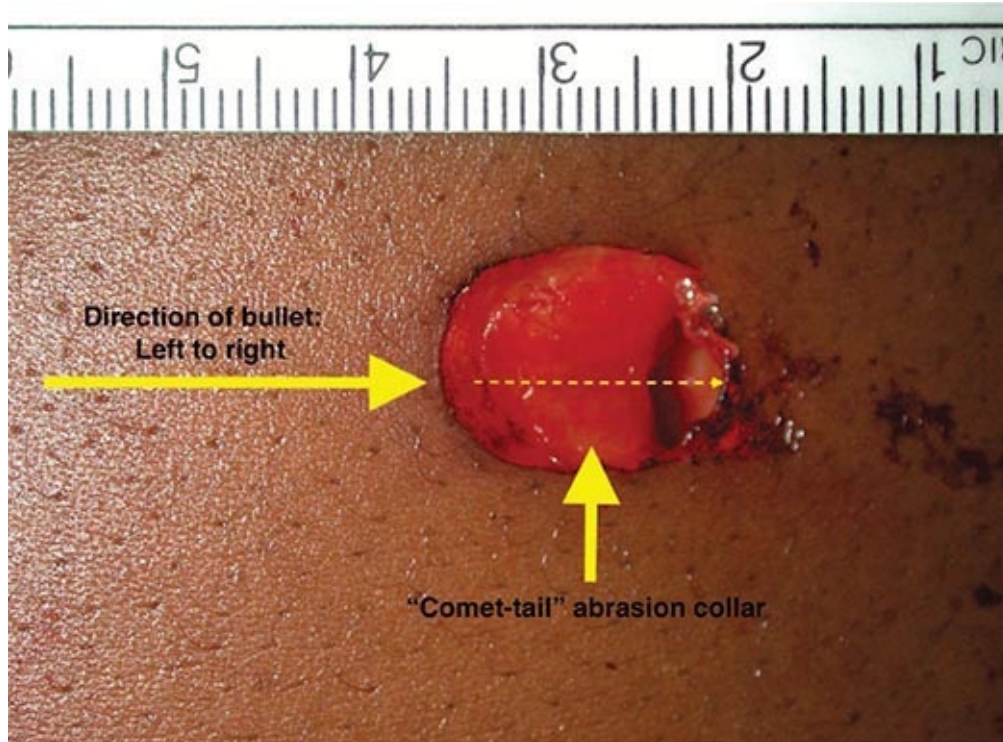


FIGURE 19.2 ■ “Comet-Tailed” Abrasion Collar. The “comet tail” abrasion collar located on the lateral aspect of the wound indicates that the bullet entered the wound at an angle. The “comet tail” also indicates the bullet’s direction of travel: from left to right. (Photo contributor: William S. Smock, MD.)



FIGURE 19.3 ■ Deformed Bullet. This bullet passed through a car windshield and deformed prior to striking the suspect in the arm. The bullet's irregular shape created the atypical abrasion collar associated with the entrance wound seen in [Fig. 19.4](#). (Photo contributor: William S. Smock, MD.)

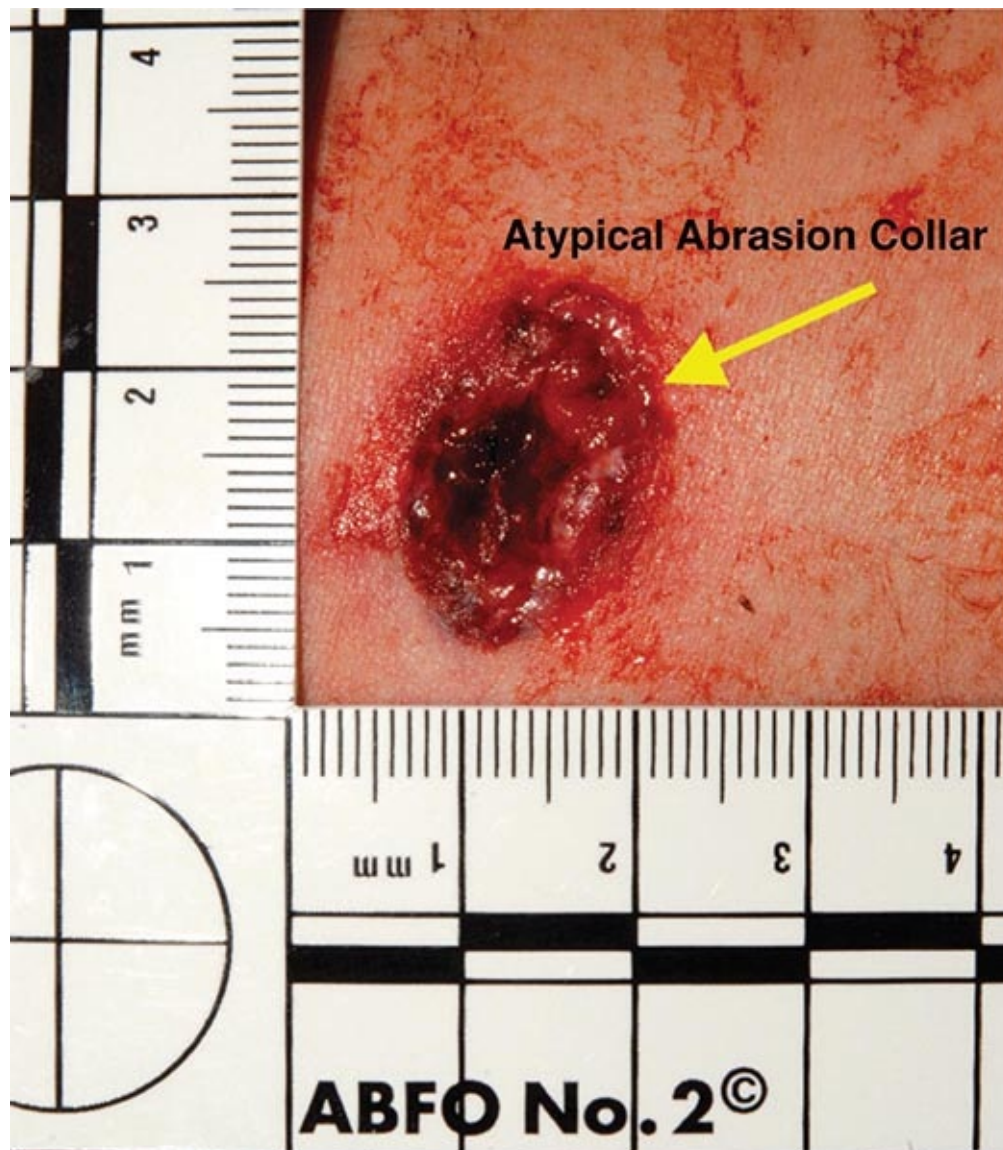


FIGURE 19.4 ■ Atypical Abrasion Collar. When a bullet passes through an intermediate object (eg, a door or windshield), it can become deformed or misshapen with an irregular surface. When a misshapen bullet ([Fig. 19.3](#)) passes through skin, the abrasion collar it generates appears irregular and jagged when compared to the smooth one caused by a nondeformed bullet ([Figs. 19.1](#) and [19.2](#)). (Photo contributor: William S. Smock, MD.)

Intermediate-Range Wounds: Tattooing is pathognomonic for an intermediate-range (less than 48 inches) gunshot wound and presents as punctate abrasions from contact with partially burned or unburned grains of gunpowder ([Fig. 19.5](#)). This tattooing cannot be wiped away. Clothing and hair, as intermediate objects, may prevent the gunpowder grains from making contact with the skin. Tattooing

can, but rarely does, occur on the palms and soles owing to the thickness of their epithelium.

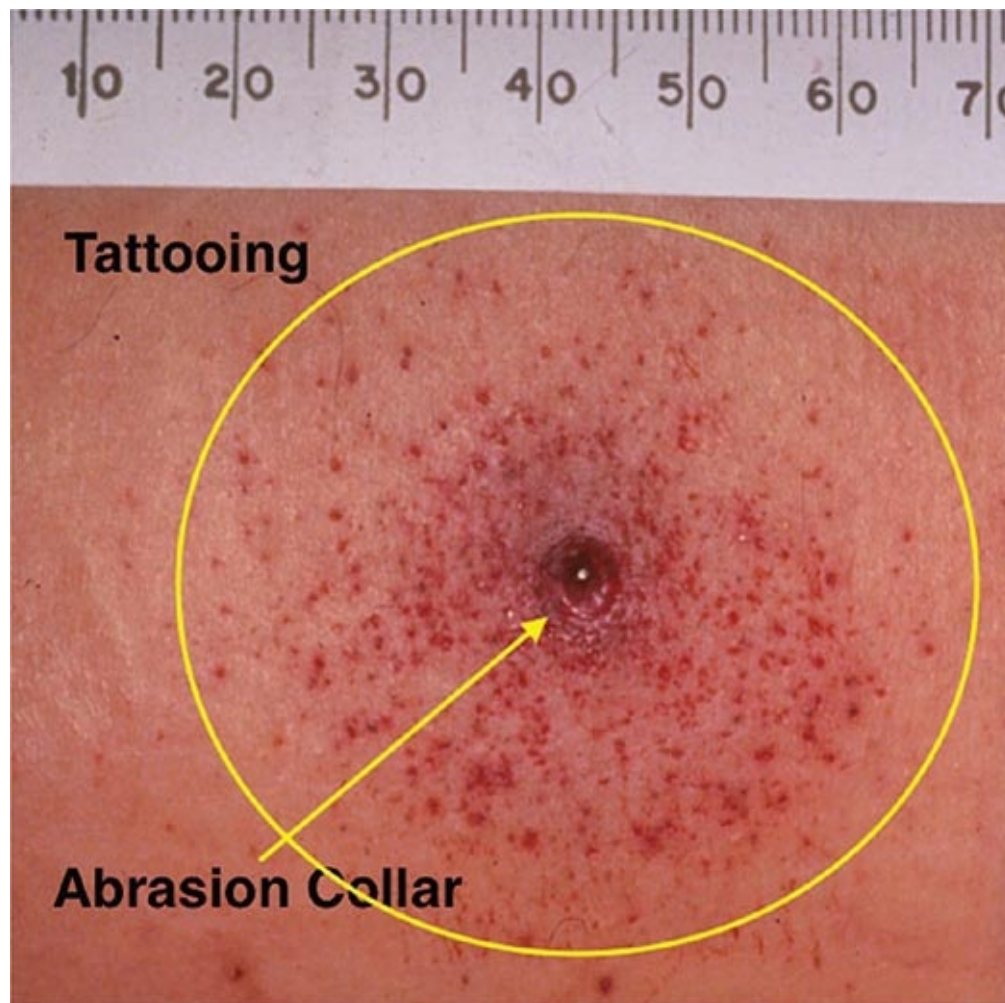


FIGURE 19.5 ■ Intermediate-Range Gunshot Wound. Punctate abrasions present on the skin are the result of impact from gunpowder that is either wholly or partially unburned. This phenomenon is termed *tattooing or stippling*. Tattooing is pathognomonic for intermediate-range (< 48 inches) gunshot wounds. (Photo contributor: William S. Smock, MD.)

Tattooing has been reported with a range of fire as close as 1 cm and as far away as 4 feet. The density of the abrasions and the associated pattern will depend on the barrel length, muzzle-to-skin distance, type of gunpowder (ball, flattened ball, or flake), presence of intermediate objects, and caliber of the weapon. Spherical powder travels farther and has greater penetration than flattened ball or flake powder. “Pseudo-tattooing” (Fig. 19.6) is punctate abrasions from fragments created when bullets pass through intermediate objects such as wood or glass.



FIGURE 19.6 ■ Pseudo-tattooing. Punctate abrasions present on the forearm are the result of impacts with glass fragments. The fragments were produced from bullets penetrating the windshield. (Photo contributor: William S. Smock, MD.)

Close-Range (Near-Contact) Wounds: “Close range” is defined as the maximum range at which soot is deposited on the clothing ([Fig. 19.7](#)) or wound ([Fig. 19.8](#)) and typically is a muzzle-to-victim distance of 6 inches or less. On rare occasions, however, soot has been found on victims as far as 12 inches from the offending weapon. The concentration of soot will vary inversely with the muzzle-to-victim distance, and its appearance will be affected by the type of gunpowder and ammunition used, the barrel length, the caliber, and the type of weapon.

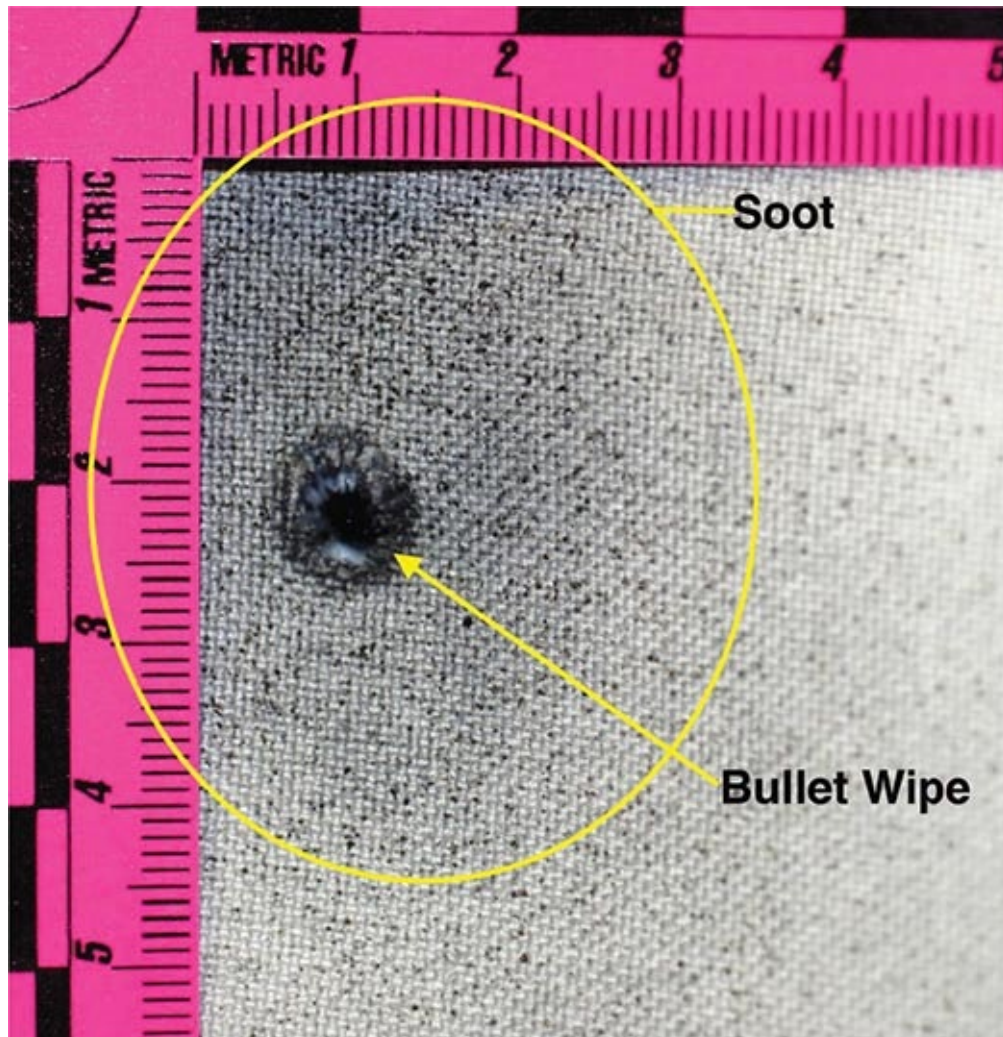


FIGURE 19.7 ■ Soot and Bullet Wipe. Soot is the carbonaceous residue from the burning of gunpowder. Soot is associated with close-range wounds (6 inches or less). Bullet wipe is residue and/or lead from the surface of the bullet that is transferred to clothing or skin. Bullet wipe can be seen at any range of fire. Clothing should be collected and packaged in separate paper bags for submission to the crime laboratory. (Photo contributor: William S. Smock, MD.)

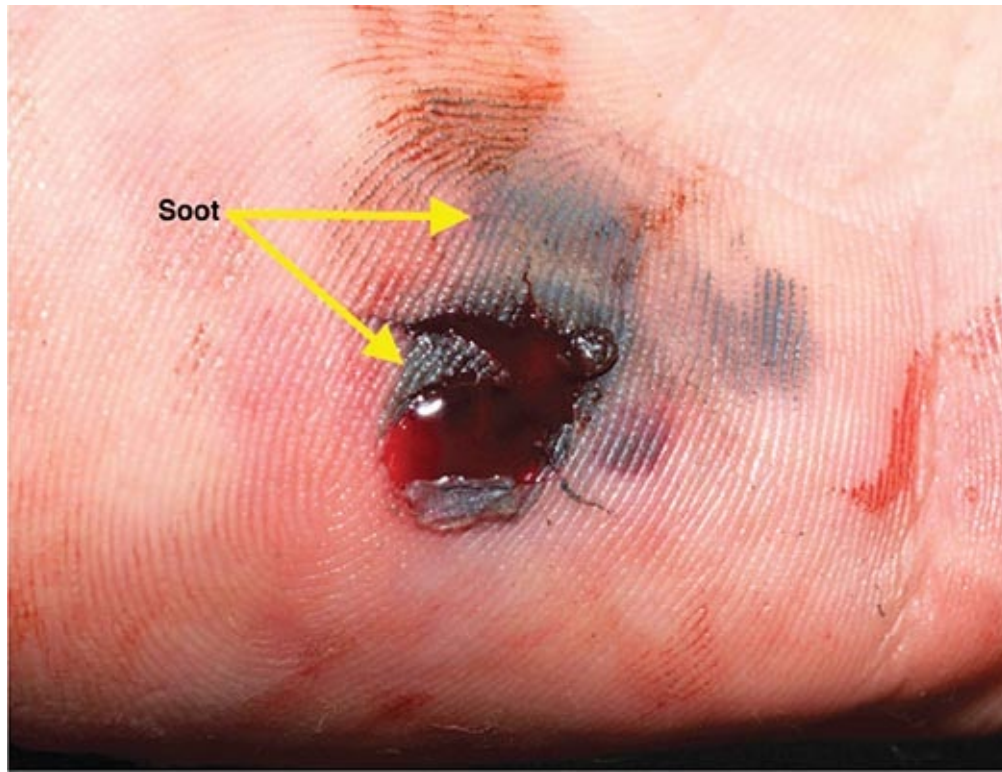


FIGURE 19.8 ■ Close-Range Gunshot Wound. The deposition of carbonaceous material or soot is seen on the palm from a close-range or near-contact gunshot wound. Soot is short-lived evidence, and its presence surrounding a wound should always be documented. (Photo contributor: William S. Smock, MD.)

Contact Wounds: A contact wound occurs when the barrel or muzzle is in contact with the skin or clothing as the weapon is discharged. Contact wounds can be described as tight, where the muzzle is pushed hard against the skin, or loose, where the muzzle is incompletely or loosely in contact with the skin or clothing. Wounds sustained from tight contact with the barrel can vary in appearance from a small hole with seared, blackened edges (from the discharge of hot gases and an actual flame), to a gaping, stellate wound (from the expansion of the skin from gases) (Figs. 19.9 and 19.10). Large stellate wounds are often misinterpreted as exit wounds based solely on their size and without adequate examination of the wound characteristics.

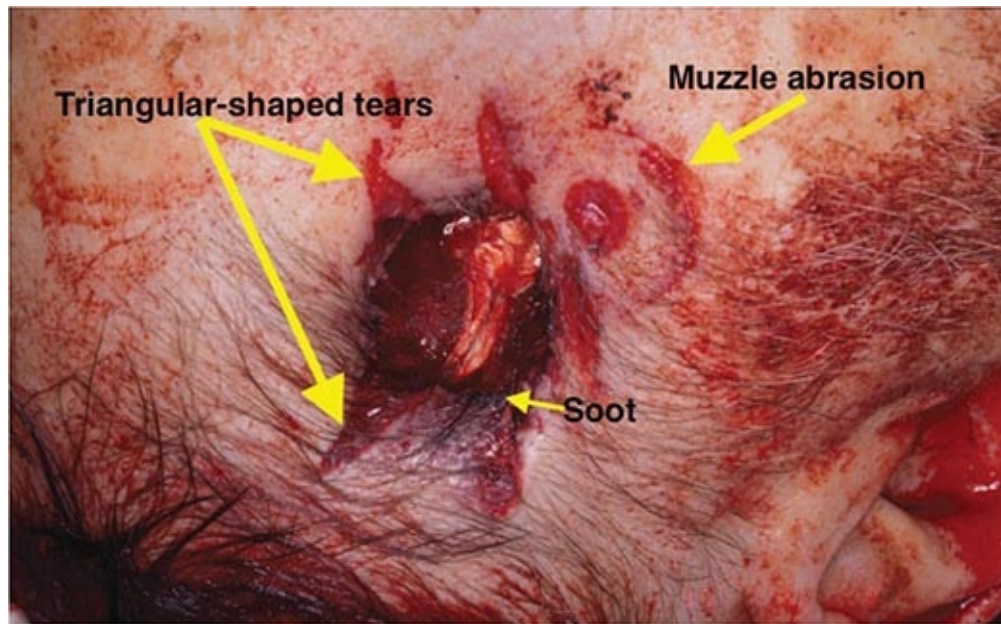


FIGURE 19.9 ■ Contact Gunshot Wound with Muzzle Abrasion. A contact gunshot wound to the right temple from a 9-mm semiautomatic handgun. Note the triangle-shaped tears, soot, seared wound margins, and a muzzle abrasion at the 2-o'clock position. The muzzle abrasion or muzzle imprint was the result of the injection of gases into the skin, causing a rapid and forceful expansion of the skin against the barrel. (Photo contributor: William S. Smock, MD.)



FIGURE 19.10 ■ Contact Gunshot Wound. A contact wound to the forehead from a .38 caliber handgun. The wound margins display triangle-shaped tears, searing, and soot deposition. (Photo contributor: William S. Smock, MD.)

In a tight-contact wound, all materials—the bullet, gases, soot, incompletely combusted gunpowder, and metal fragments—are driven into the wound. If the wound overlies thin or bony tissue, the hot gases will cause the skin to expand to such an extent that it stretches and tears. These tears will have a triangular shape, with the base of the tear overlying the entrance wound. Larger tears are associated with ammunition of .32 caliber or greater, or magnum loads.

Stellate tears are not pathognomonic for contact wounds. Tangential wounds, ricochet or tumbling bullets, and some exit wounds may also be stellate in appearance. These wounds are distinguished from tight-contact wounds by the absence of soot and powder within the wound. In some tight-contact wounds, expanding skin is forced back against the muzzle of the gun, causing a characteristic pattern contusion called a *muzzle contusion* (Fig. 19.9). An outline of the barrel can also be imprinted on the overlying clothing and is associated with contact wounds through clothing (Fig. 19.11). These patterns are helpful in determining the type of weapon (revolver or semiautomatic) used to inflict the injury and should be documented prior to wound debridement or surgery.

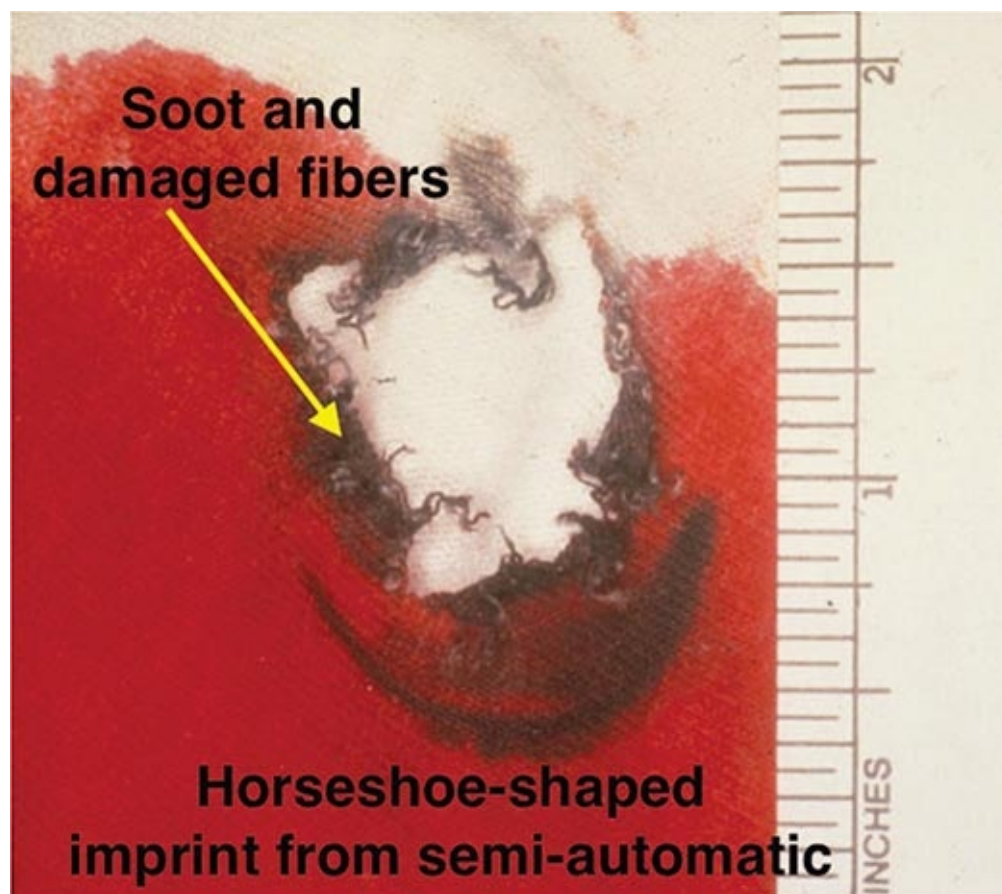


FIGURE 19.11 ■ Muzzle Imprint, Soot, and Seared Fibers. The clothing exhibits a “horseshoe-shaped” soot mark reflecting the outline of the frame of a semiautomatic handgun. Seared fibers are the result of

flame and hot gases expelled from the barrel when it is in contact with clothing. (Photo contributor: William S. Smock, MD.)

With a loose-contact wound, where the muzzle is angled or held loosely against the skin, soot and gunpowder residue will be present in and around the wound. The angle between the muzzle and skin will determine the soot pattern. A perpendicular, loose-contact or near-contact injury results in searing of the skin and deposition of the soot evenly around the wound. A tangential loose or near-contact injury produces an elongated searing pattern and deposit of soot around the wound.

“Bullet wipe” is a residue from soot, soft lead, or lubricant, which may leave a gray or black rim or streak on the skin or clothing overlying an entrance wound (Fig. 19.7). This discoloration may also be found around the abrasion collar but is usually more prominent on clothing.

Exit Wounds

Determining whether a wound is an entrance or an exit wound should be based on the physical characteristics and physical evidence associated with the wound and *never on the size* of the wound. The size of the exit wound is the result of a bullet pushing and stretching the skin from inside outward. The skin edges are generally everted, with sharp but irregular margins (Figs. 19.12-19.14). Abrasion collars, soot, searing, and tattooing are not associated with exit wounds. Soot can be seen at an atypical exit wound site if the entrance wound is close to the associated exit wound. Soot can be propelled through the wound from entrance to exit when the wound track is extremely short. If this is noted, the soot deposition will be more pronounced at the entrance and only faintly observed within the exit wound.



FIGURE 19.12 ■ Exit Gunshot Wound. A slit-like exit wound on the scalp from an intraoral gunshot. Exit wounds may take on a variety of appearances. (Photo contributor: William S. Smock, MD.)



FIGURE 19.13 ■ Exit Gunshot Wound. Stellate tears in an exit wound from a .22 caliber long rifle bullet that impacted the radius and ulna. The stellate configuration of an exit wound should not be confused with that of a contact wound. Exit wounds lack soot and seared skin. (Photo contributor: William S. Smock, MD.)



FIGURE 19.14 ■ High-Velocity Gunshot Wound. A perforating high-velocity gunshot wound to a lower extremity. The gaping exit wound resulted from the transfer of energy from the projectile to the tibia. The impact propelled multiple bony fragments through the skin. (Photo contributor: William S. Smock, MD.)

Exit wounds assume a variety of shapes and appearances and are *not* consistently larger than their corresponding entrance wounds. The size of an exit wound is determined primarily by the amount of energy transmitted to underlying tissue, bone for example, which is extruded from the wound. A bullet's size, shape, and attitude as it exits, as well as postin-jury tissue swelling, will affect the wound size. A bullet's usual nose-first attitude can change upon entering the skin to a tumbling and yawing one. A bullet with sufficient energy to exit the skin in a sideways attitude or one that has increased its surface area by mushrooming may produce an exit wound larger than its entrance wound. Energy transferred to bone, with resultant ballistic fracture, may also result in an exit wound larger than the entrance wound (Fig. 19.14). A "false abrasion collar" or "shored exit" wound may mimic an entrance wound. This occurs when the epithelium is pressed against a supporting surface such as a floor, wall, chair, firm mattress, or wallet (Figs. 19.15 and 19.16).



FIGURE 19.15 ■ Shored Gunshot Exit Wound. A "shored exit" or "false abrasion collar" associated with a gunshot wound of exit. The false abrasion collar results when the skin is supported by a firm surface as the bullet exits. Shored exits occur when epithelium is pressed against a supporting surface (ie, floor, wall, chair, firm mattress, or, as in this case, wallet). (Photo contributor: William S. Smock, MD.)



FIGURE 19.16 ■ Wallet Causing Shored Gunshot Exit Wound. The wound seen in [Fig. 19.15](#) occurred as a result of this wallet resisting the bullet's exit. (Photo contributor: William S. Smock, MD.)

Graze Wounds

Graze wounds are considered atypical and result from tangential contact with a passing bullet. The direction of the bullet's path may be determined by careful wound examination. The bullet produces a trough and may cause the formation of skin tags on the lateral wound margins ([Fig. 19.17](#)). The base of these tags point toward the weapon and away from the direction of bullet travel.



FIGURE 19.17 ■ Graze Gunshot Wound. A deep graze wound from a handgun is seen. The dark wound margins are the result of drying artifact and should not be confused with the deposition of soot. (Photo contributor: Lawrence B. Stack, MD.)

Evidence Collection

Preservation of clothing, bullets, and cartridge cases is critical to the investigation of gun violence. Clothing should be placed in separate paper bags and bullets in breathable containers. The microscopic marks on the exterior surface of a bullet can be used to identify the offending weapon. The use of booties on the ends of hemostats can prevent the loss of essential evidence and preserve these unique identifying microscopic marks from being altered or permanently destroyed ([Fig. 19.18](#)).



FIGURE 19.18 ■ Suture Booties. The use of booties on hemostats prevents the identifying microscopic marks on the bullet's surface from being damaged by handling. (Photo contributor: William S. Smock, MD.)

Pearls

1. Distant-range gunshot wounds are inflicted from a distance of greater than 4

- feet, and there is no tattooing, soot, or searing associated with the wound.
2. Intermediate-range gunshot wounds are inflicted at distances up to 4 feet and characteristically are associated with tattooing from partially burned and unburned gunpowder impacting the skin.
 3. Close-contact gunshot wounds are defined as the maximum range at which soot is deposited on the wound or clothing and typically occur at a distance of 6 inches or less.
 4. Contact gunshot wounds (barrel is in contact with the skin or clothing at time of discharge) vary in size but will include triangular tears, searing of the skin, and soot within or around the wound.
 5. Abrasion collars, soot, searing, and tattooing are associated with entrance wounds.
 6. Determination of whether a wound is an entrance or exit wound should **only** be based on the physical characteristics of the wound and clothing and **never** on the size of the wound.
 7. The size of an exit wound is determined by: (1) energy transferred from the bullet to underlying tissue (bone pushed out), (2) bullet shape (mushroomed) and configuration, and (3) swelling of underlying tissue.
 8. Emergency physicians should attempt to recognize, document, preserve, and collect short-lived evidence whenever the clinical situation allows.

Pattern Injuries of Domestic Violence, Assault, and Abuse

Every “weapon” (hand, belt, hot iron, knife, electrical cord, baseball bat, tire iron) can leave a mark, design, or pattern stamped or imprinted upon or just below the level of the epithelium. The imprints of these weapons are called *pattern injuries*, which are considerably reproducible. These injuries can be categorized into three major classifications according to their source: sharp-force, blunt-force, and thermal-pattern injuries.

SHARP-FORCE-PATTERN INJURIES

Clinical Summary

There are two types of sharp-force injuries: incised and stabbed. The incised wound is longer than it is deep. The stab wound is defined as a puncture wound that is deeper than it is wide (Figs. 19.19-19.21). The wound margins of sharp-force injuries are clean and lack the abraded edges of lacerations from blunt forces. Forensic information can be gathered during the examination of a stab wound. Some characteristics of a knife blade, single- or double-edged, can be determined by visual inspection. Characteristics such as serrated versus sharp can be determined if the blade was drawn across the skin during insertion or withdraw from the victim. Serrated blades do not always leave these characteristic marks.

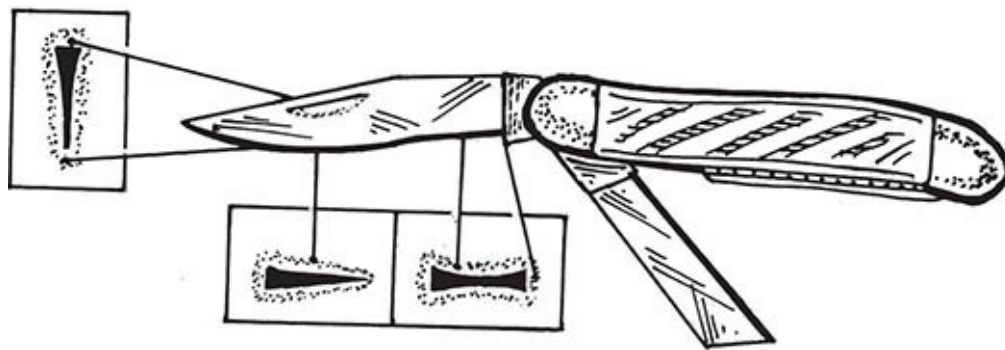


FIGURE 19.19 ■ Stab Wound. A stab wound from a single-edged knife blade will impart a sharp edge and a dull edge to the wound. If the blade penetrates to the proximal portion of the blade, a contusion may result from contact with the hilt of the knife.



FIGURE 19.20 ■ Single-Edge Stab Wound with Hilt Mark. A single-edged stab wound with a small hilt mark associated with the dull edge of the blade. (Photo contributor: William S. Smock, MD.)



FIGURE 19.21 ■ Single-Edge Stab Wound. A stab wound from a single-edged knife blade. The superior portion of the wound corresponds with the dull edge of the blade and the inferior with the sharp edge of the blade. (Photo contributor: William S. Smock, MD.)

When a patient presents with superficial and/or parallel incisions, the wounds

must be considered intentionally self-inflicted until proven otherwise (Figs. 19.22 and 19.23). The emergency physician should obtain a more detailed history, including the number of “swipes” the alleged assailant made with the knife. Accidental, self-inflicted incised wounds on the palm or palmar aspect of the fingers are common on the hands of suspects in violent stabbings (Fig. 19.24). The suspect’s hand slips down the handle and makes contact with the knife blade when the blade impacts a firm object, like bone (Fig. 19.25).



FIGURE 19.22 ■ Self-Inflicted Incised Wounds. Superficial and/or parallel incisions are pathognomonic for intentional self-inflicted wounds. This patient has acute and healed self-inflicted incisions in the same location on the anterior lateral aspect of her neck. (Photo contributor: William S. Smock, MD.)



FIGURE 19.23 ■ Self-Inflicted Incised Wounds. Examination of the “victim’s” chest reveals multiple superficial incisions. The patient initially claimed she was “assaulted” but later admitted that the wounds were self-inflicted. (Photo contributor: William S. Smock, MD.)



FIGURE 19.24 ■ Incised Wounds on the Palm. Suspects in stabbings sometimes have incised wounds on

their own palm or the palmar surfaces of their fingers. (Photo contributor: William S. Smock, MD.)



FIGURE 19.25 ■ Hand Slippage on Knife. When the knife blade suddenly stops (eg, if the blade hits bone), the hand can slip down the handle and make contact with the blade, incising wounds on the assailant's palm ([Fig. 19.24](#)). (Photo contributor: William S. Smock, MD.)

Pearls

1. Incised wounds have sharp wound margins and are longer than they are deep. Incisions result from contact with a sharp-edged implement: glass, knife blade, razor blade, or scalpel.
2. Stab wounds are puncture wounds that are deeper than they are long.
3. Knife-blade characteristics (single or dual edged, serrated or smooth) can frequently be determined by a visual inspection of the wound.
4. Superficial and/or parallel incisions should be assumed to be self-inflicted.

BLUNT-FORCE PATTERN INJURIES

Clinical Summary

The most common blunt force injury is the contusion ([Fig. 19.26](#)). The *pattern contusion* is one that helps identify the causative weapon. A blow from a linear object leaves a contusion that is characterized by a set of parallel lines separated by an area of central clearing ([Fig. 19.27](#)). The blood underlying the striking object is forcibly displaced to the sides, which accounts for the pattern's appearance. Pattern injuries that an emergency physician should recognize include those caused by the hand (slap marks [[Fig. 19.27](#)], fingertip contusions [[Fig. 19.28](#)], grab marks, ligature marks [[Fig. 19.30](#)], fingernail abrasions), those caused by solid objects (baseball bat, tire iron, 2 by 4, belt, shoe, comb), and bite marks.



FIGURE 19.26 ■ Periorbital Contusion. A contusion without a discernable pattern is the result of some type of blunt-force trauma. Victims of interpersonal violence frequently present with nonspecific, nonpattern contusions. A 24-hour-old cigarette burn is located on the victim's right upper lid. This victim was held against her will for 48 hours and repeatedly beaten about the face. (Photo contributor: William S. Smock, MD.)



FIGURE 19.27 ■ Slap-Mark Pattern Contusions. This victim of a “fall from the couch” has a pattern injury present on the right cheek. The pattern of central clearing surrounded by linear parallel contusions is pathognomonic for impact with a rounded linear object, in this case the extended fingers of a hand. (Photo contributor: William S. Smock, MD.)



FIGURE 19.28 ■ Fingertip Contusion Pattern. This patient exhibits fingertip contusions. The emergency physician should never attempt to date the age of a contusion based upon its color. (Photo contributor: William S. Smock, MD.)



FIGURE 19.29 ■ Tire Mark. This tire mark, a mirror image of the tire tread, was the only evidence that connected this pedestrian with the vehicle involved in the hit-and-run incident. (Photo contributor: William S. Smock, MD.)



FIGURE 19.30 ■ Strangulation Ligature Belt Marks. Ligature marks from a belt, with petechial hemorrhage above the marks, supported the mentally challenged patient's story of being "choked" by her caretaker. The majority of fatal strangulations do not exhibit external evidence of trauma. (Photo contributor: William S. Smock, MD.)

Other manifestations of blunt-force trauma to the skin are the abrasion and the laceration. A weapon with a unique shape or configuration may stamp a mirror image of itself on the skin (Fig. 19.29) and provide the emergency physician with additional information on the mechanism of the injury. The color of a contusion cannot be used to accurately date its age.

Pearls

1. A contusion is the most common blunt-force injury pattern in interpersonal violence.
 2. Blood underlying the force of the contusion is displaced to either side of the object, causing a *pattern contusion* in the shape of that object. This pattern is recognized by the central clearing surrounded by linear parallel contusions.
 3. Emergency physicians must be able to recognize the pattern injuries caused by the hand (Fig. 19.27), solid objects, and bites.
 4. Emergency physicians should not attempt to date the age of a contusion based on its color or stage of resolution.
-

STRANGULATION INJURIES

Clinical Summary

Manual strangulation is one of the most lethal forms of intimate partner violence and demonstrates the abuser's power and control over the victim. The brain will suffer an anoxic injury when the flow of oxygenated blood is stopped by occlusion of the carotid arteries. Strangulation victims report visual and auditory changes just prior to a loss of consciousness. The loss of bladder control indicates a deep anoxic insult to the brain of at least 15 seconds in duration. The loss of bowel control occurs with carotid artery occlusions of greater than 30 seconds. The jugular veins can be occluded with 4.4 psi, the carotids with 11 psi, and the trachea with 34 psi. Occlusion of venous return can cause capillary rupture from increased pressure and the development of petechial hemorrhage (Figs. 19.32-19.34). Petechial hemorrhages can develop in any vascularized tissue, including the brain. The most sensitive areas for petechial hemorrhage development include the conjunctiva, sclera, and intraoral mucosa. The use of an oral-pharyngeal scope (Fig. 19.35) often reveals vocal cord injury and the presence of petechial hemorrhage in the posterior pharynx. With simultaneous occlusion of both the carotid and vertebral arteries and venous systems, it is possible to induce fatal and nonfatal strangulation events without petechial hemorrhage. Victims can be fatally strangled without external evidence of trauma.



FIGURE 19.31 ■ Strangulation Ligature Mark. Ligature marks from the wires of her husband's "ear buds." The victim sustained neurologic compromise from the compression of her carotids. (Photo contributor: William S. Smock, MD.)



FIGURE 19.32 ■ Petechial Hemorrhage. Extensive facial petechiae are found above the area of neck compression in a nonfatal strangulation victim. (Photo contributor: William S. Smock, MD.)



FIGURE 19.33 ■ Subconjunctival and Scleral Petechial Hemorrhage. Increased venous pressure from the occlusion of the jugular veins during manual strangulation results in ruptured capillaries. (Photo contributor: William S. Smock, MD.)

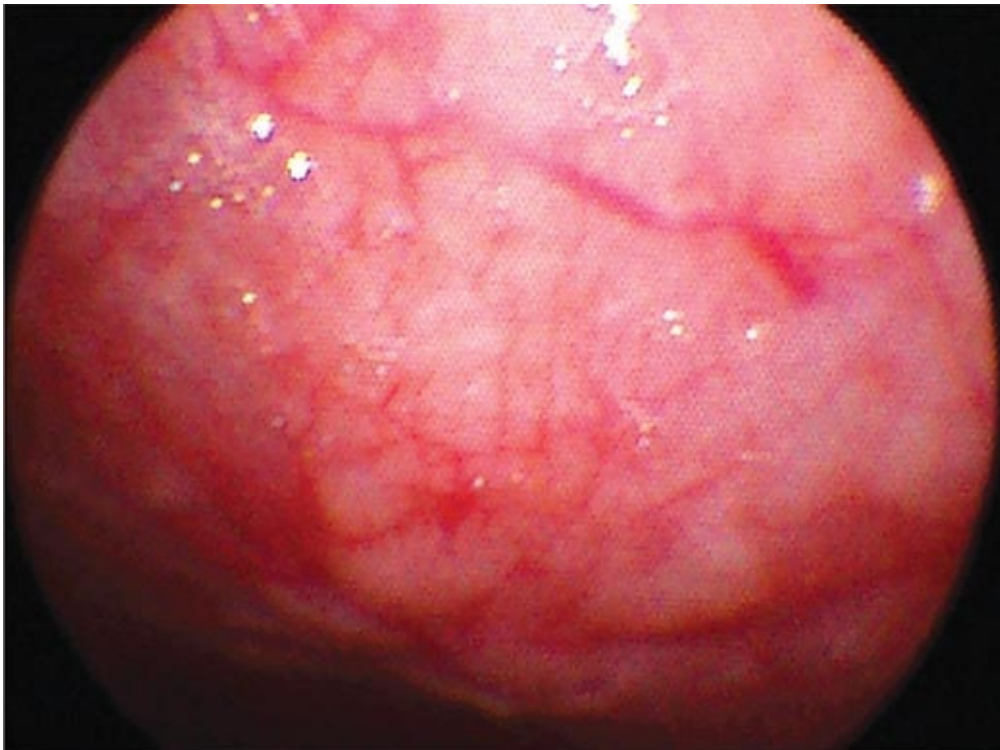


FIGURE 19.34 ■ Petechial Hemorrhage in Posterior Pharynx. Petechial hemorrhage in the posterior pharynx is a common finding in victims of strangulation. (Photo contributor: William S. Smock, MD.)



FIGURE 19.35 ■ Oral-Laryngeal Scope. An oral-laryngeal scope, equipped with a camera, is an excellent tool to document the extent of petechial hemorrhage in the pharynx and vocal cord injury in victims of strangulation. (Photo contributor: William S. Smock, MD.)

The application of external pressure to the neck, even without visible external trauma, can cause serious physical injuries and death. Asymptomatic dissections in both the carotid and vertebral arteries are a known complication of strangulation (Fig. 19.36). Use of cervical computed tomography angiography (CTA) in strangled patients will detect the dissection prior to the victim developing a clot and a subsequent embolic stroke. The use of vascular neck restraints by law enforcement officers has also resulted in carotid artery injuries, including carotid thrombosis (Fig. 19.37). Police officers have sustained embolic strokes (Fig. 19.38) from disruption of carotid plaque during vascular neck restraint trainings.

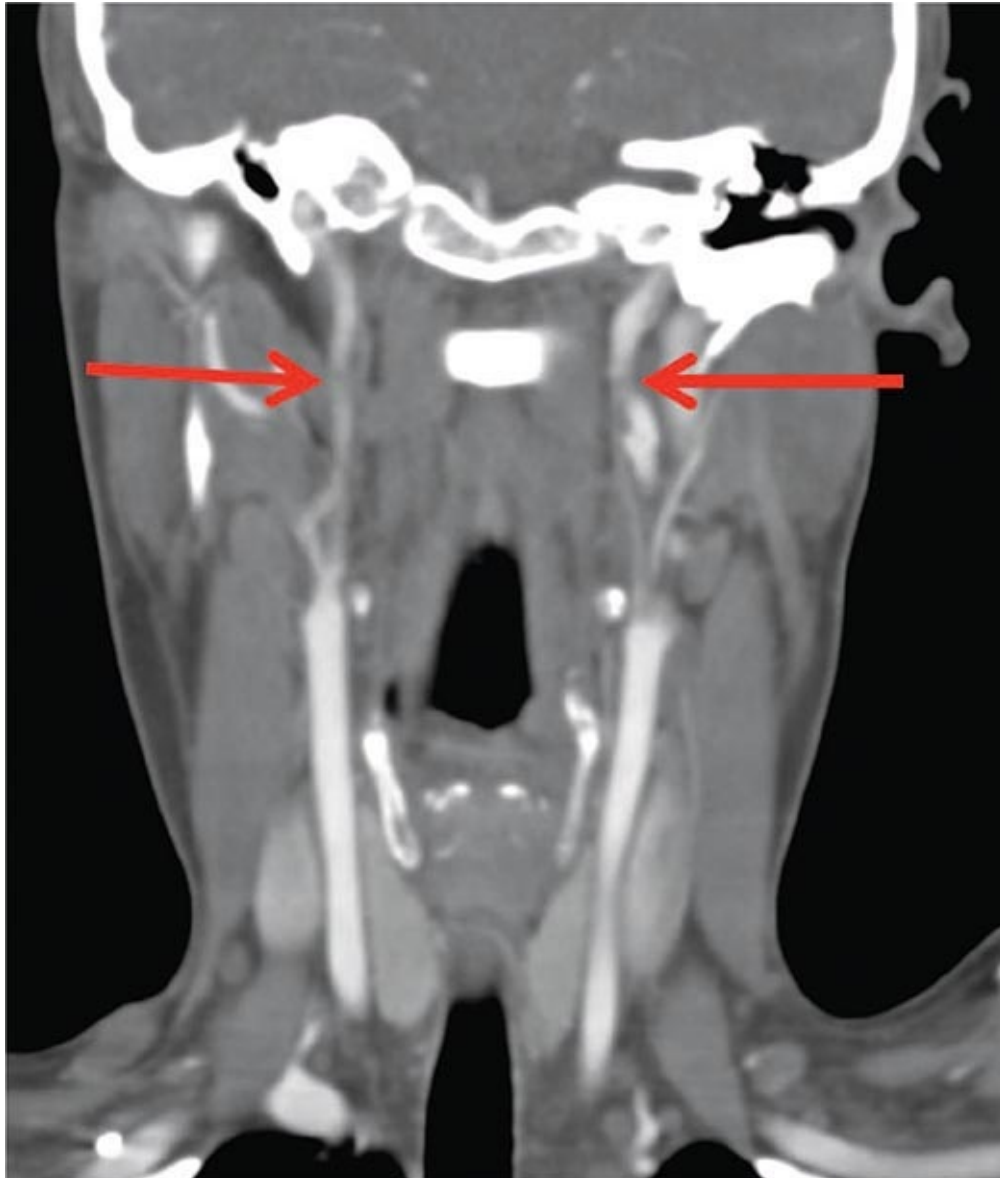


FIGURE 19.36 ■ Carotid Artery Dissection. Bilateral internal carotid artery dissections in a 36-year-old woman who was strangled 6 days prior to presentation to the emergency department. The use of cervical CTA in an asymptomatic patient with no visible neck trauma was lifesaving. (Photo contributor: William S. Smock, MD.)



FIGURE 19.37 ■ Carotid Artery Thrombosis. Complete thrombosis of the internal carotid artery after the application of a vascular neck restraint. (Photo contributor: William S. Smock, MD.)

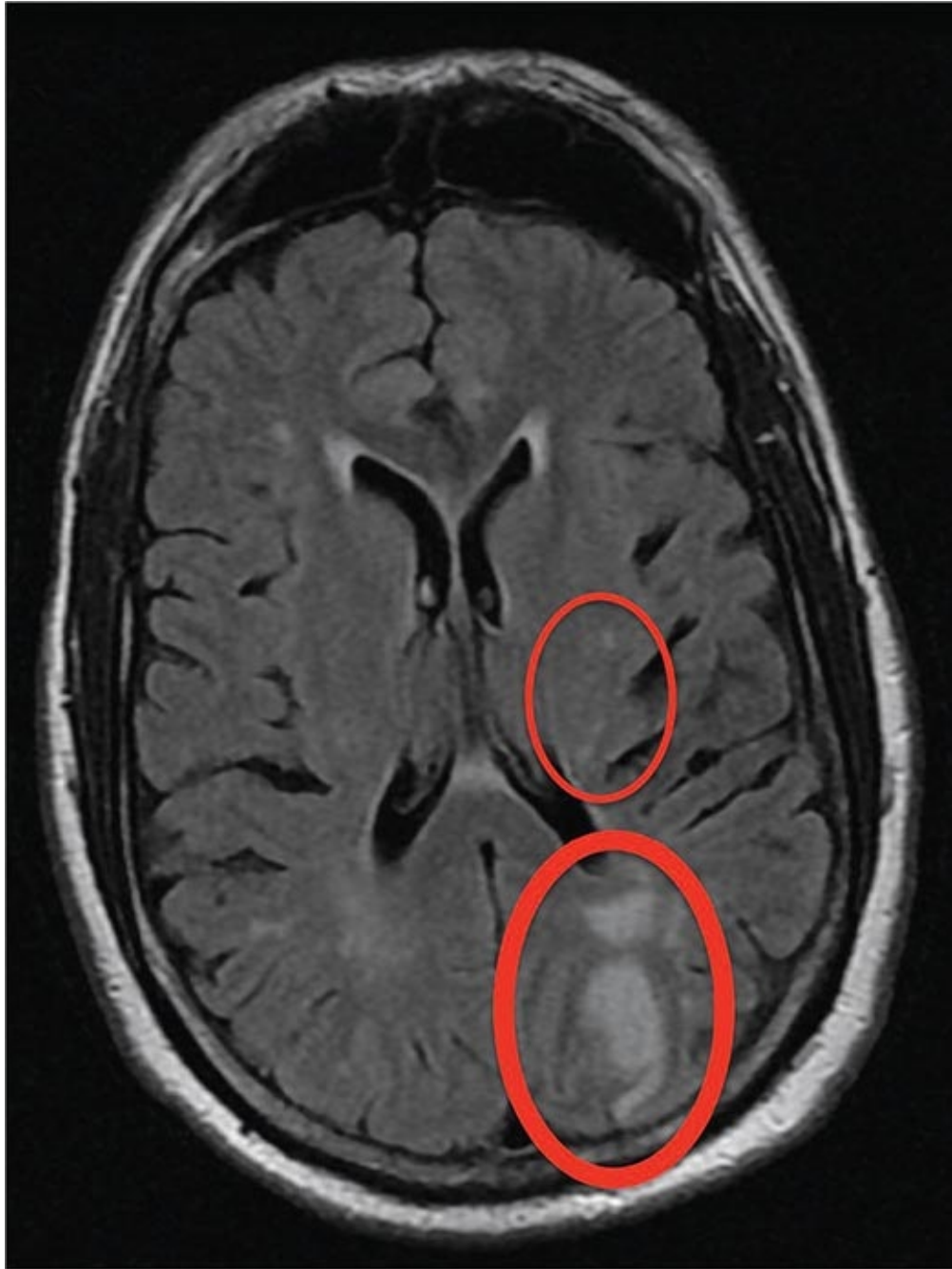


FIGURE 19.38 ■ Embolic Stroke—Magnetic Resonance Imaging (MRI). An MRI of a police officer's brain with multiple areas of infarction. The officer suffered multiple embolic strokes during vascular neck restraint training from disruption of carotid plaques. (Photo contributor: William S. Smock, MD.)

Pearls

1. Strangulation is associated with a 750% increased risk of interpersonal homicide.

2. Application of a ligature (Figs. 19.30 and 19.31) will leave a characteristic mark due to pressure applied over a small surface area.
3. Strangulation-induced petechial hemorrhage, from the rupture of venous vessels, may be found in the conjunctiva (Fig. 19.32), the soft palate, the posterior pharynx (Fig. 19.33), and areas above and below the area where pressure was applied (Fig. 19.34). Petechial hemorrhage is *not* required to prove strangulation. The majority of fatal strangulations exhibit no external evidence of trauma.
4. A victim of strangulation can be rendered unconscious in less than 8 seconds with bilateral occlusion of the carotid arteries. The carotids will be occluded with 11 pounds of pressure. Obtaining a history of neurologic changes—loss of hearing, vision, consciousness, and bowel or bladder control—confirms neurologic injury and an anoxic insult to the brain.
5. Asymptomatic carotid and vertebral artery dissections can occur during a strangulation. Liberal use of neck CTA in victims of strangulation will prevent asymptomatic dissection from going undiagnosed until the victim presents with an embolic stroke or arterial thrombosis (Figs. 19.36–19.38).

THERMAL-PATTERN INJURIES

Clinical Summary

Thermal pattern injuries are commonly seen in cases of abuse and assault, especially when the victims are children and the elderly. A detailed history of the incident should include the position of the patient relative to the thermal source. This will help determine whether the injury was inflicted or accidental. Thermal injuries characterized by a distinct pattern that frequently present to emergency departments are those caused by cigarettes (Fig. 19.39), flatirons (Fig. 19.40), curling irons (Figs. 19.41 and 19.42), and scalding hot water (Fig. 19.43). Water burns will be of two natures: immersion and splash. A sharp or clear line of demarcation between burned and unburned tissue characterizes an immersion or dipping burn. In contrast, splash burns are characterized by an irregular or undulating line or by isolated areas of thermal injury, usually round or oval in shape, caused by droplets of hot liquid. The severity of the scald injury depends on two variables: the length of time that the skin was in contact with the offending substance and the temperature of the substance itself. Tap or faucet

water causes full-thickness thermal damage in 1 second at 70°C (158°F) and in 180 seconds at 48.9°C (120°F). Law-enforcement agencies should routinely measure the household's or institution's water temperature in any investigation involving a scald injury of a child, a developmentally delayed person, or an elderly person. The water temperature will give authorities an estimate of the duration of the insult and therefore the degree of malice of intention on the part of the assailant.



FIGURE 19.39 ■ Cigarette Thermal Injury Pattern. A 24-hour-old cigarette burn is located on this victim's right medial upper eyelid. This victim was held against her will for 48 hours and repeatedly beaten about the face. (Photo contributor: William S. Smock, MD.)



FIGURE 19.40 ■ Clothes-Iron Thermal Injury Pattern. An iron was the weapon used to inflict this thermal injury. The areas of sparing are associated with its steam holes. (Photo contributor: William S. Smock, MD.)



FIGURE 19.41 ■ Curling Iron Thermal Injury Pattern. Curling irons can reach more than 200°F. This temperature can cause a full-thickness burn in less than 1 second. When this burn to the anterior surface of the leg was inflicted, someone's thumb was on the spring lever—evidence of intent ([Fig. 19.42](#)). (Photo contributor: William S. Smock, MD.)



FIGURE 19.42 ■ Curling Iron. The V-shaped configuration of the pattern injury (Fig. 19.41) indicated that the assailant's thumb was on the spring-loaded lever when the burn was inflicted. This was evidence of intent. The victim's aunt confessed to inflicting the burn. (Photo contributor: William S. Smock, MD.)



FIGURE 19.43 ■ Immersion-Line Thermal Injury Pattern. A characteristic “immersion line” is seen in a

thermal-pattern injury. The line of demarcation is associated with the depth of the immersion. (Photo contributor: William S. Smock, MD.)

Pearls

1. A thermal-pattern injury is a common form of abuse seen in infants, institutionalized patients, and the elderly.
2. Emergency physicians must recognize thermal-pattern injuries of abuse.

Chapter 20

HIV CONDITIONS

Stephen P. Raffanti
Anna K. Person



Kaposi Sarcoma (KS). Multiple KS lesions surrounding the nipple of this patient with advanced HIV.
(Photo contributor: Seth W. Wright, MD.)

PRIMARY HIV INFECTION

Clinical Summary

Establishing a definitive diagnosis in the earliest stage of HIV infection (primary infection) can have significant impact on the patient's outcome and may serve to reduce further transmission, leading to public health benefits. There is a high risk of transmission during acute seroconversion due to high HIV viral loads, and patients are often unaware that they are infected during this time. It is believed that a significant number of the new cases of HIV infection occurring in the United States are acquired from acutely infected individuals. Clinical illness accompanies primary HIV infection in approximately two-thirds of patients. The usual time from HIV exposure to the development of symptoms is approximately 10 to 20 days, with average symptom duration of 1.5 to 2 weeks. The most common symptoms following seroconversion mimic a typical viral syndrome and may (or may not) include mucocutaneous lesions and a generalized maculopapular rash located over the face, neck, and trunk. The rash is seen in over 50% of persons with symptomatic primary HIV infection. The lesions are typically small, well circumscribed, erythematous, nonpruritic, and nontender. Less frequently, patients may demonstrate neurologic signs and symptoms consistent with meningoencephalitis, myelopathy, and peripheral neuropathy. If obtained, laboratory studies may show lymphopenia and thrombocytopenia. Because acute seroconversion frequently mimics nonspecific viral illness, it is often unrecognized.

Management and Disposition

Although rapid HIV testing has become feasible in most emergency department (ED) settings, the diagnosis of acute infection cannot rely solely on rapid antibody-based tests. If the possibility of acute seroconversion is entertained, then a discussion should take place with the patient regarding level of risk behavior. If acute HIV is suspected, HIV-1 ribonucleic acid (RNA) quantitative polymerase chain reaction (PCR) is the appropriate diagnostic test. Patients who are acutely sero-converting will often have extremely high levels of HIV RNA. Importantly, a negative HIV antibody test does not rule out HIV infection in these cases. During the "window period" of acute HIV infection, antibodies against the HIV virus have not yet formed and may not be detected for several

weeks. If acute HIV is suspected or confirmed, patients should be educated about disease transmission, assessed for emotional and mental health support, and referred for prompt follow-up and further outpatient testing and evaluation.

Pearls

1. Maintain a high degree of clinical suspicion for acute primary HIV infection, especially when sexually active patients or patients who use intravenous drugs present with mononucleosis-like symptoms, unexplained rash, mucocutaneous ulcers, lymphadenopathy, or aseptic meningitis.
2. Obtain an HIV RNA quantitative PCR (and not just an HIV antibody test) if acute HIV is suspected.
3. Ensure proper follow-up for patients in whom the diagnosis of acute HIV infection is entertained, and counsel patients regarding potential risk of transmission.
4. In those who have previously achieved HIV viral suppression on antiretroviral therapy, stopping therapy can lead to viral rebound, which can mimic primary infection.



FIGURE 20.1 ■ Primary HIV Infection. A maculopapular rash is seen in over half of persons with symptomatic acute HIV infection. This less typical papular/vesicular rash was present in a patient with primary HIV infection. (Photo contributor: Gregory K. Robbins, MD, MPH.)

IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME (IRIS)

Clinical Summary

Effective antiretroviral therapy (ART) for HIV establishes viro-logic suppression and can allow the patient's immune system to rebuild to a near-normal competency. In some patients who develop a rapid immune response following ART initiation after a very advanced degree of immune suppression, the immune reconstitution inflammatory syndrome (IRIS) can develop. In these patients, it is thought that previously sub-clinical infections manifest with the development of a more robust immune status. IRIS is most often seen in patients who have recently started ART (usually within 60 days of starting therapy) with an initial CD4 count below 100 cells/mm³. The most common infections that can “trigger” IRIS include *Mycobacterium* (tuberculosis and *Mycobacterium avium* complex [MAC]), cytomegalovirus (CMV), cryptococcal disease, and histoplasmosis. Patients typically present with fever, malaise, lymphadenopathy, and symptoms associated with the active opportunistic infection.

Management and Disposition

The key for the ED physician is to identify patients who have recently started ART and may be presenting with IRIS-related findings. Appropriate evaluation and management depend on the underlying opportunistic infection. Patients will often require admission to the hospital and consultation with an infectious diseases specialist.

Pearl

1. HIV-infected patients who are currently taking ART and present with a febrile illness should be questioned regarding the duration of ART and pretreatment CD4 count. Patients with advanced disease who have recently started ART may be presenting with signs and symptoms of IRIS-related disease.



FIGURE 20.2 ■ IRIS. This patient presented with fever and diffuse lymphadenopathy secondary to MAC infection. The patient had no manifestations of his disseminated disease until being started on anti-retroviral therapy. (Photo contributor: Stephen P. Raffanti, MD.)

ORAL HAIRY LEUKOPLAKIA

Clinical Summary

Oral hairy leukoplakia (OHL) is a disease of the lingual squamous epithelium caused by the Epstein-Barr virus (EBV). OHL generally affects the lateral portion of the tongue, although the floor of the mouth, palate, or buccal mucosa may also be involved. The lesions are white corrugated plaques that, unlike *Candida*, cannot be scraped from the surface to which they adhere. Most often OHL is asymptomatic, although occasionally this condition can be painful. Diagnosis is usually clinical, although definitive diagnosis can be made by biopsy characteristically revealing acanthosis and parakeratosis.

Management and Disposition

Patients who are known to be HIV seropositive can be educated about the

disease and reassured. OHL is not considered to be a premalignant lesion. If the patient is not known to be infected with HIV, primary care provider referral and HIV screening, if available, should be offered.

Pearls

1. Oral candidiasis can be distinguished from OHL by using a swab in an attempt to remove the exudate characteristic of thrush and by observing pseudohyphal elements microscopically with *Candida*. OHL cannot be scraped off and usually involves the lateral aspect of the tongue.
2. OHL is fairly specific for HIV infection as it is rarely observed in patients with other immunodeficiencies. If OHL is identified in a patient not known to be HIV infected, risk factors and HIV screening options should be discussed and performed.



FIGURE 20.3 ■ Oral Hairy Leukoplakia. Typical-appearing lesions on side of tongue in this patient with HIV. (Photo contributor: Robert Brandt, MD.)



FIGURE 20.4 ■ Oral Hairy Leukoplakia. Exudate does not scrap off the tongue in oral hairy leukoplakia, differentiating it clinically from oral thrush. (Photo contributor: Kevin J. Knoop, MD, MS.)

CANDIDIASIS ASSOCIATED WITH HIV

Clinical Summary

Oral *Candida* infections are often seen in individuals with HIV/AIDS, with the severity of infection correlating with the degree of immunosuppression. Oral candidiasis can occur at all stages of HIV disease. The usual causative agent is *Candida albicans*, but other *Candida* species have been isolated. Oral candidiasis, or “thrush,” can be classified as pseudomembranous, angular, or erythematous. Pseudomembranous candidiasis can be diagnosed by identifying removable whitish plaques on the tongue, uvula, and buccal mucosa. Erythematous or atrophic candidiasis appears as smooth red patches along the soft and hard palate. Although isolated oral candidiasis is not an AIDS-defining illness (although esophageal candidiasis is), oral candidiasis is an indication for pneumocystis prophylaxis regardless of CD4⁺ cell count.

Vaginal candidiasis is common in HIV-positive patients and can cause a severe whitish discharge with vulvar erythema.

Management and Disposition

Patients presenting to the ED for dysphagia or odynophagia thought to be due to candidiasis should be evaluated for dehydration and may require inpatient admission. Most cases of minimally symptomatic oral candidiasis respond well to nystatin suspension or clotrimazole troches. More symptomatic oral or esophageal candidiasis usually responds to fluconazole. Rare cases of resistant candidiasis may require intravenous echinocandin therapy and consultation with an infectious disease physician.

Pearls

1. Symptomatic oral and esophageal candidiasis is still a common ED presentation in HIV patients who are not receiving or are not compliant with effective ART.
2. The diagnosis of oral candidiasis in a patient not already diagnosed with HIV infection should lead to a discussion of risk factors and should prompt HIV screening.
3. Mildly symptomatic oral, esophageal, or vaginal candidiasis usually responds to a one-time oral dose of fluconazole. Close clinical follow-up should be arranged.



FIGURE 20.5 ■ Oral Candidiasis. Removable whitish plaques on the palate are seen in this HIV infected patient with pseudomembranous candidiasis. (Photo contributor: Thea James, MD.)

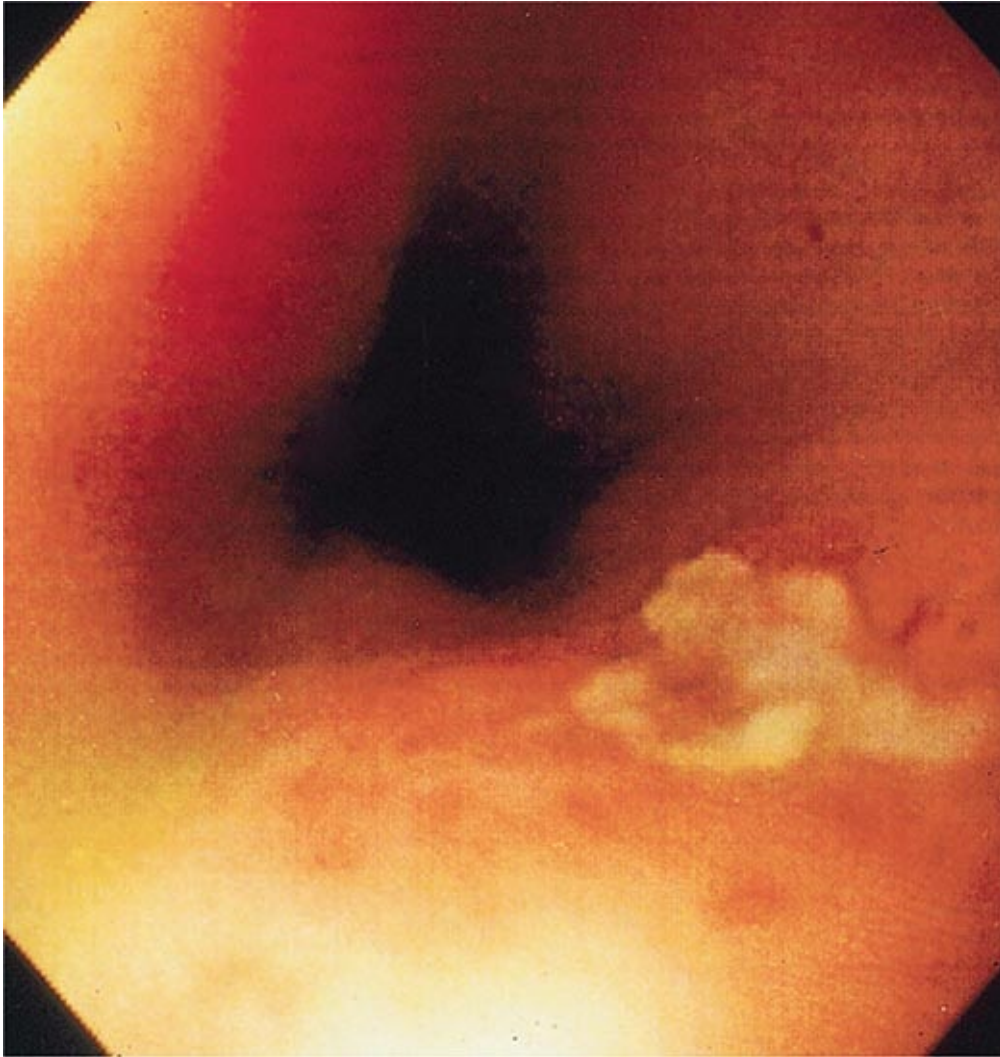


FIGURE 20.6 ■ Esophageal Candidiasis. Endoscopy demonstrating esophageal candidiasis in this HIV-infected patient. (Photo contributor: Edward C. Oldfield III, MD.)



FIGURE 20.7 ■ Oral Thrush. Typical plaque lesions are seen on the palate and uvula of this HIV patient. (Photo contributor: Seth W. Wright, MD.)

KAPOSI SARCOMA

Clinical Summary

Kaposi sarcoma (KS) is a low-grade vascular tumor associated with human herpesvirus 8 (HHV-8). Since the introduction of highly active antiretroviral therapy (HAART), the incidence of KS in HIV-infected persons has significantly declined. Skin involvement is characteristic, but extracutaneous spread of KS is

common, particularly to the oral cavity, gastrointestinal (GI) tract, and respiratory tract. The skin lesions appear most often on the lower extremities, face (especially the nose), oral mucosa, and genitalia. Most commonly, the lesions are papular, ranging in size from several millimeters to centimeters in diameter. Less commonly, the lesions may be plaque-like, especially on the soles of the feet, or exophytic and fungating with breakdown of overlying skin. Papular lesions may resemble lesions associated with bacillary angiomatosis or herpesvirus infections.

Pulmonary involvement can occur in AIDS-related KS. Affected persons may present with shortness of breath, fever, cough, hemoptysis, or chest pain or as an asymptomatic finding on chest x-ray. Diagnosis can be confirmed via bronchoscopy.

Management and Disposition

All HIV-infected patients with KS should receive ART, which is generally effective in treating most cutaneous lesions. Systemic chemotherapy and immunomodulator therapy (interleukin-12, angiogenesis inhibitors) are reserved for more extensive disease including patients with visceral involvement.



FIGURE 20.8 ■ Kaposi Sarcoma. A single violaceous plaque is seen on the face of an HIV-positive patient. (Photo contributor: George W. Turiansky, MD.)

Pearls

1. HIV patients who present with new papular lesions should be referred for dermatologic evaluation and biopsy.
2. A careful examination of the skin and oral cavity is appropriate in HIV-infected patients presenting with GI or respiratory complaints.
3. KS herpesvirus inflammatory cytokine syndrome (known as KICS) should be suspected when a patient with HIV and KS presents with fever, respiratory or GI symptoms, or a sepsis-like picture.



FIGURE 20.9 ■ Cutaneous Kaposi Sarcoma. Characteristic violaceous KS lesions on the leg of this Caucasian patient with HIV. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 20.10 ■ Kaposi Sarcoma. Multiple black KS lesions, as typically seen in darker skinned patients. (Photo contributor: Seth W. Wright, MD.)



FIGURE 20.11 ■ Kaposi Sarcoma. Multiple raised KS lesions are seen on the leg of this patient with advanced HIV. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 20.12 ■ Kaposi Sarcoma. Multiple KS lesions surrounding the nipple of this patient with advanced HIV. (Photo contributor: Seth W. Wright, MD.)



FIGURE 20.13 ■ Oral Kaposi Sarcoma—Tongue. Raised palpable erythematous KS on the tongue of this HIV-infected patient. (Photo contributor: Seth W. Wright, MD.)

TOXOPLASMA GONDII INFECTION

Clinical Summary

Toxoplasma gondii is a widespread intracellular protozoan parasite with a definitive host stage in cats. Immunocompetent persons are usually asymptomatic, but immunocompromised persons (especially HIV-positive persons with <100 CD4 cells/mm³) are susceptible to reactivation of latent disease with considerable morbidity and mortality.

Patients with central nervous system (CNS) toxoplasmosis most often present with symptoms consistent with intracranial mass lesions (headache, focal neurologic deficits, nausea/emesis, and/or seizure) or, less likely, encephalitis

(fever, confusion, altered mental status). Contrasted computed tomography (CT) of the head typically reveals ring-enhancing lesions with a predilection for the basal ganglia. Magnetic resonance imaging (MRI) of the head is more sensitive than CT for identifying these lesions. Serologic tests for *T gondii* antibodies (IgG) are usually positive. Other causes of mass lesions in the brain in HIV-infected individuals include CNS lymphoma, cryptococcoma, tuberculoma, and brain abscess.

Ocular toxoplasmosis is a less frequent complication of HIV disease. Patients typically present with eye pain and decreased visual acuity. Retinitis may be diagnosed by ophthalmoscopic evaluation revealing characteristic exudates and hemorrhage. Toxoplasmosis retinitis appears as raised yellow-white, cottony lesions in a nonvascular distribution, unlike the edematous perivascular exudates of CMV retinitis.

Management and Disposition

There are two combination regimens considered to be first choice for the treatment of toxoplasmosis. The most commonly used regimen is pyrimethamine and sulfadiazine for 6 weeks or until neurologic findings have resolved; however, for those with sulfa allergies, pyrimethamine plus clindamycin is an alternative. If the patient presents with seizures, loading doses of antiepileptics such as fosphenytoin or levetiracetam should be administered along with benzodiazepines. All patients with acute symptomatic CNS toxoplasmosis infection should be admitted for treatment.

Pearls

1. HIV-infected patients presenting with focal neurologic findings or seizures should be evaluated for meningitis and space-occupying lesions in the brain. AIDS-specific differential diagnoses include toxoplasmic encephalitis, CNS lymphoma, tuberculosis, and cryptococcal disease.
2. All HIV-infected patients should have a CNS space-occupying lesion excluded by imaging prior to lumbar puncture (LP).
3. Initial evaluation of the HIV-infected patient with possible CNS lesions should include CT or MRI imaging, LP, and triage management for bacterial meningitis. Further workup will require admission and infectious diseases consultation.



FIGURE 20.14 ■ *Toxoplasma gondii* Infection. Contrast head CT showing typical multiple ring-enhancing lesions seen in *T gondii* CNS infection. (Photo contributor: Edward C. Oldfield III, MD.)

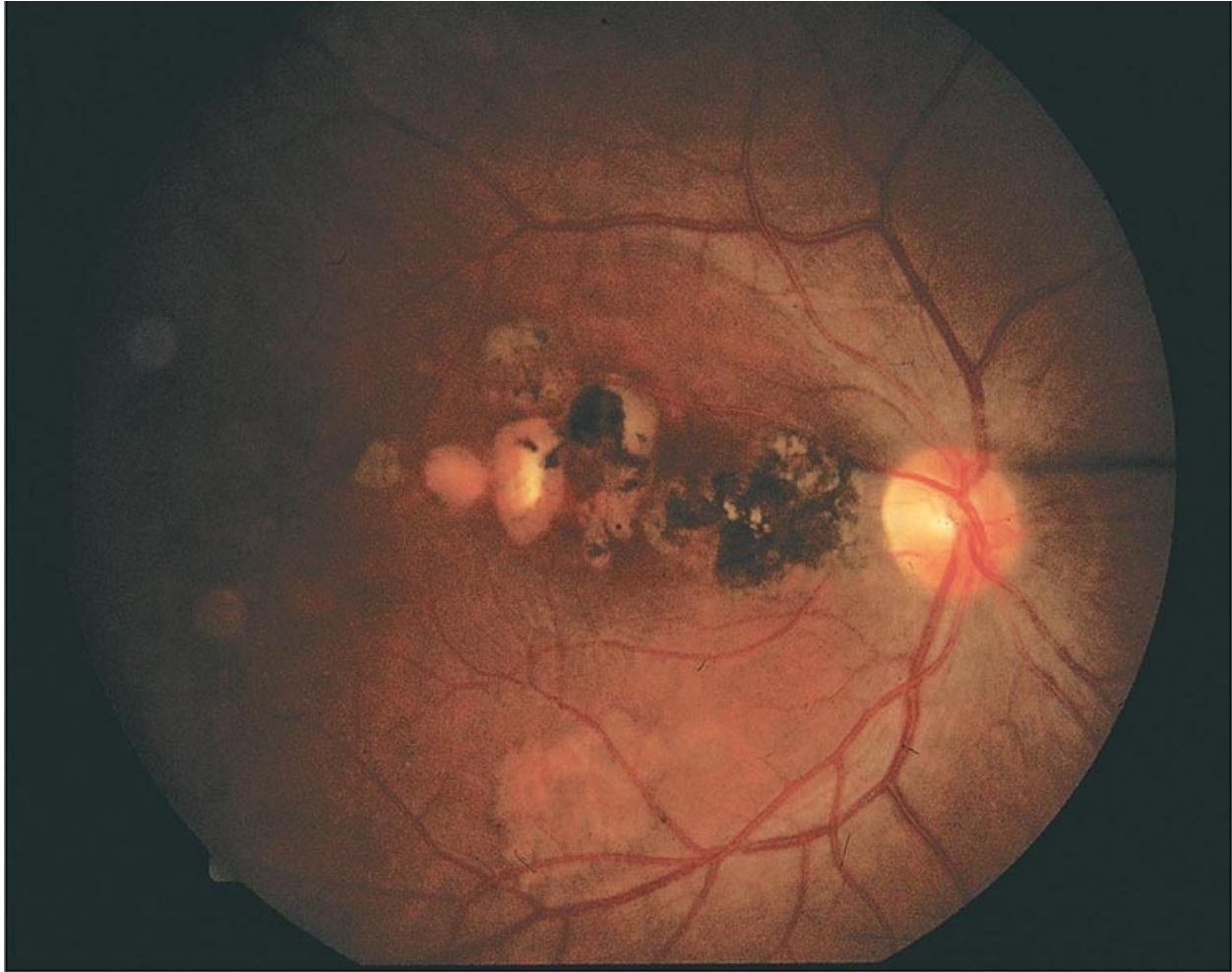


FIGURE 20.15 ■ Toxoplasmosis Retinitis. Ocular toxoplasmosis is a common complication of HIV disease. The lesion is a focal destructive chorioretinitis that leaves well-defined, heavily pigmented scars, especially in the macular area. (Photo contributor: Department of Ophthalmology, Naval Medical Center, San Diego, CA.)

PNEUMOCYSTIS

Clinical Summary

Pneumocystis jirovecii (formerly *carinii*) pneumonia (PJP) is the most common opportunistic infection in HIV-infected patients. Clinical suspicion for PJP pneumonia in any HIV patient presenting with complaints of dyspnea and nonproductive cough should remain high, especially in those with CD4 counts <200 cells/mm³ who are not on appropriate PJP prophylaxis. Presentations can be indolent, acute, or subacute, with associated symptoms including fever,

fatigue, anorexia, weight loss, and chest pain. The complete blood count is usually normal except for lymphopenia, whereas serum lactate dehydrogenase (LDH) is often elevated. Arterial blood gases (ABGs) most often reveal a respiratory alkalosis, Po_2 of 70 mm Hg or less, and an increased A-a gradient of 35 mm Hg or more. Radiographic chest findings are variable with diffuse interstitial alveolar infiltrates being common. Patients may have normal oxygen saturation levels at rest, but with ambulation, they often have a rapid desaturation.

Management and Disposition

The initial treatment of PJP is often based on a presumptive diagnosis pending definitive results, which is most often obtained through a bronchial lavage sample via bronchoscopy. It is essential that any individual with HIV with respiratory signs or symptoms and a CD4 count below 200 cells/mm³ or CD4 cell percent less than 14 be evaluated for PJP. Initial evaluation should include history of medications, especially adherence with prophylactic medications, clinical exam, chest x-ray, and room air ABG. Trimethoprim-sulfamethoxazole, orally or intravenously, is the first-line standard treatment. Clindamycin-primaquine, pentamidine, atovaquone, and trimethoprim-dapsone are less effective alternatives. Treatment with corticosteroids should be initiated if the Po_2 is less than 70 mm Hg or the A-a gradient is greater than 35 mm Hg. Any patient treated with steroids for presumptive PJP must have a definitive diagnosis made before discharge, as other pathogens that can cause pneumonia in HIV-infected patients may initially respond to steroid therapy.

Pearls

1. Include PJP in the differential diagnosis of any HIV patient who presents with a persistent fever or respiratory complaint.
2. Patients with significant dyspnea should have a room air ABG obtained. Oxygen saturation measures alone are not adequate to evaluate oxygenation level or the need for adjunct steroid therapy in a patient with suspected PJP.
3. The classic clinical presentation of PJP is a quiet patient with an increased respiratory rate, poor air movement, and a nonproductive cough on deep inspiration.
4. Over 30% of patients with PJP will have a normal chest x-ray. CT scanning of

the chest is much more sensitive to reveal the classic interstitial ground-glass pattern.



FIGURE 20.16 ■ *Pneumocystis jirovecii* Pneumonia (PJP). Chest radiograph showing diffuse interstitial alveolar infiltrates of PJP. (Photo contributor: Edward C. Oldfield III, MD.)

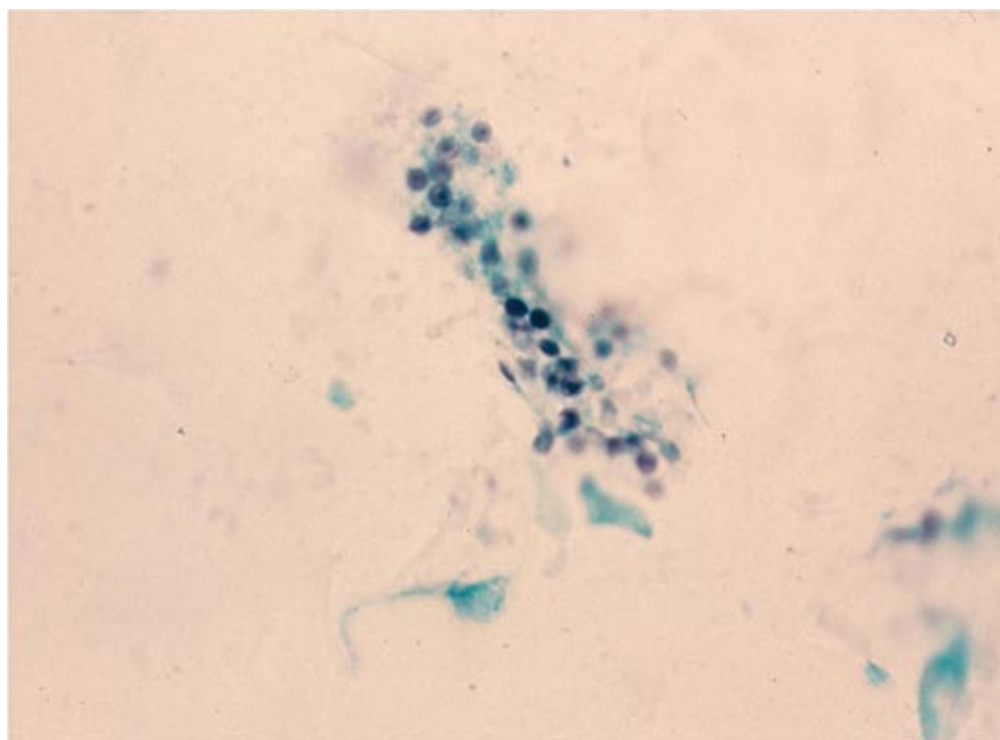


FIGURE 20.17 ■ PJP on Silver Stain. *Pneumocystis jirovecii* seen on methenamine-silver stain of a bronchoalveolar lavage sample from an HIV patient with PJP. (Photo contributor: Lawrence B. Stack, MD.)

CRYPTOCOCCAL INFECTIONS

Clinical Summary

Cryptococcus neoformans is the most common cause of meningitis in patients with HIV/AIDS. Cryptococcal meningoencephalitis typically manifests itself in patients whose CD4 cell counts are less than $50/\text{mm}^3$. The onset tends to be insidious with fairly nonspecific symptoms such as fever, nausea, and headache. Symptoms may be present for several weeks, and diagnostic delay is common. Seizures or focal neurologic presentations are rare, and neck stiffness and/or photophobia are usually absent. Diagnosis is usually made on examination of cerebrospinal fluid (CSF). Opening pressures may be quite elevated on LP, and CSF values usually reveal a normal CSF glucose concentration, a mildly elevated CSF protein concentration, and a CSF leukocyte count of less than 20/mL. India ink staining shows the organisms directly with an approximate sensitivity of 70%, whereas CSF cryptococcal latex antigen testing has a sensitivity approaching 90%. Fungal CSF cultures should also be sent. Cutaneous manifestations are seen in disseminated disease.

Management and Disposition

Treatment should begin for presumptive meningitis pending completion of CSF studies. Most often, the decision to treat for cryptococcal meningitis will be based on the results of CSF studies, along with consultation with infectious disease specialists. *Cryptococcus* can also cause pneumonia and skin lesions. Treatment regimens are usually based on amphotericin preparations plus flucytosine, followed by fluconazole.

Pearls

1. Perform the LP only after CT with contrast rules out space-occupying lesions, and do so with the patient in a lateral decubitus position so as to obtain an accurate opening pressure.

2. CSF antigen testing is most often used to obtain diagnosis, and repeat LPs may be needed to manage elevated intracranial pressures. India ink stains may also be positive with other encapsulated organisms such as *Klebsiella pneumoniae*, *Rhodotorula*, *Candida*, and *Proteus* species.



FIGURE 20.18 ■ Cryptococcal Infection. Cryptococcal skin lesions in disseminated form. Note that the umbilicated centers give a similar appearance to that of molluscum contagiosum. (Photo contributor: Briana Hill, MD.)



FIGURE 20.19 ■ Cryptococcal Infection. Cutaneous cryptococcal lesions are often clinically difficult to distinguish from other cutaneous eruptions. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 20.20 ■ Cryptococcal Infection. This patient had extensive cutaneous involvement with disseminated cryptococcal infection. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

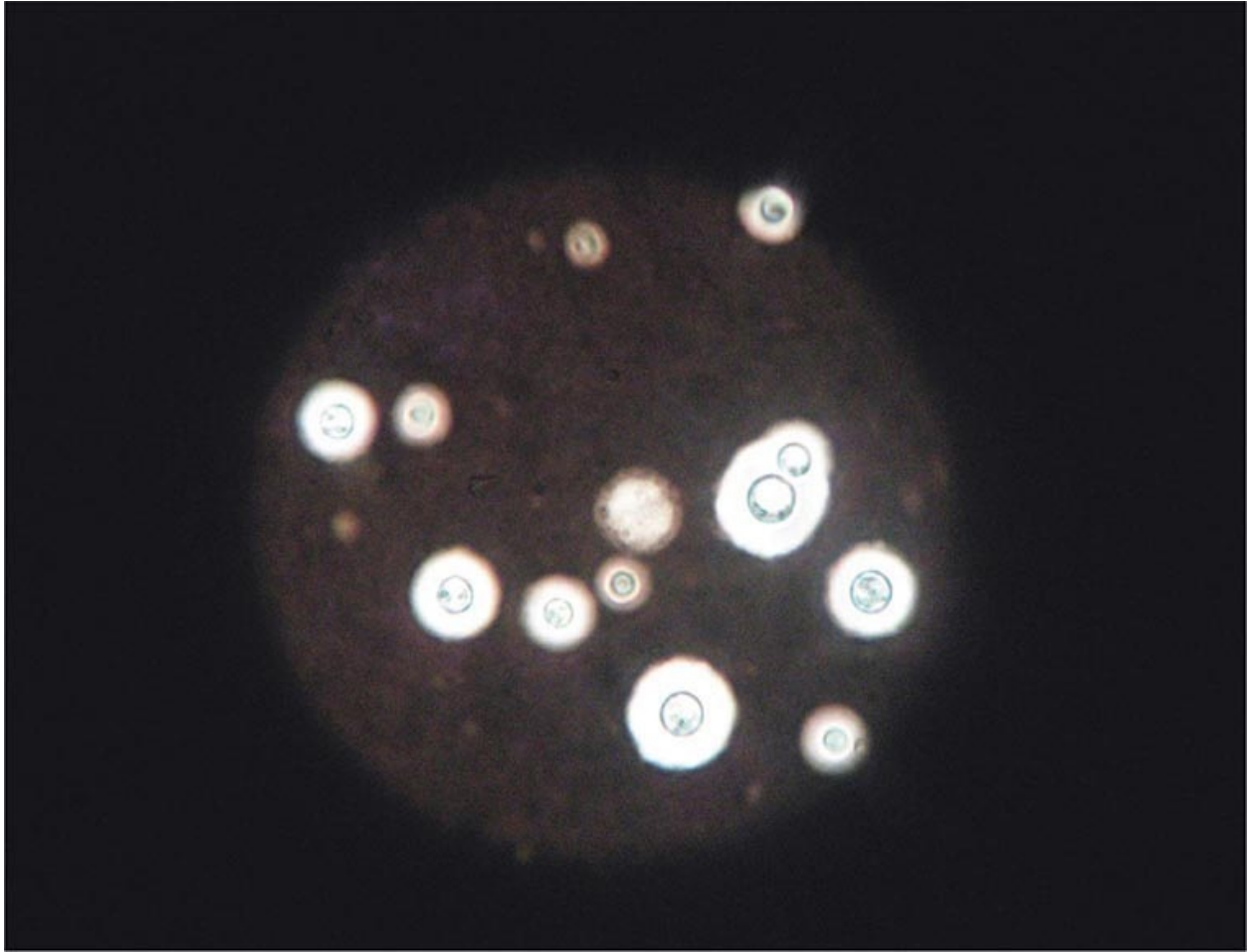


FIGURE 20.21 ■ India Ink Preparation. *Cryptococcus neoformans* seen as encapsulated yeast on India ink preparation of the CSF from an HIV patient with cryptococcal meningitis. (Photo contributor: Seth W. Wright, MD.)

HISTOPLASMOSIS

Clinical Summary

Disseminated histoplasmosis most commonly occurs in immunocompromised patients living in the Ohio and Mississippi River Valleys. HIV-infected patients with disseminated histoplasmosis usually have a CD4 count <200 cells/mm³ and present with fever, weight loss, malaise, and pulmonary symptoms such as cough and dyspnea. Pulmonary symptoms predominate, and in severe cases, patients may present with acute respiratory distress syndrome. Patients may present with a diffuse papular rash in disseminated disease. Mediastinitis is a rare but serious

complication of histoplasmosis. Urine and serum antigen testing carries a high sensitivity, but bronchoalveolar lavage may be needed to make the diagnosis in cases of pulmonary involvement. Serum LDH and alkaline phosphatase levels may be markedly elevated as well.



FIGURE 20.22 ■ Disseminated Histoplasmosis. Skin lesions indicative of cutaneous involvement in an AIDS patient with disseminated histoplasmosis. (Photo contributor: Stephen P. Raffanti, MD.)

Management and Disposition

Disseminated histoplasmosis should be considered in any patient from an endemic region with moderate to advanced HIV infection and constitutional symptoms. Acutely ill patients with fever, weight loss, and functional decline should be admitted for evaluation treatment. Some patients with less severe disease may be treated with oral itraconazole but should be referred for evaluation and a definitive diagnosis.

Pearl

1. Consider disseminated histoplasmosis in any patient with moderate to advanced HIV infection and constitutional symptoms, rash, and appropriate

geographic exposure.



FIGURE 20.23 ■ Pulmonary Histoplasmosis. Chest x-ray demonstrating severe miliary histoplasmosis in a patient with AIDS. The patient presented with fever, cough, and respiratory distress. (Photo contributor: Jake Block, MD.)

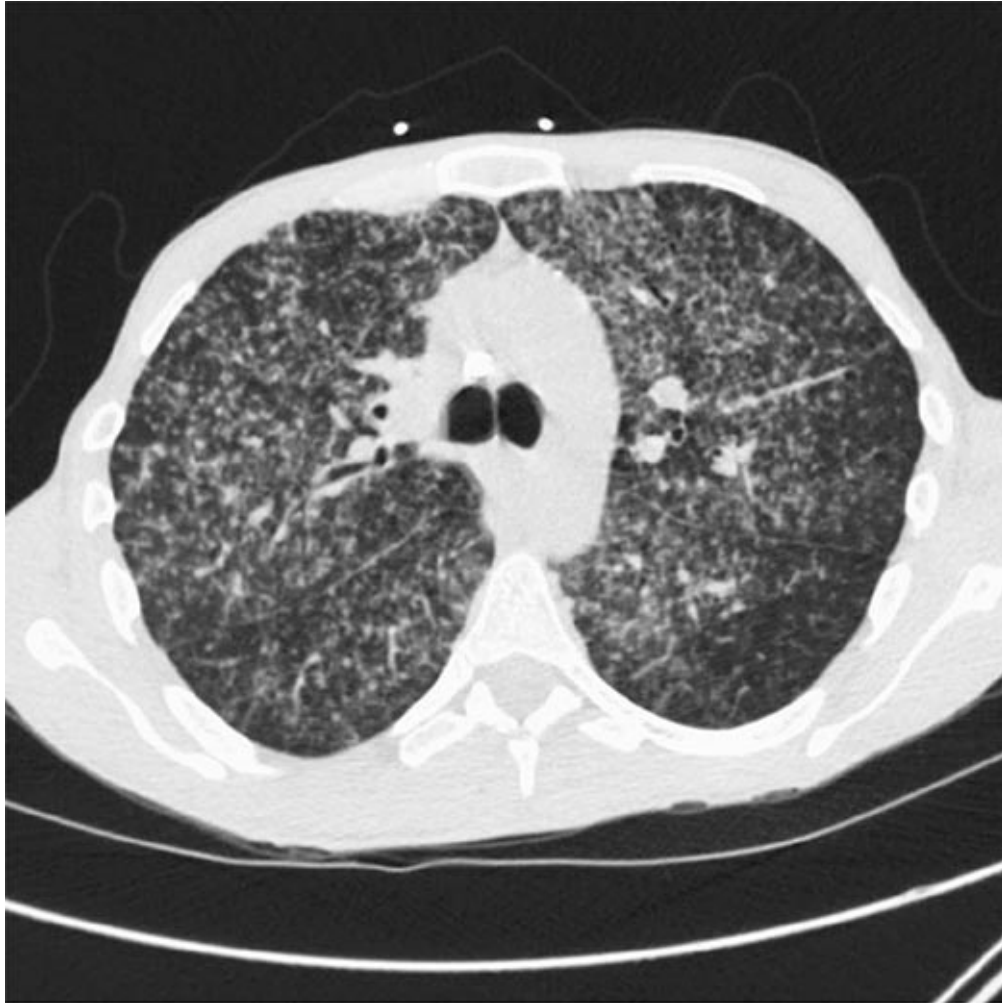


FIGURE 20.24 ■ Pulmonary Histoplasmosis. CT scan from the same patient in [Fig. 20.23](#). (Photo contributor: Jake Block, MD.)

CYTOMEGALOVIRUS INFECTIONS

Clinical Summary

Cytomegalovirus (CMV) can be a cause of considerable morbidity in severely immunocompromised HIV-infected individuals. The major problems encountered are, in order of frequency, retinitis, colitis, esophageal ulceration, encephalitis, and pneumonitis.

CMV retinitis is the most common cause of blindness and eye disease in patients with HIV/AIDS. Patients typically present with loss of vision, floaters, scotoma, or visual field loss. Fundoscopic examination reveals exudates and

hemorrhages, which follow the vasculature of the retina, giving it the typical “pizza pie” or “cottage cheese and ketchup” appearance.

CMV colitis is an uncommon but serious complication of HIV. The usual presenting features include generalized abdominal pain, diarrhea (which may be bloody), and a low-grade fever. Loops of dilated large bowel may be seen on abdominal imaging, but definitive diagnosis is made with mucosal biopsy revealing characteristic “owl’s eye” inclusion bodies.

Management and Disposition

Treatment for CMV retinitis can be sight-saving and should be started as soon as the diagnosis is considered. Emergent ophthalmologic consultation is needed. Treatment can be initiated with intravenous, intravitreal, or oral ganciclovir formulations. Relapse is common with CMV retinitis, and if left untreated, it may progress to complete blindness.

Pearls

1. Ocular complaints in HIV patients require a comprehensive eye examination along with ophthalmology involvement if lesions are observed or complaints are significant.
2. Patients presenting with odynophagia or dysphagia may require esophagogastroduodenoscopy to evaluate for esophageal involvement of CMV (and to distinguish from other causes, like *Candida*). Those with severe immunosuppression, diarrhea, and CMV viremia may require colonoscopy.



FIGURE 20.25 ■ Cytomegalovirus Retinitis. Fundusoscopic examination shows exudates and hemorrhages (“cottage cheese and ketchup” appearance) seen with CMV retinitis. (Photo contributor: Edward C. Oldfield III, MD.)

HERPES ZOSTER

Clinical Summary

Varicella-zoster virus (VZV) infection presents as shingles in HIV-infected patients at a greater frequency and severity than the general population. Despite the high incidence of shingles in this population, severe disseminated disease is rare.

The diagnosis of herpes zoster in the ED is often made clinically, by either recognizing the pain syndrome or visualizing the rash. Patients can present with a well-defined area of hyperesthesia for days prior to an outbreak of the characteristic lesions. Immunocompromised patients may demonstrate multidermatomal distribution or scattered vesicles at a distant site.

Approximately 20% of patients will have systemic symptoms such as malaise, fever, headache, or fatigue. Culture, serologic testing, or PCR confirms the diagnosis, but treatment may be empiric based on the clinical appearance of the lesions or a well-defined area of hyperesthesia. A Tzanck test can be obtained by scraping the base of a lesion in an attempt to demonstrate multinucleated giant cells.

Management and Disposition

Three antiviral drugs are used to treat herpes zoster infections: acyclovir, famciclovir, and valacyclovir. Patients with severe disseminated disease or ophthalmic zoster should receive intravenous acyclovir (10-12.5 mg/kg intravenously every 8 hours for 10-14 days), often in conjunction with infectious diseases consultation. Treatment of zoster ophthalmicus should include topical antibiotics and immediate ophthalmology consultation. Providing treatment of the disease within 72 hours of rash onset will result in a more rapid resolution of cutaneous lesions and decrease viral shedding but will not change the incidence of postherpetic neuralgia. Immunocompromised patients should not be placed on glucocorticoids. Narcotics, capsaicin cream, and tricyclic antidepressants can be used for pain control.

Pearls

1. Worsening cases of herpes zoster, complicated herpes zoster, or ophthalmic zoster all require intravenous acyclovir and admission.
2. Herpes zoster encephalitis is rare but can occur months after the cutaneous phase and can be difficult to diagnose. Common presenting symptoms include mental status changes, headache, fever, photophobia, vomiting, or even focal neurologic deficits.
3. Patients presenting with localized hyperesthesia may have a zoster-related prodrome, and treatment should be initiated if clinical suspicion is high.
4. The presence of vesicles on the tip of the nose (Hutchinson sign) indicates involvement of the nasociliary branch of cranial nerve V and is associated with a higher risk of ocular involvement.



FIGURE 20.26 ■ HZV (Shingles). A painful eruption of many tiny vesicles on an erythematous base in this patient with HIV. (Photo contributor: Jeffery Gibson, MD.)



FIGURE 20.27 ■ HZV—Multiple Dermatomes. Severe painful shingles (vesicles and bullae) spanning multiple cutaneous dermatomes in this HIV patient. (Photo contributor: John O'Mara, MD.)



FIGURE 20.28 ■ Disseminated Herpes Zoster Infection. Vesicles are seen over the entire face, representing disseminated HZV infection (multiple dermatomal distributions). (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)



FIGURE 20.29 ■ Ophthalmic Herpes Zoster. This patient has vesicles in the ophthalmic division of cranial nerve V bilaterally. (Photo contributor: Daniel L. Savitt, MD.)

EOSINOPHILIC FOLLICULITIS

Clinical Summary

Eosinophilic folliculitis, commonly seen in advanced HIV patients, is a pruritic skin eruption usually involving the face, neck, trunk, and extremities manifesting in the form of pustules and papules. The skin lesions usually start as small pustular groups that later coalesce to create irregular erosions and plaques with central hyperpigmentation. The associated pruritus is often so intense that the lesions become excoriated. Most patients with this condition will demonstrate CD4 cell counts of less than $250/\text{mm}^3$. The etiology of eosinophilic folliculitis is unknown but is commonly thought of as an inflammatory process associated

with immune dysfunction. It has become less common with the advent of HAART.

Management and Disposition

The diagnosis requires dermatology consultation and referral for biopsy. Antihistamines, potent topical steroids, itraconazole, topical permethrin, retinoids, metronidazole, dapsone, and ultraviolet B phototherapy have all shown variable levels of efficacy.

Pearls

1. The severe pruritus associated with this condition helps distinguish it from bacterial folliculitis. Symptomatic treatment for pruritus is necessary, and the disease generally improves with effective ART.
2. Dermatologic referral is warranted prior to initiating definitive therapy.
3. Patients with eosinophilic folliculitis may present with prurigo nodularis and lichen simplex chronicus as a result of severe itching and rubbing.
4. Secondary syphilis should be considered in all HIV-infected patients presenting with rash.



FIGURE 20.30 ■ Eosinophilic Folliculitis. The rash of eosinophilic folliculitis consists of small groups of pustules and vesicles, as seen on the face of this patient. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)



FIGURE 20.31 ■ Eosinophilic Folliculitis. A magnified view showing pustules at the base of each hair follicle. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

HERPES SIMPLEX VIRUS

Clinical Summary

Infection with herpes simplex virus (HSV) is extremely common in HIV-infected patients and may present with oral, genital, anal, esophageal, or ophthalmologic involvement. The hallmark for most clinical presentations of HSV outbreaks is an inflammatory cutaneous eruption, with or without vesicles, and pain. HSV esophagitis is seen in immunocompromised HIV-infected patients and presents as dysphagia and odynophagia with or without oral lesions. Idiopathic aphthous ulcerations in HIV-infected patients are indistinguishable from HSV lesions. Perirectal lesions are often erythematous, ulcerative, and extremely tender, with a predilection for the gluteal cleft. Perirectal HSV may also be associated with proctitis and anal fissures.

Ocular HSV may be demonstrated by observing dendritic lesions after fluorescein staining. HSV is associated with a syndrome of acute retinal necrosis characterized by pain, keratitis, and iritis that may lead to retinal detachment. The diagnosis is usually made clinically but can be confirmed by a Tzanck test, biopsy, or culture.

Management and Disposition

Multiple oral treatment regimens are available to treat HSV. Duration of treatment is usually increased for immunocompromised patients. Ocular HSV requires prompt ophthalmology consultation.

Pearls

1. Anticipate involvement of multiple sites in HIV patients; presentations of HSV may be atypical compared to HSV in immunocompetent individuals.
2. Suspect HSV in any HIV patient with a chronic painful ulcerative lesion.
3. Perirectal HSV outbreaks may be missed on a superficial history and physical examination. Ask if it is painful to wipe after stool and examine the perianal area. Do not assume hemorrhoids in HIV-infected patients at risk for HSV.

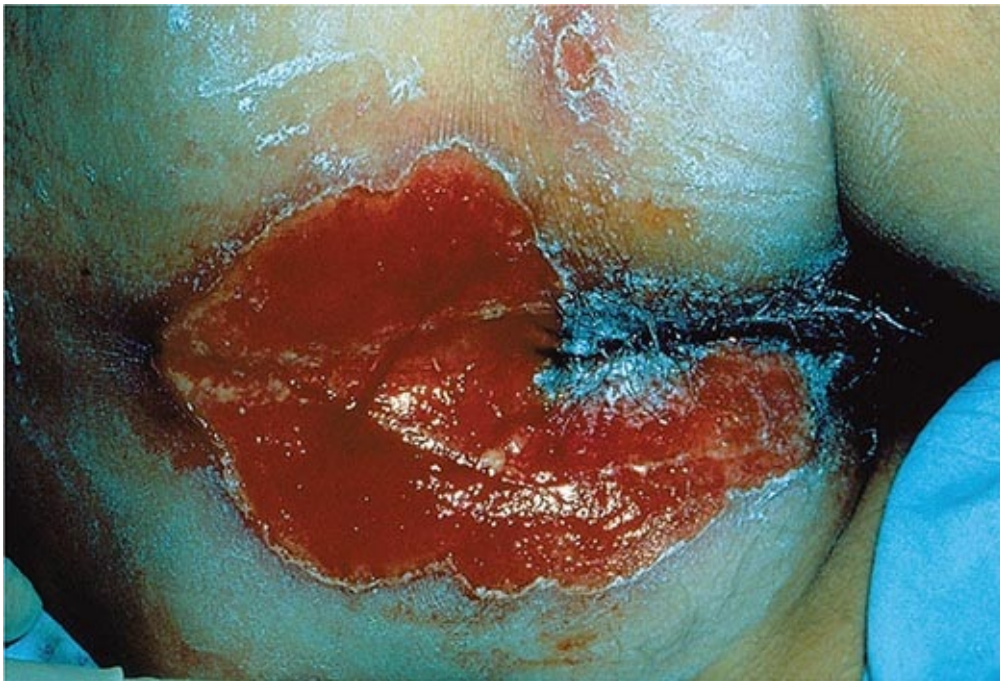


FIGURE 20.32 ■ Herpes Simplex Virus in an HIV Patient. Severe, recurrent perirectal HSV lesions in an HIV patient. (Photo contributor: Briana Hill, MD.)



FIGURE 20.33 ■ Severe HSV on Genitalia. Severe ulcerative HSV lesions on the penis and scrotum of an HIV patient. (Photo contributor: John O'Mara, MD.)

SCABIES

Clinical Summary

Human scabies, caused by the *Sarcoptes scabiei* mite, is one of the most common contagious dermatoses. In HIV patients, this organism can cause “crusted scabies,” also known as Norwegian scabies, which denotes an overwhelming scabies infestation. In typical scabies, the mites cause extremely pruritic burrows, vesicles, and papules in a characteristic distribution involving the finger webs, sides of the hands and feet, breasts, waist, and groin. Pruritus is most intense at night. In contrast, crusted scabies typically affects the hands and the feet with asymptomatic crusting and does not cause significant pruritus. Transmission through infected linens or clothing is common in cases of Norwegian scabies. Risk factors include poor hygiene, crowding, and exposure to pets.

Most often the diagnosis of scabies is made clinically, with evidence of burrows and severe pruritus in a characteristic distribution. Definitive diagnosis

is made from examination of shavings from the lesions. Placing mineral oil over a suspected lesion and then shaving it with a number 15 blade can demonstrate the mites, which are usually 0.3 to 0.4 mm in length.

Management and Disposition

Topical permethrin 5% cream has a low toxicity and is the treatment of choice for scabies. Patients are instructed to apply the cream from the neck down and leave it on for 8 to 14 hours before removal. One application is usually sufficient for treatment in nonimmunocompromised patients, but a 2nd application may be needed for severe disease. The patient's clothes and bedding should be washed in hot water. Antihistamines should be prescribed to alleviate the pruritus. Often the pruritus persists after treatment as a hypersensitivity reaction to the mites and their feces and eggs. Any complicating secondary skin infection should be treated with appropriate systemic antibiotics. Localized infections can be treated with topical steroids.

For HIV patients with Norwegian scabies, permethrin should be applied to the face, scalp, behind the ears, and from the neck downward. Repeat treatments may be needed. For severe or refractory cases, oral ivermectin (200 mg/kg for one dose) can be tried with the exception of pregnant or lactating women.



FIGURE 20.34 ■ Scabies. Typical scabies rash showing unroofed papules secondary to scratching. Several small burrows are also seen. (Photo contributor: George W. Turiansky, MD.)



FIGURE 20.35 ■ Norwegian Scabies. Overwhelming scabies infestation causing “crusted scabies” in this patient with HIV. Hyperinfestations involve thousands to millions of mites. (Photo contributor: Francisco Bravo Puccio, MD, with permission from The Gorgas Course in Clinical Tropical Medicine, Universidad Peruana Cayetano Heredia, Lima, Peru.)

Pearls

1. Often secondary staphylococcal infections complicate the diagnosis, including impetigo, ecthyma, paronychia, and furunculosis.
2. Oral antibiotics are often indicated for Norwegian scabies because of skin breakdown and secondary infections.
3. Close contacts of infected patients should be treated simultaneously. Inform patients that although the scabicide will kill the mites, itching may last for weeks. Patients often seek repeat treatments and inappropriately receive additional scabicides, which can cause contact dermatitis.



FIGURE 20.36 ■ Norwegian Scabies. Crusted lesions with hyper-keratotic scales on this patient's foot. Millions of mites were seen under microscopy in scrapings of the lesions. (Photo contributor: Larry Mellick, MD.)

MOLLUSCUM CONTAGIOSUM

Clinical Summary

Molluscum contagiosum is caused by a poxvirus that presents flesh-colored, dome-shaped lesions on affected individuals. Usually, molluscum contagiosum is an asymptomatic benign disease but may become severe in HIV patients. Typical presentations consist of groups of 2 to 20 small, discrete, shiny lesions with central umbilication.

Management and Disposition

If the diagnosis is suspected in the ED, the patient should be reassured and referred to a dermatologist. Usually self-limited with spontaneous remission, treatment outside of genital lesions is for cosmetic reasons only.

Pearl

1. Clinically, it may be difficult to distinguish between cutaneous *Cryptococcus* and molluscum contagiosum. Dermatology or infectious disease consultation and biopsy may be required if the patient is clinically ill. With ART and immune reconstitution, these lesions generally resolve.



FIGURE 20.37 ■ Molluscum Contagiosum. Facial rash with ocular involvement is a common site of infection in HIV patients. Note the central umbilication. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

DERMATOPHYTE (TINEA) INFECTIONS

Clinical Summary

Dermatophyte infections are common superficial fungal infections involving the scalp (tinea capitis), body (tinea corporis), feet (tinea pedis), crural fold (tinea cruris), and nails (onychomycosis). Infection is very common in healthy individuals, but can be varied and atypical in patients with HIV. HIV/AIDS patients also can demonstrate a much more severe and extensive form of disease. Three different dermatophytes are the usual source of infection: epidermophyton, trichophyton, and microsporum.

Tinea capitis is seen as an enlarging scaling patch on the scalp that may progress to a kerion. The initial lesion can be overlooked until alopecia is present. Untreated permanent scarring can occur. Tinea pedis is the most common dermatophyte infection seen in practice. Usually self-limited, intermittent, recurrent, and intensely pruritic, these lesions can often be secondarily infected due to mechanical irritation. Tinea corporis is the most common fungal infection seen in HIV patients. Usually, it begins as an intensely pruritic, oval scaling erythematous lesion that spreads centrifugally and can merge with other lesions. Tinea cruris is an infection involving the crural fold, much more common in men than women. It usually begins as a macular patch on the inner thigh, opposite the scrotal sac.



FIGURE 20.38 ■ Tinea Infection. Tinea infection involving the scalp, neck, and upper back of this HIV-infected patient. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

Tinea capitis responds to griseofulvin and other oral agents such as terbinafine, itraconazole, or fluconazole. Treatment usually is carried out for at least 3 weeks, with topical treatments ineffective. Tinea pedis can usually be treated with a topical antifungal cream for 4 weeks. Often patients have tried outpatient, over-the-counter antifungal medications without success. Tinea corporis often responds to daily application of topical antifungal medications; however, the

systemic oral agents described above are also effective. Tinea cruris usually responds to topical antifungal medications.

Pearls

1. Confirming the diagnosis with a KOH preparation is important before initiating therapy, especially in diffuse disease. Often HIV patients suffer from a variety of skin infections that mimic dermatophyte lesions (eczema).
2. Athletes are especially at risk when close skin-to-skin contact occurs (wrestling, football). Oral treatment in these cases is preferred.
3. Some dermatophyte infections fluoresce under the Wood lamp examination.



FIGURE 20.39 ■ Tinea Corporis. Tinea infection involving the lower back and buttocks of the patient in Fig. 20.38. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Thrombocytopenia occurs in 40% to 70% of all HIV patients. It can occur independently at all stages of HIV infection and may be encountered as the initial presentation of disease. HIV-associated anemia and granulocytopenia can occur concomitantly as the course of the HIV infection worsens, with thrombocytopenia seen in 30% of patients with CD4 counts less than $200/\text{mm}^3$. HIV patients with thrombocytopenia often present to the ED with bleeding (especially from the oral mucosa), ecchymosis, and petechiae. Secondary causes of thrombocytopenia are generally seen as the result of opportunistic infections, malignancy, or medications.



FIGURE 20.40 ■ Thrombocytopenia. Ecchymosis in an HIV patient with thrombocytopenia. (Photo contributor: Edward C. Oldfield III, MD.)

Management and Disposition

The emergency physician's efforts are initially focused on stabilization of the patient with two large intravenous lines, type and cross-match, and crystalloid infusion if significant bleeding has occurred. Because of the complexity of the differential diagnosis and potentially complicated treatment of HIV thrombocytopenia, an infectious disease specialist should be consulted early. In most cases, HIV patients with platelet count greater than 50,000 can be managed conservatively with spontaneous remission of approximately 20%. Zidovudine can increase platelet counts up to twofold in over 50% of patients. If the platelet count is less than 20,000, many infectious disease specialists recommend γ -globulin infusion and parenteral steroids. Other possible treatments include dapsone, danazol, interferon- α , vincristine, anti-D immunoglobulin, splenic irradiation, and splenectomy. Even if the patient is to be managed conservatively, bone marrow analysis should be performed to rule out other causes of thrombocytopenia.

Pearls

1. Perform thorough skin and oral examinations in all patients with HIV looking for manifestations of thrombocytopenia.
2. Take a careful drug history and consider other infectious etiologies before assuming the thrombocytopenia is directly secondary to HIV infection.
3. Spontaneous bleeding is rare unless the platelet count is less than 10,000.



FIGURE 20.41 ■ HIV-Associated Thrombocytopenia. A large spontaneous subconjunctival hemorrhage is noted in this patient with HIV and severe thrombocytopenia. (Photo contributor: John O'Mara, MD.)

DRUG REACTIONS

Clinical Summary

HIV patients have a 5 to 20 times higher rate of drug reactions than non-HIV patients. Up to 5% of ED visits by HIV-infected patients are due to complications of therapy. Many of these manifest dermatologically, in order of decreasing frequency: (1) exanthems, (2) urticaria/angioedema, (3) fixed drug reactions, (4) erythema multiforme, and (5) photosensitivity reactions. The most common medications associated with rashes are antivirals, antibiotics, and antifungals.



FIGURE 20.42 ■ Drug Reaction. Exanthematous drug reaction in an HIV patient. (Photo contributor: Kenneth Skahan, MD.)



FIGURE 20.43 ■ Efavirenz Rash. Drug exanthem typical of efavirenz. (Photo contributor: Stephen Burdette, MD.)

Management and Disposition

The emergency physician may need to consult an infectious disease specialist, a pharmacist, or a dermatologist to help clarify the existence of a drug reaction. Clues besides recent initiation of a new drug are eosinophilia or elevated liver function tests. Individual treatment varies depending on the situation. The offending agent should be discontinued. Antihistamines and steroids are indicated in certain situations.

Pearls

1. Ask about alternative and nonprescription medicines.
2. Consult a pharmacist or infectious disease specialist if there are concerns for drug reactions related to ART.
3. Beware of serious drug reactions such as Stevens-Johnson syndrome and toxic epidermal necrolysis.



FIGURE 20.44 ■ Efavirenz Rash. Close-up image of drug exanthem caused by efavirenz in an HIV patient. (Photo contributor: Stephen Burdette, MD.)



FIGURE 20.45 ■ Nevirapine Rash. Drug exanthema caused by nevirapine in this HIV patient. (Photo contributor: Seth W. Wright, MD.)

ACUTE NECROTIZING ULCERATIVE GINGIVITIS

Clinical Summary

Acute necrotizing ulcerative gingivitis (ANUG), also known as Vincent angina or trench mouth, is commonly seen in HIV-infected patients. It is a distinct and rapidly progressive ulceration typically starting at the tip of the interdental papilla, spreading along the gingival margins, eventually destroying the periodontal tissue. The triad associated with ANUG is oral pain, halitosis, and ulcerations. Other signs and symptoms include “metallic taste,” “wooden teeth” sensation, tooth mobility, fever, adenopathy, and malnutrition. The cause of this aggressive, destructive process in HIV patients is a polymicrobial infection by oral anaerobes (*Treponema*, *Selenomonas*, *Fusobacterium*, *Porphyromonas*, *Prevotella*). ANUG represents a spectrum of disease from mild ulcerations to severe cellulitis and spread of the infection to the soft tissues, cheeks, lips, and bones.

Management and Disposition

ANUG is most frequently seen in four population groups: (1) HIV patients, (2) malnourished children, (3) young adults who are under a great deal of stress, and (4) polysubstance abusers. The first steps for the emergency physician are to eliminate other potentially more serious life-threatening infections and to address hydration status. Treatment includes (1) eliminating contributing factors (stress, poor nutrition, poor sleep, alcohol, and tobacco use), (2) chlorhexidine rinses twice a day, (3) surgical debridement by an oral surgeon if needed, and (4) oral penicillin and metronidazole.

Pearls

1. Beware of Ludwig angina (brawny submandibular induration and tongue elevation) associated with severe progression of ANUG as acute airway compromise is possible.
2. Noma (cancrum oris, gangrenous stomatitis), a rare disease of childhood associated with malnutrition, is characterized by an anaerobic destructive infectious process of the orofacial tissues that can clinically resemble ANUG.



FIGURE 20.46 ■ Acute Necrotizing Ulcerative Gingivitis (ANUG). ANUG (Vincent angina or “trench mouth”) caused by spirochetal and fusiform bacteria in an HIV patient. Note the punched-out ulcerations of

the interdental papillae, which are pathognomonic. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

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Chapter 21

TROPICAL MEDICINE

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Rural Health Clinic in Uganda. (Photo contributor: Seth W. Wright, MD.)

FREE-LIVING AMEBA INFECTION

Clinical Summary

Free-living amebas, usually harmless protozoan residents of soil and water, can cause three distinct, occasionally devastating, human illnesses. Primary amebic meningoencephalitis (PAM) is a disease of the previously healthy and is caused by *Naegleria fowleri*. Granulomatous amebic encephalitis (GAE) is caused by *Acanthamoeba* species or *Balamuthia mandrillaris*, and occurs in both healthy and immunocompromised persons. In wealthier countries, contact lens users may suffer from chronic amebic keratitis, also caused by *Acanthamoeba*. While these diseases are found worldwide, they are more common in tropical and subtropical regions.

PAM is devastating, usually fatal, and found mostly in children or young adults with a history of recent freshwater exposure. The organism enters through the nose and penetrates the cribriform plate to the subarachnoid space and brain. The acute illness is indistinguishable from bacterial meningitis. Patients with GAE often present with an initial focus of infection in the skin or respiratory tract followed by neurologic changes reflective of extensive brain involvement.

Management and Disposition

The mainstay of management is consideration of these uncommon diseases. PAM is almost always fatal, but one survivor was successfully treated with amphotericin B, miconazole, and rifampin. Isolated cutaneous disease from *Acanthamoeba* and *B mandrillaris* can be cured, but brain involvement is fatal and often diagnosed at autopsy. Patients with suspected amebic keratitis should have immediate ophthalmologic referral.

Pearls

1. Lack of response to usual antimicrobials in a patient with severe meningitis symptoms should lead to the suspicion of PAM, particularly with recent freshwater exposure.
2. Global warming might increase the rate of *N fowleri* infection as the organism thrives in freshwater over 30°C.
3. Fatal cases of PAM have been reported following tap water nasal irrigation using a “neti pot” (a device used to clean nasal passages). Use of sterile or

previously boiled water when irrigating eliminates the risk.

4. Consider *Acanthamoeba* infection in all contact lens wearers with a corneal infection. Early amebic keratitis can mimic the dendritic pattern of herpes simplex infection.
5. Immunocompromised patients with space-occupying lesions should have GAE on the differential.



FIGURE 21.1 ■ Ameba. Twenty-one-year-old patient from South America with 1 year of symptoms from *Balamuthia mandrillaris*. The primary site often involves the mid-face and oral cavity. This patient did not have intracranial involvement. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

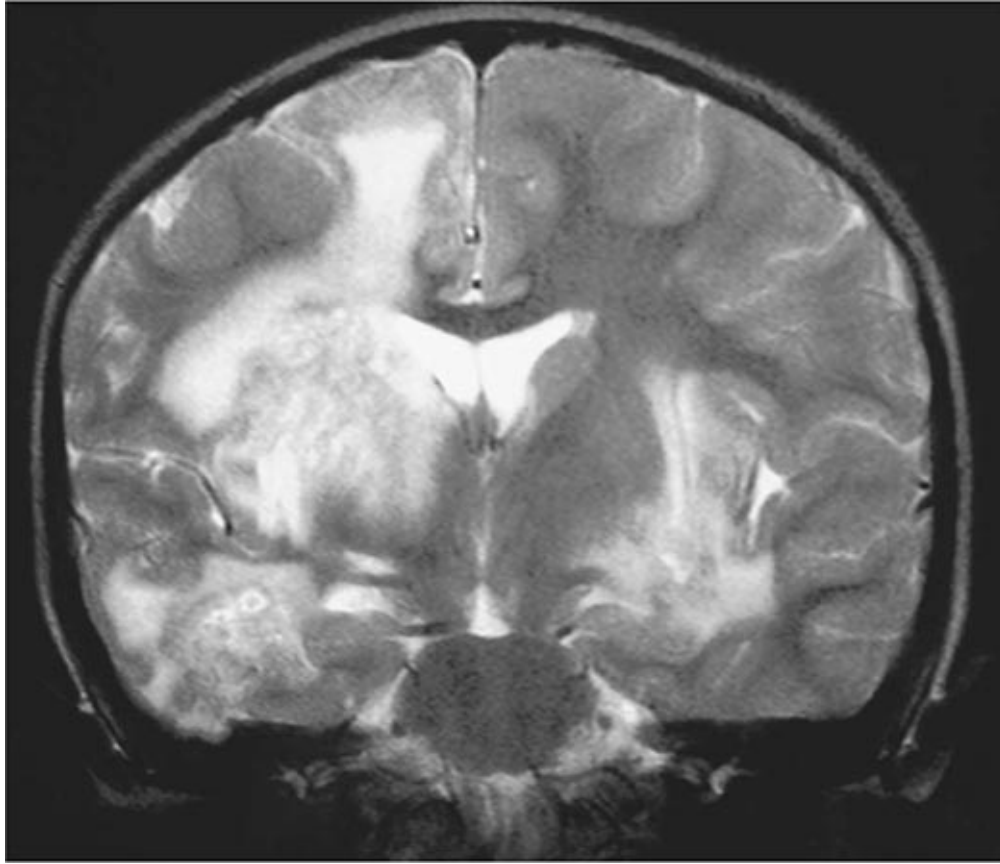


FIGURE 21.2 ■ Ameba Imaging. MRI showing extensive cerebral involvement in a fatal case of *Balamuthia mandrillaris*. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

ANEMIA IN THE TROPICS

Clinical Summary

Anemia in the tropics is a common medical condition resulting from various nutritional deficiencies, infections, parasites, and inherited disorders. Its prevalence and causes differ significantly from developed countries and are often multifactorial.

Overall, iron deficiency is the most common cause of anemia worldwide. Similar to deficiencies of folic acid and vitamin B₁₂, it results from diets rich in carbohydrates, but poor in meats, proteins, and vegetables. Most patients with AIDS in developing countries are anemic, with more severe cases seen in those coinfecting with tuberculosis. Malaria causes hemolysis and hypersplenism and

should always be considered in anemic patients. Hookworm infection is another common cause of anemia, particularly in children and pregnant women. Other parasitic illnesses causing anemia include visceral leishmaniasis (kala-azar) and African trypanosomiasis (sleeping sickness). Hemoglobinopathies, including sickle cell anemia and thalassemia, are common in Africa, the Middle East, the Americas, and South Asia. Severe hemolytic anemia can occur in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency.

Management and Disposition

Treatment depends on the suspected cause(s). Iron and folate supplementation can lead to a rapid and dramatic increase in red cell counts in patients with nutritional deficiencies. A low threshold for malaria diagnosis and treatment is important. Empiric treatment for hookworm is advisable. Red blood cell transfusion is rarely needed in stable patients, even those with extremely low counts.

Pearls

1. Administration of the antimalarial agent primaquine to patients with G6PD deficiency can lead to fatal hemolysis.
2. Thrombocytopenia in conjunction with anemia is highly suggestive of malaria in endemic areas.
3. Anemia in pregnancy should be screened for and aggressively treated as this improves both maternal and fetal outcomes.



FIGURE 21.3 ■ Anemia. Conjunctival pallor in a Haitian woman with complaints of weakness and a hemoglobin (Hgb) of 2.2. (Photo contributor: Andreas Fischer, RN.)

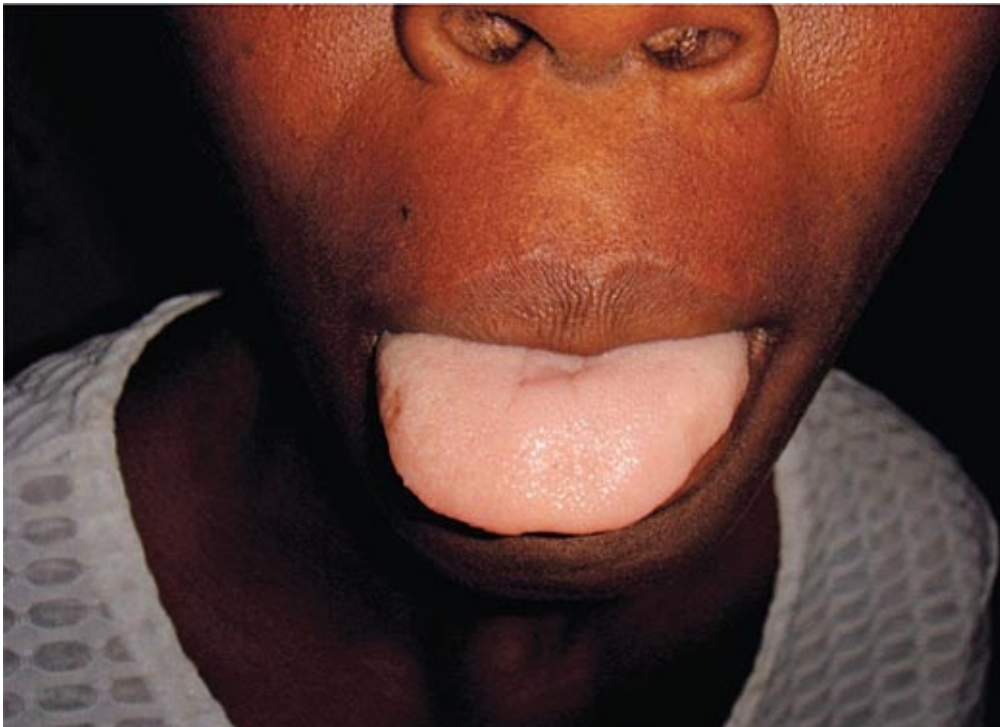


FIGURE 21.4 ■ Anemia. Pale tongue in a severely anemic Haitian woman. Pale mucous membranes, palms, and soles are often good indicators of anemia, particularly in dark-skinned individuals. (Photo

contributor: Andreas Fischer, RN.)



FIGURE 21.5 ■ Anemia in AIDS. Gums of Ugandan patient with end-stage AIDS and Hgb of 3.0. (Photo contributor: Seth W. Wright, MD.)

ANTHRAX

Clinical Summary

Anthrax, a worldwide zoonotic infection caused by *Bacillus anthracis*, was the 1st definitively recognized bacterial pathogen in human history. Most cases are sporadic, although occasional large outbreaks have been reported in Africa. Control of the disease in livestock has made endemic anthrax uncommon in developed countries, but it has taken on increased importance due to its use as a bioterrorism agent.

Cutaneous anthrax is the most common form, accounting for 95%. Infection occurs after spore exposure, with the initial manifestation of a small, painless, pruritic papule. The papule develops a large, central vesicle, followed by the classic necrotic ulcer with a black, depressed eschar. Local edema, regional lymphadenopathy, and systemic symptoms can occur. About 20% will progress

to systemic bacteremia. *Inhalational* anthrax occurs after aerosolized spore exposure and typically follows a biphasic course: (1) nonspecific upper respiratory infection– like symptoms and (2) then, in 1 to 4 days, fulminant bacteremia with fever, respiratory distress, hemorrhagic mediastinitis, and shock. *Gastrointestinal* (GI) anthrax is rare, affects the oropharynx or alimentary tract, and occurs after contaminated meat ingestion. It causes a nonspecific gastroenteritis and necrotic GI tract ulcers and may progress to shock or death 2 to 5 days after symptom onset.



FIGURE 21.6 ■ Anthrax. A black eschar with a central hemorrhagic ulceration on the thumb associated with massive edema on the hand. (Used with permission from Wolff K, Johnson RA, Suurmond D. *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*. 5th ed. New York, NY: McGraw Hill; 2005:631.)

Management and Disposition

Uncomplicated endemic cutaneous anthrax is typically treated with oral or intramuscular penicillin. Doxycycline, erythromycin, and ciprofloxacin are alternatives. High-dose intravenous (IV) penicillin is used in cutaneous disease with systemic findings and for inhalational or GI disease.

Patients with suspected bioterrorism-related anthrax require hospital admission with immediate public health notification. They should be decontaminated with removal (and sealed storage) of all clothing and cleansing with soap and water. The Centers for Disease Control and Prevention (CDC) guidelines recommend ciprofloxacin or doxycycline as first-line therapy.

Inhalational and other severe forms are treated with additional multidrug antimicrobial therapy. Glucocorticoid therapy is controversial but might be beneficial in meningitis or cutaneous disease with extensive head and neck edema.

Pearls

1. All forms can progress to hemorrhagic meningitis, which can mimic a traumatic tap on lumbar puncture.
2. Cutaneous and inhalational anthrax have been reported from use of imported animal hide drums.
3. Injection anthrax is rare and recently found among heroin users in Great Britain and Germany. Symptoms include abscess, cellulitis, necrotizing fasciitis, and sepsis.
4. The length of antibiotic therapy is usually 7 to 10 days, but postexposure prophylaxis involves 60 days, including vaccine administration.
5. The classic pathologic finding of inhalational anthrax is hemorrhagic mediastinitis, demonstrated as a widened mediastinum on chest radiography.



FIGURE 21.7 ■ Anthrax Progression. (A) Initial facial cutaneous anthrax with early lesion development and massive facial edema. (B) Interval development of septicemia. The lesion is developing a more typical dark coloration. (C) The patient following resolution of sepsis. (Photo contributor: Seth W. Wright, MD.)

ASCARIASIS

Clinical Summary

Ascaris lumbricoides is the most common human intestinal roundworm infection. The parasites are found worldwide and are highly endemic where sanitation and hygiene are poor; they commonly cause infection in regions where human feces are used as fertilizer. Adult worms live in the small intestine and produce enormous numbers of eggs, which are excreted in the feces. Passed eggs require at least several weeks in warm, moist soil before embryonating into an infective egg. Ingested eggs hatch in the jejunum, migrate through the intestinal wall into the bloodstream, and are transported to the lungs. Larval worms burrow through the alveolar walls, ascend through the trachea, and are swallowed back into the small intestine where they develop into adults.

Most infections are asymptomatic. Patients may present for care if they pass a worm in their stool. A heavy worm burden may lead to abdominal pain, pancreatic/biliary disease, or intestinal obstruction. Failure to thrive and decreased cognitive development are seen in heavily infected children. Migrating worms may cause biliary obstruction, appendicitis, or liver abscesses.

Diagnosis is from identification of a passed worm or examination of stool for eggs.

Management and Disposition

Outpatient treatment with mebendazole or albendazole is usually effective but only on worms in the adult intestinal stage. Thus, it is recommended that a stool examination be done at 2 to 3 months and retreatment initiated if positive for eggs.

Pearls

1. Marked peripheral eosinophilia can be seen during the migratory stage.
2. While in the lungs, migrating worms can cause an eosinophilic pneumonitis with asthma-like symptoms, or Löffler syndrome.
3. *Ascaris* worms occasionally migrate from the anus, mouth, or nose following antihelminthic treatment.
4. *Ascaris* is the most common cause of acute abdominal surgical emergencies in some highly endemic countries.



FIGURE 21.8 ■ Ascariasis. Worms spontaneously extruding from the anus of a boy with a high fever. Worms often migrate in patients with fever. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.9 ■ Ascariasis. Adult ascaris worm after passage from the anus. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.10 ■ Ascariasis. Distended abdomen of a Haitian girl with a large ascaris worm burden. (Photo contributor: Seth W. Wright, MD.)

CHAGAS DISEASE

Clinical Summary

American trypanosomiasis, or Chagas disease, is a zoonotic protozoal infection caused by *Trypanosoma cruzi*. Chagas is spread by triatomine insects with a large variety of mammals serving as natural reservoirs. Disease is limited to the Western hemisphere and is most common in areas of substandard housing as the vector insects proliferate in adobe, unfinished brick walls, and in thatched roofs. Chagas is considered the most important protozoal disease in the Americas, surpassing malaria in morbidity and mortality. It is estimated that 10 million people are infected, including 300,000 immigrants within the United States.

Chagas is usually acquired during childhood and persists for life. Infection occurs when the insect defecates while taking a blood meal and the infectious stool is rubbed into the bite wound or a mucous membrane (often the conjunctiva). Acutely, infection is often asymptomatic but can present with a skin lesion at the infection site (chagoma), fever, adenopathy, myocarditis, and hepatosplenomegaly. The organism can be isolated during the indeterminate phase (years or even lifespan). Chronically, about 30% develop a

cardiomyopathy with progressive biventricular heart failure. Severe arrhythmias, including complete heart block, are common. Approximately 10% of patients will develop digestive complications due to loss of lumen wall neurons, including megaesophagus and megacolon.

During acute infection, trypanosomes are seen on the blood smear and polymerase chain reaction (PCR) is useful for diagnosis, but serology is typically negative. Serologic assays are used to diagnose chronic disease. Blood culture and xenodiagnosis can also be used but lack sensitivity.

Management and Disposition

Patients presenting with acute symptoms are treated with benz-nidazole or nifurtimox. Patients with chronic disease should be worked up with an electrocardiogram (ECG) and echocardiography and referred as indicated. Antitrypanosomal treatment is warranted in the indeterminate phase of chronic infection and in those with early-stage cardiomyopathies.

Pearls

1. Chagas is the most common cause of cardiomyopathy in many Latin American countries. The disease should also be considered in patients with complete heart block or other arrhythmias.
2. Chagas is surprisingly common in wild and domestic mammals in the Southern and Southwest United States. The prevalence in Tennessee dogs is 6.4% and is higher in wild animals such as raccoons, skunks, and rodents.
3. Human infection in the United States is rare since the vector insects are uncommon inside modern housing, but occasional cases have been reported in those without a history of travel.
4. Universal screening of blood for Chagas is now routine in the United States.



FIGURE 21.11 ■ Chagas Disease. A child with unilateral periorbital swelling from local inflammation at the site of a Chagas organism inoculation (Romana's sign). (Photo contributors: Jorge Kleisinger, MD, and Ana Rosa de Benedetto.)

CHOLERA

Clinical Summary

Cholera is a severe diarrheal disease caused by *Vibrio cholerae*, a gram-negative bacterium, and is spread via the fecal-oral route. It is associated with poor hygiene and overcrowding, as well as the resultant contamination of food and water. Endemic disease is present in many areas of the world with occasional epidemics. Massive outbreaks in previously disease-free areas can occur following disasters or breakdowns in public health measures, such as seen in Haiti following the 2010 earthquake.

Cholera is characterized by massive, watery, gray, and painless diarrhea. The stool resembles “rice water” without blood or pus. Patients may have associated vomiting. Renal failure, hypotension, and circulatory collapse can occur within hours of diarrhea onset. Mortality rates as high as 20% to 50% among severe cases can be seen if adequate rehydration is not available. Death rates of less

than 2% are seen with good case management.



FIGURE 21.12 ■ Cholera. Severely dehydrated child during an outbreak in Uganda. Lethargy and sunken eyes are typical findings. IV fluids are typically reserved for severe cases. The child is on a typical cholera cot. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

The mainstay of treatment is hydration. Oral rehydration solutions are adequate for mild and moderate dehydration. Patients with severe dehydration are treated with IV lactated Ringer solution and oral rehydration. Per World Health Organization (WHO) guidelines, adult patients may require 10 to 15 L in the first 24 hours. Monitoring of electrolytes and renal function is ideal, but often not available in areas struck by cholera epidemics. Hypokalemia is common. Antibiotics are not required for recovery as the illness is self-limiting, but doxycycline may reduce the volume of diarrhea and shorten the duration of illness.

Pearls

1. Cholera, plague, and yellow fever are the three diseases internationally notifiable to WHO.
2. John Snow's removal of the Broad Street water pump handle during the 1854 cholera epidemic in London is considered to be the beginning of modern field epidemiology.
3. Rapid cholera test kits are available and are essential for early confirmation of disease in the 1st suspected cases.
4. An oral cholera vaccine is available but only confers short-term immunity and is not available in the United States.



FIGURE 21.13 ■ Dehydration in Cholera. “Tenting” of the skin of a dehydrated cholera patient. The presence of tenting implies moderate to severe dehydration. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.14 ■ Rice Water Stool. Typical “rice water” stool from cholera patient. Patients with cholera often lose a liter or more of watery stool an hour. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.15 ■ Cholera Cot. Patient on cholera cot. Use of the cholera cot allows for monitoring of fluid output and allows for easier waste control in patients too weak to leave the bed. (Photo contributor: Seth W. Wright, MD.)

CUTANEOUS LARVA MIGRANS

Clinical Summary

Cutaneous larva migrans (CLM), also known as “creeping eruption,” is the most common dermatologic problem following a trip to the tropics. CLM is a worldwide, parasitic infection commonly seen in warm, tropical environments; it is uncommon in developed countries due to shoe-wearing habits and routine deworming of pets.

CLM is most caused by dog and cat hookworms, *Ancylostoma caninum* and *Ancylostoma braziliense*; humans are an accidental host. The infected animal passes eggs in its feces where they hatch, molt, and feed on soil bacteria. Upon

contact with a human host, the larval worm penetrates the skin and attempts dermal migration. The worm remains under the skin since it lacks collagenase and cannot penetrate deeper layers. The larvae are unable to complete their life cycle, and, if left untreated, are trapped in the epidermis and will die in 2 to 8 weeks.

CLM is commonly seen on the lower extremities of travelers who walk barefoot on beaches with contaminated sandy soil. Symptoms are manifested by an erythematous tract with a distinctive serpiginous dermal pattern. The tract is markedly pruritic and may feel like a thread on palpation.

Management and Disposition

Treatment of CLM consists of mebendazole, albendazole, ivermectin, or topical application of thiabendazole. Antipruritics may also help.

Pearls

1. The lesions advance a few millimeters to several centimeters daily as the larva migrates.
2. Excoriation and secondary infections frequently occur. This can make the distinctive wandering rash more difficult to visualize.
3. Biopsy is usually not helpful for diagnosis since the organism lies 1 to 2 cm away from the leading edge of the eruption.



FIGURE 21.16 ■ Cutaneous Larva Migrans. A serpiginous, linear, raised, tunnel-like erythematous lesion outlining the path of migration in the larva. Upon palpation, it feels like a thread within the superficial layers of the skin. (Photo contributor: Janet Rohde.)



FIGURE 21.17 ■ Cutaneous Larva Migrans. CLM with secondary infection on the foot of a toddler from Zambia. (Photo contributor: Meg Jack, MD.)

CYSTICERCOSIS

Clinical Summary

The larval form of the pork tapeworm *Taenia solium* causes cysticercosis and affects 50 million people worldwide. Disease in developed countries is usually due to immigration or foreign travel. Taeniasis (intestinal tapeworm) is acquired from ingesting cysts from undercooked pork. Eggs passed in the stool from these carriers are highly infectious and may survive in the environment for months. Humans acquire cysticercosis from the ingestion of these eggs/larva, usually through unhygienic food preparation. Once the eggs are consumed, they hatch, penetrate the bowel wall, and travel to the subcutaneous tissue, skeletal muscle, and brain, though they may involve any organ. Two distinct types of diseases exist: *neurocysticercosis* (which can be parenchymal or extraparenchymal) and *extraneural cysticercosis*. Symptoms are dependent on the affected organ system; significant morbidity is associated with ocular, cardiac, and neurologic involvement. The larval worm will eventually die and leave calcified lesions.

Intestinal taeniasis is diagnosed through egg identification on stool exam or by passage of an intact worm or worm segment. Computed tomography (CT) and magnetic resonance imaging (MRI) have facilitated recognition of neurocysticercosis with visualization of a contrast-enhancing ring lesion. Diagnosis may also be made by biopsy, serum, or cerebrospinal fluid (CSF) antibody testing. Neurocysticercosis should be strongly considered in endemic regions or in immigrants with new-onset seizures.

Management and Disposition

Seizures are treated with standard medications. Calcified lesions do not require specific anticysticercal therapy. Viable cysts can be treated with albendazole or praziquantel but should be done with expert consultation. Inpatient management is advisable if viable cysts are to be treated as inflammatory reactions may occur. Corticosteroids are recommended in this situation. Surgical intervention may be indicated for obstructing neurologic lesions or intraocular lesions.

Pearls

1. Infection is most common in rural areas of developing countries where pigs are allowed to roam freely and ingest infected human feces. This completes the cycle and propagates infection.

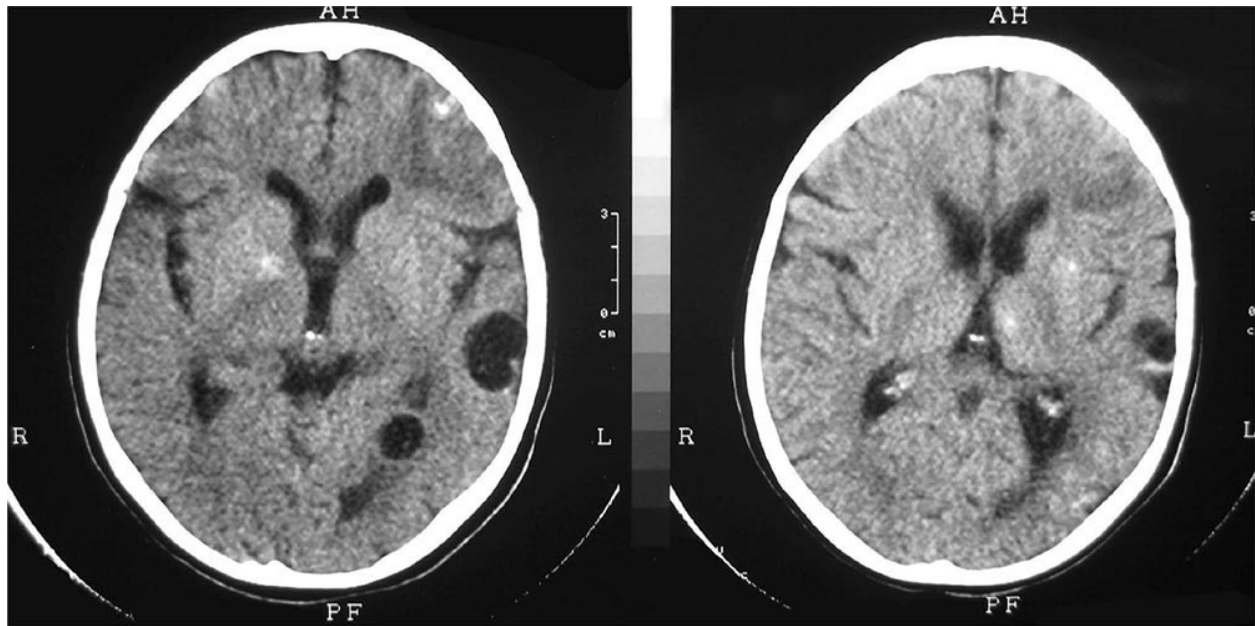


FIGURE 21.18 ■ Neurocysticercosis. Noncontrast head CT showing new (cystic) and old (calcified) lesions of neurocysticercosis. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

2. Cysticercosis and taeniasis are rare in predominantly Muslim countries where eating pork is uncommon; however, cysticercosis is still possible due to ingestion of contaminated nonpork food products.
3. The adult tapeworm can attain a length of 20 feet or more and can live up to 20 years in the intestine.
4. Most people with an intestinal pork tapeworm do not have cysticercosis.
5. The only pathognomonic CT or MRI finding of neurocysticercosis is a 2- to 4-mm bright nodule within the cyst (“cyst with dot sign”) that represents a solid larval tapeworm.

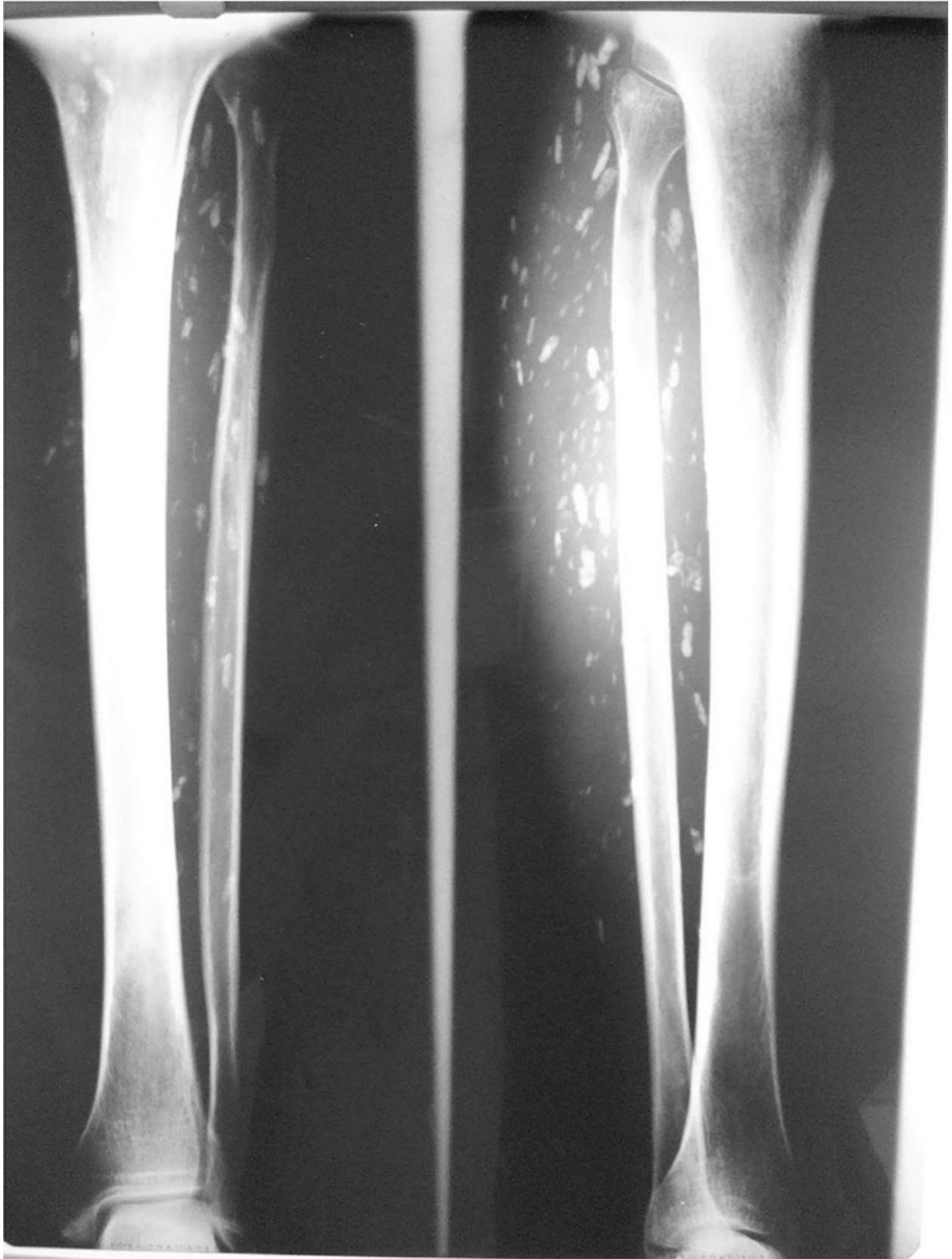


FIGURE 21.19 ■ **Cysticercosis Cysts.** Multiple calcified soft tissue cysts noted as an incidental finding on

a lower leg radiograph in a patient with disseminated cysticercosis. The “cigar-shaped” calcifications are highly suggestive of cysticercosis. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

DENGUE FEVER

Clinical Summary

Dengue fever, also known as “breakbone fever,” is a mosquito-borne viral disease. A flavivirus, dengue is a rapidly emerging illness due to reintroduction of *Aedes* species mosquitoes into areas of previous eradication. It is now distributed throughout tropical and subtropical regions, and widespread epidemics have occurred in the Caribbean and Southeast Asia, among other world regions. Dengue is one of the most common causes of fever in returning travelers.

The incubation period is usually 3 to 7 days, with symptom resolution within 10 days. Infection ranges from subclinical symptoms to a hemorrhagic state with possible shock. Most commonly, it is characterized by fever, severe myalgias, retro-orbital pain, and headache. A rash, seen in approximately half of patients, is usually maculopapular, but may be mottled, flushed, or petechial. The recently revised classification of dengue severity consists of dengue without warning signs, dengue with warning signs, and severe dengue based on the degree of plasma leakage, spontaneous bleeding, organ impairment, and shock.

There are four serotypes; exposure to one serotype provides lifelong immunity to that type. Previous infection with one serotype may predispose an individual to a more severe infection with another serotype. Thus, severe dengue is more common in indigenous individuals than the previously unexposed traveler. The diagnosis is primarily clinical. The presence of thrombocytopenia, leukopenia, and hemoconcentration is suggestive, and confirmatory tests with PCR or immunoglobulin M antibodies via enzyme-linked immunosorbent assay (ELISA) are available.

Management and Disposition

Supportive therapy is the only treatment available, with a focus on fluid repletion. Patients with mild illness are treated as out-patients. Admission of those with severe illness, hemorrhagic manifestations, shock, or an uncertain

diagnosis is warranted.

Pearls

1. Consider dengue fever in recently returned travelers with fever, headache, and myalgias, particularly travelers from the Caribbean or Southeast Asia.
2. The fever tends to be high and may suddenly resolve and then return, also known as a “saddle back” fever pattern.
3. A positive “tourniquet test” is suggestive of dengue. A blood pressure cuff is applied and inflated to a point between the systolic and diastolic pressures for 5 minutes. The test is positive if there are more than 20 petechiae per square inch.
4. Occasional cases of dengue occur in South Texas, and an outbreak recently occurred in Key West, Florida.



FIGURE 21.20 ■ Dengue Rash. Maculopapular rash from dengue on legs of an American medical worker during an outbreak in Guyana. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.21 ■ Dengue Petechiae. Measurement of petechiae following a tourniquet test in a patient with dengue. Twenty or more petechiae in the template area are suggestive of dengue diagnosis. (Photo contributor: WHO/TDR.)



FIGURE 21.22 ■ Dengue Rash. A hemorrhagic bullae on the arm of a patient with dengue hemorrhagic fever. (Photo contributor: WHO/TDR.)

DRACUNCULIASIS

Clinical Summary

Dracunculiasis is a debilitating roundworm infection caused by *Dracunculus medinensis*, commonly known as the Guinea worm. Dracunculiasis was historically widespread throughout equatorial Africa, the Middle East, and South Asia, but the disease is now isolated to limited areas of Africa due to intensive eradication efforts. Dracunculiasis has a complex life cycle beginning when infected water fleas, or cyclops, are ingested while drinking from unclean water sources. Following ingestion, the larval worm penetrates through the stomach and intestine and migrates to the subcutaneous tissue, where it matures and reproduces. A painful wound develops, usually on the lower extremity, when the

female emerges to release larvae. Immersion in water relieves the pain, allowing the worm to release larvae and complete the life cycle.

Emergence of the worm leads to a painful wound that is often accompanied by fever, nausea, vomiting, edema, intense pruritus, ulceration, and eosinophilia, all of which can last for months. The wounds often become secondarily infected, leading to significant disability. Drinking boiled or filtered water prevents dracunculiasis.

Management and Disposition

The worm is carefully extracted, usually around a stick or rolled gauze, in order to avoid retained worm products and the resultant inflammatory reaction. This process can take days to weeks. Daily immersion in water, with proper disposal to avoid larval spread, helps facilitate worm removal. Surgical excision is possible.

Pearls

1. The Guinea worm is the largest tissue parasite of humans. While very thin (2 mm in diameter), a full-grown worm is about a meter in length.
2. Dracunculiasis is rarely fatal, but the wound can cause significant morbidity and economic loss in areas with high infection rates.
3. It is predicted that dracunculiasis will be the 1st parasitic disease to be fully eradicated, largely through patient education and use of filtered water.

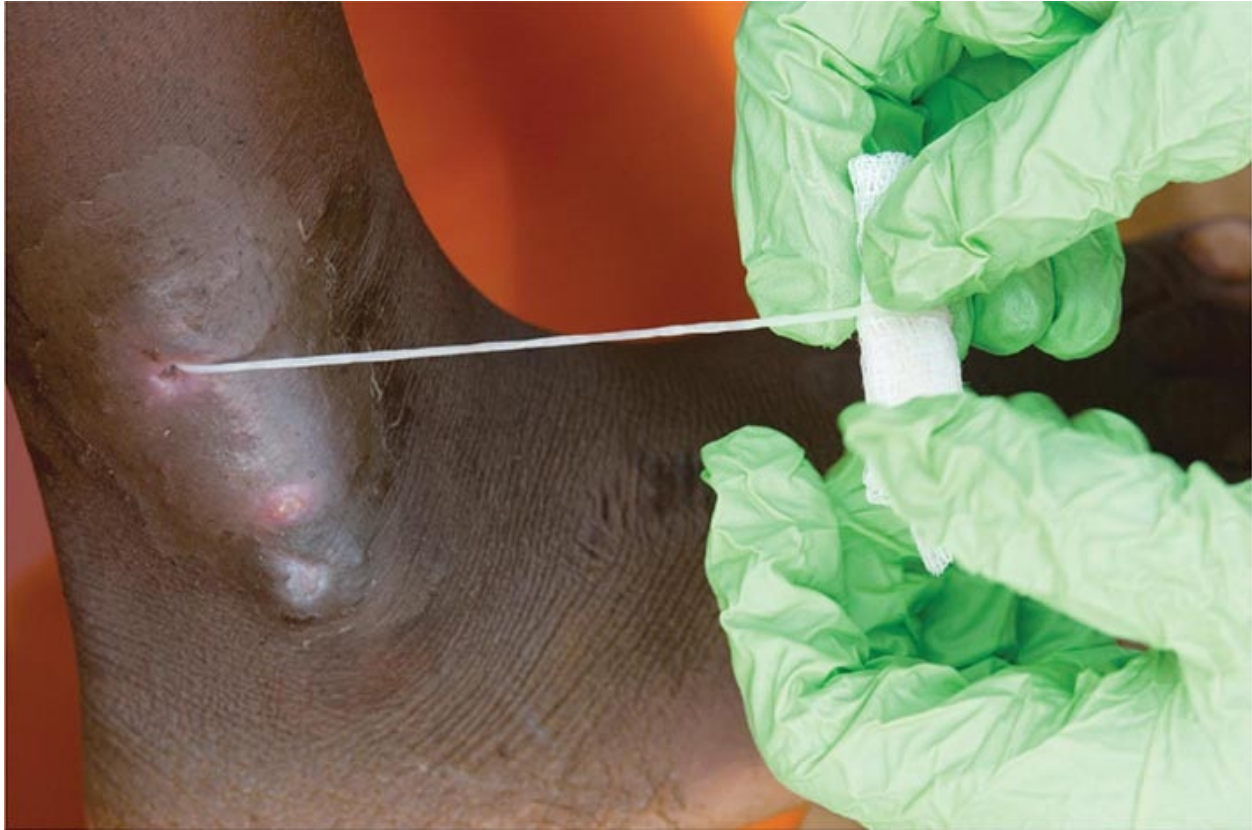


FIGURE 21.23 ■ Dracunculiasis. Gradual extraction of a Guinea worm using rolled gauze in Ghana. (Photo contributor: The Carter Center/Louise Gubb.)

ELEPHANTIASIS

Clinical Summary

Elephantiasis affects more than 120 million people worldwide with over 40 million severely disfigured. It is not a specific disease, but rather a syndrome caused by chronic obstruction of lymphatics. The most common cause is lymphatic filariasis, which is caused by the thread-like worms *Wuchereria bancrofti*, *Brugia malayi*, and *Brugia timori*. The infection is transmitted to humans by mosquitoes. Adult worms lodge in the lymphatics, disrupting the fluid balance between tissues and blood vessels, causing lymphedema of the extremities, breast, or genitourinary (GU) system. The infection is generally acquired in childhood, although clinical manifestations may take years to develop. Adult worms live 4 to 6 years, producing millions of blood circulating microfilariae. Acute symptoms of lymphadenopathy and dermal inflammation

may precede and later accompany chronic swelling. With persistent infection and inflammation, the skin develops a hyperkeratotic, pebbly, or warty appearance that may become ulcerated and darkened. Bacterial and fungal superinfections contribute to morbidity.

Mosquito nets and insect repellants are the main means of prevention of lymphatic filariasis. Parasites may be detected microscopically in the blood, but the nocturnal periodicity makes identification challenging. Eosinophilia is common but is nonspecific. Availability of a rapid card test that identifies circulating antigens has overcome this problem and is available in some endemic areas.

Management and Disposition

Treatment of lymphatic filariasis depends on the presence or absence of other filarial organisms and includes various combinations of albendazole, ivermectin, and diethylcarbamazine. Cleansing of the affected areas and topical antibiotics aid in thwarting secondary disease. Local massage and elevation of the extremity improve lymphatic flow.

Pearls

1. Elephantiasis bears a heavy social burden due to physical limitations, disfigurement, sexual disability, and social stigmatization. Affected patients may be shunned by their families, are often unwed, and are unable to work.
2. Testicular hydrocele is the most common manifestation of chronic *W bancrofti* infection for males in endemic areas.
3. Symptomatic filariasis is occasionally seen in travelers, even in those with only short-term stays in endemic regions.
4. *W bancrofti* occasionally causes an acute asthma-like condition known as tropical pulmonary eosinophilia.
5. Podoconiosis is a noninfectious cause of elephantiasis that occurs in people who walk barefoot in areas with large amounts of volcanic ash. Kaposi sarcoma is another emerging cause of nonfilarial elephantiasis in sub-Saharan Africa.



FIGURE 21.24 ■ Elephantiasis. Unilateral lymphatic filariasis. Note diffuse edema with early evidence of chronic skin changes. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 21.25 ■ Elephantiasis. Late-stage bilateral elephantiasis with chronic nodular skin changes. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.26 ■ Elephantiasis. Large hydrocele from lymphatic filariasis in a man from Haiti. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.27 ■ Elephantiasis. Nonfilarial elephantiasis due to extensive Kaposi sarcoma in a 26-year-old Ugandan woman with AIDS. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.28 ■ Elephantiasis. Bilateral disfigurement from lymphatic filariasis in an elderly Haitian man. (Photo contributor: WHO/TDR.)

EPIDEMIC MENINGITIS

Clinical Summary

Meningococcal disease in developed countries usually consists of sporadic cases

and small outbreaks. In contrast, massive epidemics of serogroup A or C meningococcal meningitis occur in tropical countries, most notably in sub-Saharan Africa. These seasonal outbreaks tend to occur during the dry season along a wide swath of equatorial Africa known as the “meningitis belt.”

The clinical presentation of epidemic meningitis is the same as in developed countries. Patients will typically present with fever, headache, nausea/vomiting, photophobia, and neck stiffness. Coma and death typically ensue if not treated. Diagnosis is with a spinal tap, although patients in resource-limited countries are often treated based on clinical grounds during a known outbreak. Mortality rates of less than 10% are obtainable. Large-scale vaccination programs are effective in decreasing spread of the disease within the affected areas and in adjacent population centers.

Management and Disposition

The mainstays of treatment are antibiotics and supportive care. Penicillins, cephalosporins, and ampicillin are still the treatments of choice in many settings and are usually efficacious.

Pearls

1. Single-dose oily chloramphenicol injections have been used with success and allowed for easier management of large numbers in limited-resource settings. A 2nd dose is given in 24 hours if there has been no improvement.
2. Massive outbreaks of meningitis have occurred during the Hajj, a pilgrimage to Mecca in Saudi Arabia. They are the only country requiring proof of vaccination before entry.
3. An often-used critical threshold to define an epidemic in Africa is 15 cases per 100,000 population per week. This would be equal to over 1400 cases per week in the Chicago metropolitan area.
4. Both serogroup A and C are covered by the meningococcal vaccine recommended for travelers by the WHO and CDC.

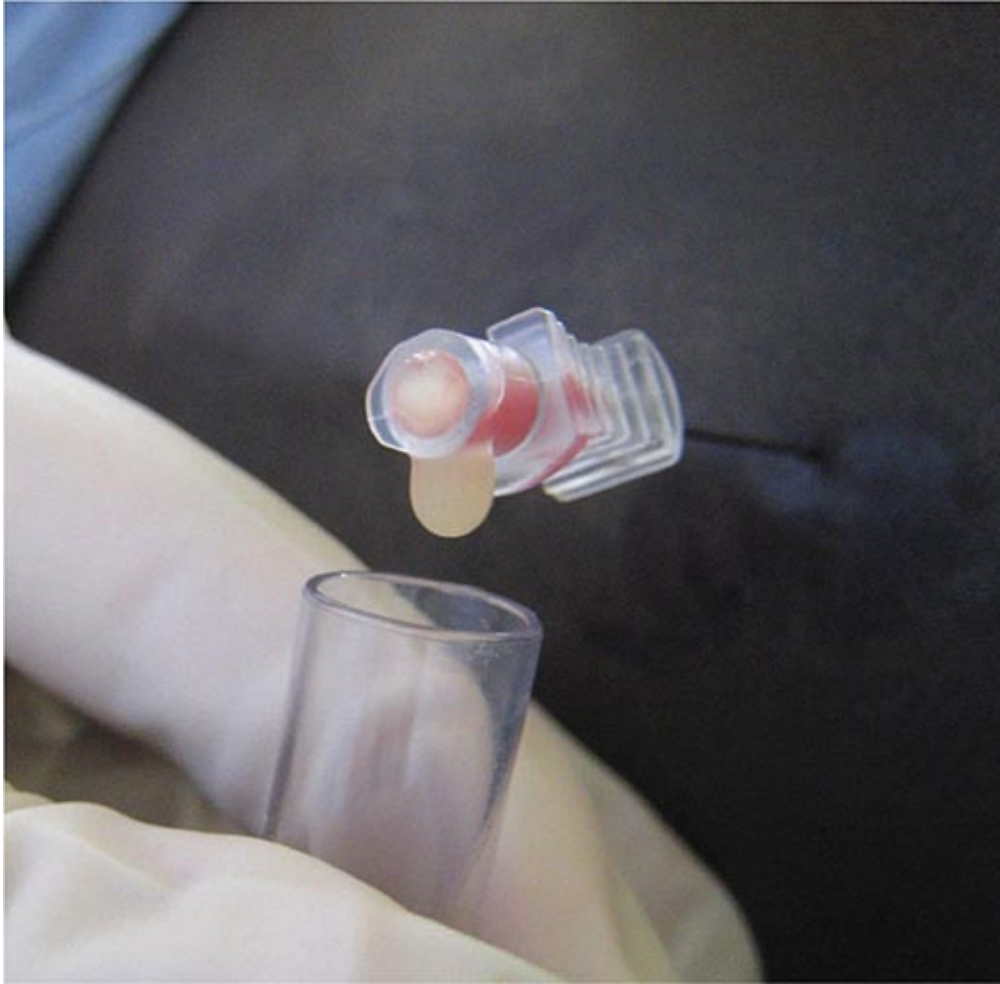


FIGURE 21.29 ■ Meningitis Spinal Fluid. CSF obtained from a 33-year-old Ugandan woman treated during an epidemic of sero-group A meningococcal meningitis. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.30 ■ Meningitis in a Child with a Very Rigid Neck. Cerebral malaria should also be in the initial differential diagnosis. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. *The Color Atlas of Family Medicine*. 2nd ed. New York, NY: McGraw Hill; 2013: Fig. 7-14.)

GOITER

Clinical Summary

Endemic goiter is one of a spectrum of iodine deficiency disorders and is characterized by thyroid gland enlargement. It can occur in any location where environmental iodine is limited but is rarely seen in developed countries as dietary iodine supplementation is routine. Goiters are often seen in inland and mountainous areas where iodine is leached from the soil and access to iodine-rich foods (ie, fish) is limited. Iodine deficiency disorders are common in widespread areas of Africa, Asia, and South America.

Patients with inadequate iodine in their diet have decreased T_3 and T_4 production and resultant increased pituitary thyroid-stimulating hormone (TSH) secretion. The thyroid becomes hyperplastic and enlarged; however, patients are typically euthyroid.

Management and Disposition

Prevention with dietary supplemental iodine is the mainstay of goiter management at the community level. Individual patients with goiter can be treated with potassium iodide solution or Lugol iodine, with iodized salt as a permanent solution. Surgical treatment can be indicated for massive goiter, particularly in the setting of tracheal or esophageal compression.

Pearls

1. Goiters in developed countries are most likely to be caused by defects in thyroid hormone production, resulting in an increase in TSH.
2. Iodine deficiency is the most common cause of preventable mental retardation (endemic cretinism) in the world.
3. Some dietary staple items, such as cabbage, cassava, lima beans, and sweet potatoes, have a goitrogenic factor, which may be superimposed upon primary iodine deficiency.
4. Malignancy is not a common complication despite the massive size of many goiters, although it may interfere with detection.
5. In children, the goiter is usually diffuse; in adults, however, it is more commonly nodular since individual thyroid nodules proliferate at different rates.



FIGURE 21.31 ■ **Goiter.** Easily visualized and palpable goiter. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.32 ■ Goiter. Patient from mountainous inland region of Haiti with massive goiter. This island nation has a high rate of goiter due to the complex interaction of poverty, political instability, deforestation, and erosion of soil. (Photo contributor: Ian D. Jones, MD.)

HYDATID CYST

Clinical Summary

Hydatid cysts are formed by the larval stage of *Echinococcus granulosus* tapeworms. The life cycle is maintained between canines and various farm animals (usually sheep or cattle). Adult worms inhabit the canine small intestine, and eggs are passed in the stool. Farm animals become infected when they feed upon stool-contaminated material. The life cycle is completed when dogs or other canines ingest larval cysts formed in infected farm animals. Humans are infected from inadvertent ingestion of canine fecal material and are considered accidental hosts.

Hydatid cysts most commonly affect the liver and the lungs. Most patients who are infected have no symptoms until the cysts rupture or are large enough to produce mass effect. Diagnosis is centered upon ultrasonography and serologic testing. Changes in farming practices have led to a marked decline in most industrialized countries, but they remain common in many developing areas. Most cases in the United States are immigrants from endemic regions.

Management and Disposition

Hydatid cysts are often an incidental finding when x-rays, CT scans, or ultrasounds are done for other purposes. These patients can be referred for follow-up. Patients with symptomatic cysts may need admission. Depending on cyst size and location, treatment may include percutaneous aspiration, surgical resection, or pharmacotherapy. Care must be taken with surgery to avoid spillage of cysts, which may lead to anaphylaxis.

Pearls

1. Liver cysts may produce obstructive jaundice, abdominal pain, or cholangitis.
2. Cysts are slow growing with an estimated average growth of 1 to 1.5 cm per year.
3. A chronic cough, pleuritic chest pain, hemoptysis, and dyspnea may be seen with lung involvement.
4. Although cerebral involvement is uncommon, it is seen more often in children.



FIGURE 21.33 ■ Hydatid Cyst Radiograph. Chest x-ray showing multiple hydatid cysts. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.34 ■ Hydatid Cyst CT. Scan demonstrating large hydatid cyst in the liver. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

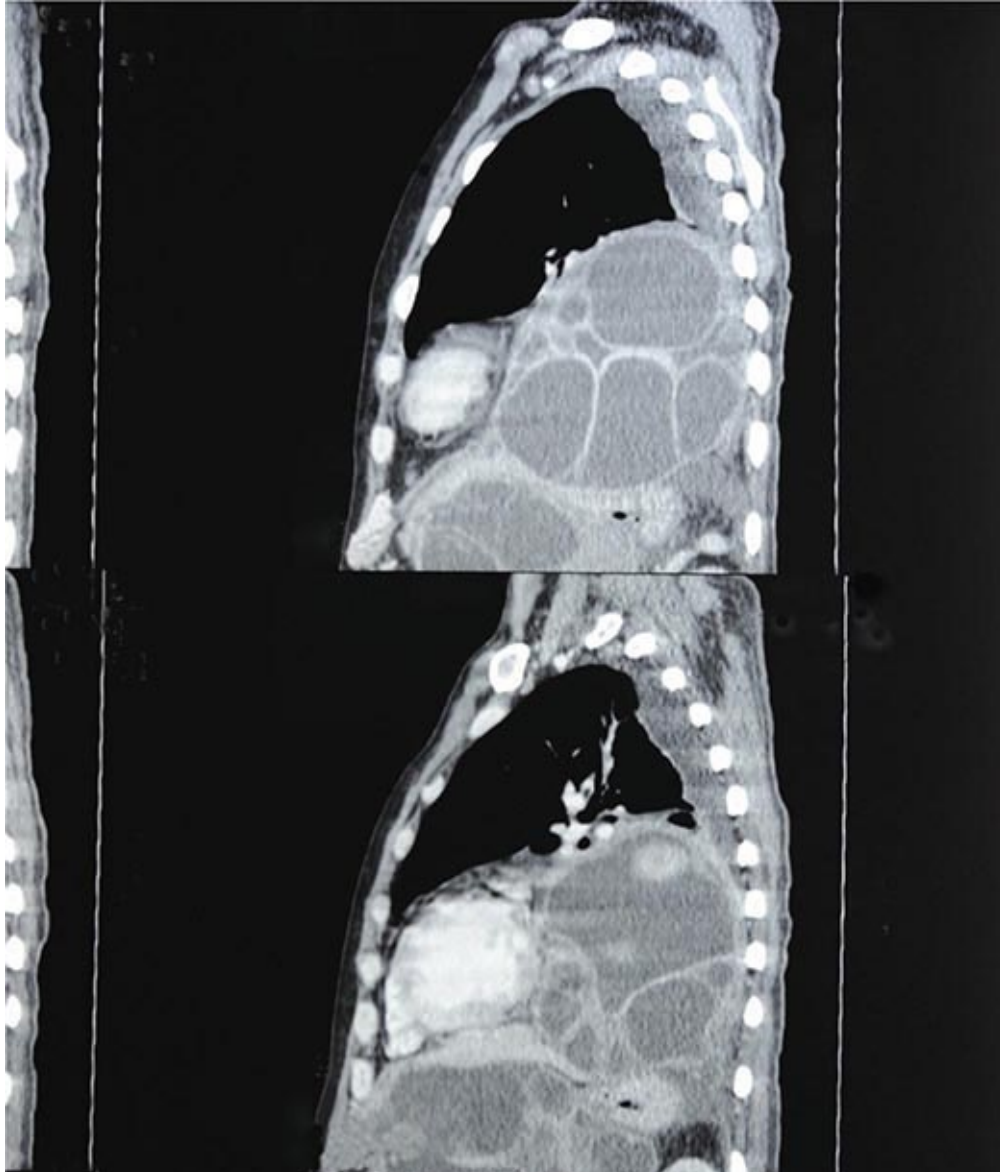


FIGURE 21.35 ■ Hydatid Cyst CT. Multiple echinococcal cysts are seen in the chest and liver of this middle-aged Latin American man with chronic cough, chest pain, and hemoptysis. (Photo contributor: Lawrence B. Stack, MD.)

LEECH BITES

Clinical Summary

Leeches are parasitic blood-sucking aquatic or land-based invertebrates. Aquatic leeches are worldwide, have relatively weak jaws, and usually attach to thinner

tissues such as the pharynx or vagina. Land leeches are primarily found in tropical and subtropical areas of Asia, the South Pacific, and South America; typically have powerful jaws; can attach to any body surface; are fast and painless feeders; and often detach from the host without being noticed.

Leeches have been historically used for bloodletting, their saliva used to derive modern anticoagulants, and are considered currently for elimination of venous congestion after microvascular surgery. They are not known to directly transmit infectious diseases, but bacterial infection can follow bites. Leeches are prodigious feeders; large infestations can cause anemia. Bites from detached land leeches can bleed for prolonged periods due to injected anticoagulants. Allergic and anaphylactic reactions may occur. Aquatic leeches have potential to cause complications, particularly when attached within the esophagus or airway.



FIGURE 21.36 ■ Leech. Nonengorged leech attached to thigh of patient in remote area of Tasmania. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.37 ■ Leech. Engorged leech attached to toe of a backpacker in Papua New Guinea. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

Treatment of detached leech bites consists of local wound care, hemostasis if needed, tetanus prophylaxis, and antibiotics if secondarily infected. Attached leeches can be detached by using a fingernail to break the suction under the sucker. Other modalities (perhaps associated with increased risk of infection or injury) have been suggested, including saline solution, vinegar, or a lighted match.

Pearls

1. The presence of a small y-shaped lesion on the lower extremity suggests a detached land leech bite.
2. *Aeromonas* infections have been reported following use of medicinal leech therapy. Ciprofloxacin may aid in prevention.
3. Hemostasis with QuikClot gauze has been reported to be effective in patients with prolonged bleeding.



FIGURE 21.38 ■ Leech. Leech bite wound on the leg seen after detachment of a land leech. Note the sharp wound edges and surrounding ecchymosis. (Photo contributor: Seth W. Wright, MD.)

LEISHMANIASIS

Clinical Summary

Leishmaniasis is a protozoan, zoonotic parasite spread by sand fly bites. It is endemic in over 80 countries and has an incubation period of 2 to 6 months, although it may range from days to years, with relapse possible. Infection occurs when flagellated forms of more than 20 infective species of *Leishmania* are injected into the skin, taken up by macrophages, and multiply. Often, the bite goes unnoticed.

The primary *cutaneous* lesion begins as an enlarging papule, develops a scaly or ulcerative lesion with indurated edges and a central crater, and usually heals spontaneously in 6 to 12 months, but may progress to more diffuse syndromes. *Mucocutaneous* involvement or “espundia” is mostly seen in Latin America following initial cutaneous infection with *Leishmania braziliensis*. Nasal congestion and epistaxis may progress to septum perforation and nasal bridge collapse, causing a “tapir nose” deformity. *Visceral* involvement, known as “kala-azar,” involves the spleen and liver. It is associated with fever, anemia, cachexia, and splenomegaly, and may progress to hemorrhagic symptoms,

secondary infections, and GI involvement. Visceral leishmaniasis is generally fatal without treatment.

Coinfection with advanced HIV causes synergistic immunologic disturbances due to the lack of a critical CD⁺ T-cell response.

The characteristic parasites can be identified from a smear or biopsy of the lesion identifying parasite amastigotes (Leishman-Donovan bodies). Culture and PCR are also diagnostic options.



FIGURE 21.39 ■ Cutaneous Leishmaniasis. Lesions in a girl from the highlands of Peru. Most *Leishmania* lesions are on exposed body areas. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.40 ■ Cutaneous Leishmaniasis. Lesion involving the face of an HIV-infected patient. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.41 ■ Mucocutaneous Leishmaniasis. Mucocutaneous leishmaniasis in a patient from the Andes region of Peru caused by *Leishmania peruviana*. (Photo contributors: Shannon Langston, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Management and Disposition

No specific emergency treatment exists for leishmaniasis. Treatment of cutaneous or mucosal disease is with pentavalent antimony, miltefosine, pentamidine, or amphotericin B after referral to an experienced clinician. Protective clothing, bed nets, and insect repellent are the most effective ways of avoiding transmission as the flies are usually active from dusk to dawn. Visceral leishmaniasis should be managed by a tropical medicine or infectious disease specialist as the treatment is difficult and often toxic.

Pearls

1. More than 90% of visceral leishmaniasis cases are in India, Bangladesh, Nepal, Sudan, and Brazil, whereas more than 90% of cutaneous forms are in Afghanistan, Algeria, Brazil, Iran, Iraq, Peru, Saudi Arabia, and Syria.
2. Leishmaniasis is common in the Middle East, and many cases have been identified in troops deployed to that region.
3. Leishmaniasis should be considered in immigrants from Latin America with chronic skin or mucosal lesions.

4. With visceral involvement, the primary cutaneous lesion will usually have resolved before clinical symptoms of kala-azar have developed.
5. After treatment of the visceral form, depigmented or nodular cutaneous lesions that are often confused with leprosy may occur.



FIGURE 21.42 ■ Mucosal Leishmaniasis. Oral mucosal leishmaniasis caused by *L braziliensis*. Mucosal involvement can occur years after spontaneous resolution of a nontreated cutaneous lesion. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

LEPROSY

Clinical Summary

Leprosy (Hansen disease) is caused by *Mycobacterium leprae* and presents with anesthetic skin lesions and peripheral nerve complications. This chronic disease is common in many parts of the developing world, including areas of South America, South Asia, and Africa. Illness severity is inversely proportional to the patient's ability to produce a cell-mediated response and creates a range from localized (tuberculoid leprosy) to disseminated disease (lepromatous leprosy), with many having borderline cases falling between the two extremes.

Localized disease is limited to one to three skin lesions, typically sharply demarcated, flat, hypopigmented, anesthetic plaques with elevated margins. This is often accompanied by easily discernible peripheral nerve thickening. The most severe forms are characterized by poorly demarcated erythematous macules, papules, or nodules. The peripheral nerves have less palpable findings, but more diffuse nerve involvement is seen in a stocking glove pattern. This results in loss of bone length and insensate extremities, often leading to repetitive trauma, infection, and loss of digits. With diffuse infiltration of the face, the characteristic "leonine facies" is exhibited.

Diagnosis is primarily clinical, along with staining of a slit skin smear or biopsy for acid-fast bacilli. PCR can aid in the diagnosis.

Management and Disposition

There is no specific emergency therapy for leprosy. Patients should be referred to the national treatment program or a clinician experienced with leprosy management. Multidrug therapy is curative using varying combinations of dapsone, rifampin, and clofazimine. The class of disease dictates type and length of treatment. An ophthalmic exam is essential due to a high propensity for corneal abrasions and ulcers.



FIGURE 21.43 ■ Leprosy. Typical thickened skin on the external ear of a 31-year-old male with lepromatous leprosy. (Photo contributors: Shannon Langston, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.44 ■ Leprosy Skin Lesions. Anesthetic skin lesions in a Peruvian patient with borderline leprosy. Borderline disease is not as localized as tuberculoid leprosy and not as widespread as lepromatous disease. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Pearls

1. Leprosy acquired in the southern United States is highly associated with armadillo handling.
2. The presence of an anesthetic skin lesion should suggest the diagnosis of leprosy.
3. Multidrug therapy has been made available by WHO free of charge to all patients worldwide and provides a highly effective cure for all types.
4. The simpler WHO classification categorizes leprosy into localized (paucibacillary) or disseminated (multibacillary) disease.



FIGURE 21.45 ■ Leprosy. Thickened skin due to bacillary infiltration. Later stage may require digit amputation. (Photo contributor: David Effron, MD.)



FIGURE 21.46 ■ Leprosy. Chronic foot changes with ulceration and shortening of the toes. Extremity damage in leprosy results from loss of sensation, repeated trauma, neurotrophic atrophy, and direct bacillary deposition. (Photo contributor: Seth W. Wright, MD.)

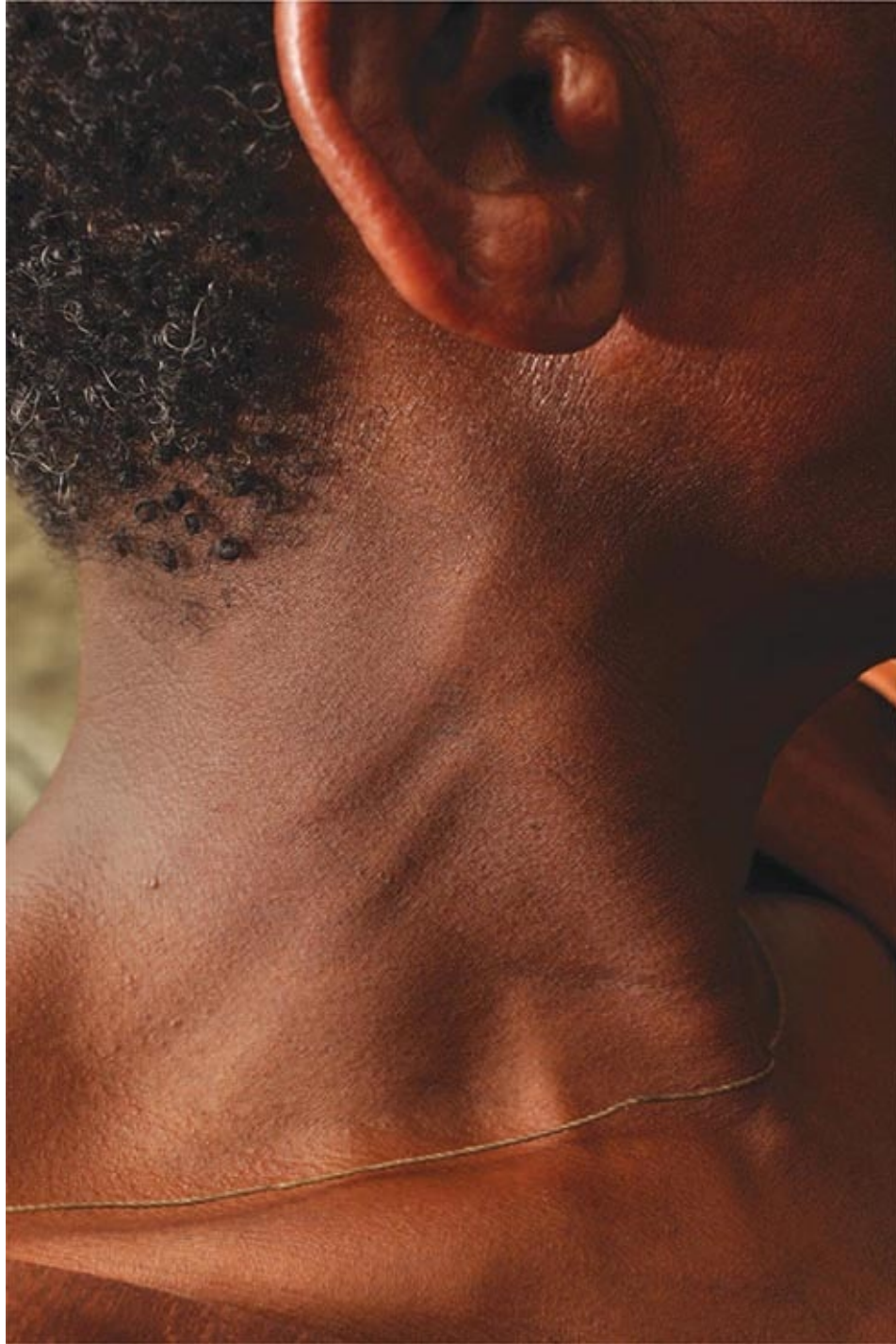


FIGURE 21.47 ■ Leprosy. Enlargement of the greater auricular and transverse cervical nerves of a patient with leprosy. Enlarged peripheral nerves are a cardinal manifestation. (Photo contributor: Lawrence B. Stack, MD.)

LEPTOSPIROSIS

Clinical Summary

Leptospirosis is a zoonotic bacterial disease acquired by exposure to water contaminated by the urine of infected animals. Rodents are the usual reservoir, although pigs, dogs, and cattle can harbor the organism. It is most commonly attained by indirect skin or mucosal contact through occupational work, such as sugarcane or rice farming, or through recreational activities such as rafting, swimming, and hiking. Slaughterhouse workers can acquire leptospirosis through contact with infected body fluids. Leptospirosis is most common in tropical regions, especially during the rainy season.

The incubation period is usually 7 to 14 days; more than 90% of cases are asymptomatic or mild and self-limited. Typical symptoms include the abrupt onset of high fever, chills, headache, severe myalgias, conjunctival suffusion (dilatation of vessels without inflammation or exudate), cough, jaundice, abdominal pain, rash, or diarrhea. Aseptic meningitis is common. Dermatologic findings are nonspecific and include macular, papular, and petechial rashes. Approximately 10% of patients develop the potentially life-threatening icteric form known as Weil syndrome. The initial symptoms are indistinguishable from the milder form until several days after the onset when complications such as renal failure, liver failure, rhabdomyolysis, pulmonary hemorrhage, or cardiopulmonary failure develop.

Laboratory findings in leptospirosis are nonspecific. An elevated creatine kinase is common. Markedly elevated bilirubin values are seen in Weil syndrome. Culture of the organism is possible, but diagnosis is usually serologic. Rapid test kits are available in some countries.

Management and Disposition

Empiric treatment is often indicated. Outpatient management with doxycycline, penicillin, amoxicillin, or a macrolide can be considered for mild illness. Severe disease is treated with IV penicillin or ceftriaxone. Supportive care, including optimal fluid management, is essential to help prevent or ameliorate renal dysfunction. Severely ill patients often need ventilatory support in an intensive care unit and dialysis.

Pearls

1. About half of the cases reported in the United States are from Hawaii, with

- increased outbreaks during periods of heavy rainfall and flooding.
2. Consider leptospirosis in an acutely febrile adventure traveler with recent freshwater exposure.
 3. The presence of conjunctival suffusions, with or without jaundice, is highly suggestive of leptospirosis.
 4. A Jarisch-Herxheimer reaction may occur after treatment with antibiotics.
 5. Weekly doxycycline is effective prophylaxis for short-term travelers to highly endemic areas.



FIGURE 21.48 ■ Leptospirosis. Jaundice and conjunctival suffusions in a Haitian patient with laboratory-confirmed leptospirosis. The patient also had renal failure and myocarditis, both commonly seen in Weil syndrome. (Photo contributor: Seth W. Wright, MD.)

MALARIA

Clinical Summary

Malaria infects up to 300 million people annually with over 1 million deaths; it is the deadliest vector-borne disease in the world. This parasitic disease is transmitted by the night-biting female *Anopheles* mosquito and is caused by four

protozoa of the genus *Plasmodium* (*Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium ovale*, and *Plasmodium vivax*), with *P falciparum* causing the most morbidity and mortality. It is most common in tropical areas, particularly sub-Saharan Africa and Southeast Asia, with specific species predominating in each area.

The parasites undergo a hepatic cycle before entering circulating red blood cells and replicating. Lysis of the cell then occurs, releasing toxic by-products and further parasites into the blood, thereby causing cyclical clinical manifestations. Symptoms include fever, rigors, headache, myalgias, and malaise. The classic cyclical fevers do not always occur. *P falciparum* infection can cause massive hemolysis due to overwhelming parasitemia. Parasitized erythrocytes lose flexibility, leading to microcirculatory obstruction, hypoxia of vital organs, and splenomegaly. Symptoms from *P ovale* and *P vivax* may be delayed for many months due to hepatic dormancy. Pregnancy is a risk factor for severe *P falciparum* infection and can result in maternal anemia, prematurity, and increased infant mortality.

Diagnosis is established by identification of the parasites on thick and thin smears. Rapid antigen kits are available in some regions. Bed nets, insecticides, and protective clothing are effective adjuncts for prevention. Chemoprophylaxis with an appropriate agent is highly recommended for travelers to endemic regions.

Management and Disposition

Treatment depends on geographic location, suspected species, and illness severity. Close adherence to CDC, WHO, or national guidelines is highly recommended for both prophylaxis and treatment. Oral treatment of uncomplicated malaria is feasible with one of several regimens. Quinidine is the only approved treatment for severe malaria in the United States, although artesunate is available on an emergency basis from the CDC when quinidine is not tolerated or available. Artemisinin combination therapy for severe disease is now available in many endemic countries, although IV quinine remains a common treatment. Patients in developing countries are often treated as outpatients. With rare exception, returning travelers with suspected malaria should be admitted for management and treatment. Admission is always warranted for patients with suspected or confirmed *P falciparum*, symptoms of cerebral malaria, children, pregnant women, or immunocompromised individuals.

Pearls

1. Malaria should be strongly considered in any patient exhibiting fever following recent travel to the tropics. It is the single most common cause of fever in this population.
2. An emerging species, *Plasmodium knowlesi*, causes severe disease, but is currently limited in distribution to Southeast Asia.
3. The average incubation period for *P falciparum* is about 13 days; average incubation periods are longer for the other species. Incubation periods for all species can be variable.
4. Parasitemia fluctuates over time, with the highest incidence during episodes of fever. Failure to identify the parasites on initial smears is not an indication to withhold therapy when the diagnosis is likely.
5. Residents of highly endemic areas will often have partial immunity to malaria due to repeated exposure. Immigrants visiting their native country are at high risk for severe infection due to loss of immunity.



FIGURE 21.49 ■ Malaria Jaundice. Jaundice due to acute hemolysis in a Haitian child with documented *P falciparum* infection. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.50 ■ Malaria. Hemoglobinuria following hemolysis in the patient from [Fig. 21.49](#). The patient was severely anemic with a hemoglobin of 3.0 mg/dL. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.51 ■ Malaria. Massive hyperreactive malarial splenomegaly in a surgical patient. This condition can occur with chronic infection, but malaria smears are often negative. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.52 ■ Malaria. A toddler with cerebral malaria. Abnormal motor posturing including decorticate, decerebrate, and opistho-tonic posturing are all common in this condition. (Photo contributor: Seth W. Wright, MD.)

MUMPS

Clinical Summary

Mumps is a highly contagious viral disease best known as a cause of parotitis. Vaccination has markedly reduced the rate of disease in the developed world, but many resource-limited countries do not include mumps in their vaccine programs. It remains common worldwide; occasional large outbreaks occur even in countries with well-developed vaccine programs. Mumps is most common in children and spread by respiratory droplets, direct contact, or fomites. Subclinical infection is common in younger children. Disease in adolescents and adults is associated with a higher rate of serious complications.

A nonspecific illness with malaise, myalgias, fever, and headache follows an incubation period of about 2 to 3 weeks. Unilateral or bilateral parotid swelling is the hallmark, although orchitis is seen in about one-third of postpubertal males and pancreatitis in about 4% of patients. Diagnosis is usually clinical, particularly in developing countries. A high amylase from a salivary or pancreatic source might be present; viral isolation, PCR, and serology are

available for definitive diagnosis.

Management and Disposition

Mumps is a self-limited, usually mild, disease. Treatment is supportive and consists of antipyretics, fluids, and analgesics. Patients with orchitis might benefit from scrotal support. Patients should be advised to stay home from school or work for 5 days after symptom onset.

Pearls

1. Natural infection confers life-long protection, although rare recurrent cases have been reported.
2. Aseptic meningitis with CSF pleocytosis occurs in more than one-half of patients with mumps, although most are minimally symptomatic. Mumps encephalitis also occurs but is extremely rare.
3. Mumps should be considered in the differential diagnosis of aseptic meningitis, even in the absence of parotitis.
4. Mumps is one of the leading causes of acquired deafness in developing countries.
5. Ovarian and breast inflammation are occasionally seen in females, although it is not as common as orchitis among males. Sterility is rarely seen with either condition.



FIGURE 21.53 ■ Mumps. Parotid swelling in a Haitian girl with mumps. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.54 ■ Mumps. Note the classic submandibular and preauricular enlargement of the parotid gland. (Photo contributor: Centers for Disease Control and Prevention.)

MYCETOMA

Clinical Summary

Mycetoma is a localized, chronic, granulomatous infection of subcutaneous tissue with possible extension to underlying bone. Two classifications include eumycetoma, caused by filamentous fungi, and actinomycetoma, caused by bacteria of actinomycetes species. The organism is inoculated into subcutaneous tissue following minor trauma, most commonly the lower extremity and hand, although it may arise anywhere on the body. Both types are similar clinically; eumycetoma, however, causes more morbidity. Initially, a painless subcutaneous swelling is seen with induration, numerous suppurative nodules, and chronically draining sinus tracts. Remote abscesses may rarely be seen due to hematogenous extension. Actinomycetoma occurs more frequently (60%) and has a much better outcome. Mycetomas are rarely fatal but may cause significant dysfunction and disfigurement.



FIGURE 21.55 ■ Mycetoma. A eumycetoma with typical chronic sinus drainages in the foot and ankle of a 33-year-old agricultural worker in Peru. Treatment was with itraconazole. (Photo contributors: Rob Griedanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Expulsion of “grains” containing aggregates of the organisms is common. The presence of black grains is diagnostic of a fungal origin, while pale grains could be either fungal or from an actinomycetes species. Further identification of the causative organism may be done by means of various stains, culture, or serology.

Management and Disposition

There is no specific emergency management except recognition and referral. Surgical resection of large lesions may reduce organism load, but relapse rates can be as high as 50%. Amputation is common. Eumycetoma is treated with 1 to 2 years of antifungal agents, while actinomycetoma responds to various combinations of trimethoprim-sulfamethoxazole, dapsone, streptomycin, and amikacin.

Pearls

1. Mycetoma is usually seen in young male adults living in rural areas of Africa, Mexico, South America, or India who work as farmers or laborers. Walking or working barefoot is a risk factor for lower extremity disease.



FIGURE 21.56 ■ Mycetoma. A large actinomycetoma of the upper leg with extensive active and healed sinus tracts. (Photo contributors: Stuart Skinner, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

2. Patients may complain of a deep itching sensation rather than pain. If pain is present, it may indicate secondary infection or bone involvement.
3. Sweating of the affected area is commonly seen.

4. A similar condition, botryomycosis, is caused by a chronic *Staphylococcus* infection with sinus formation.
5. Mycetoma was first described in the Madura district of India and is often referred to as “Madura foot.”

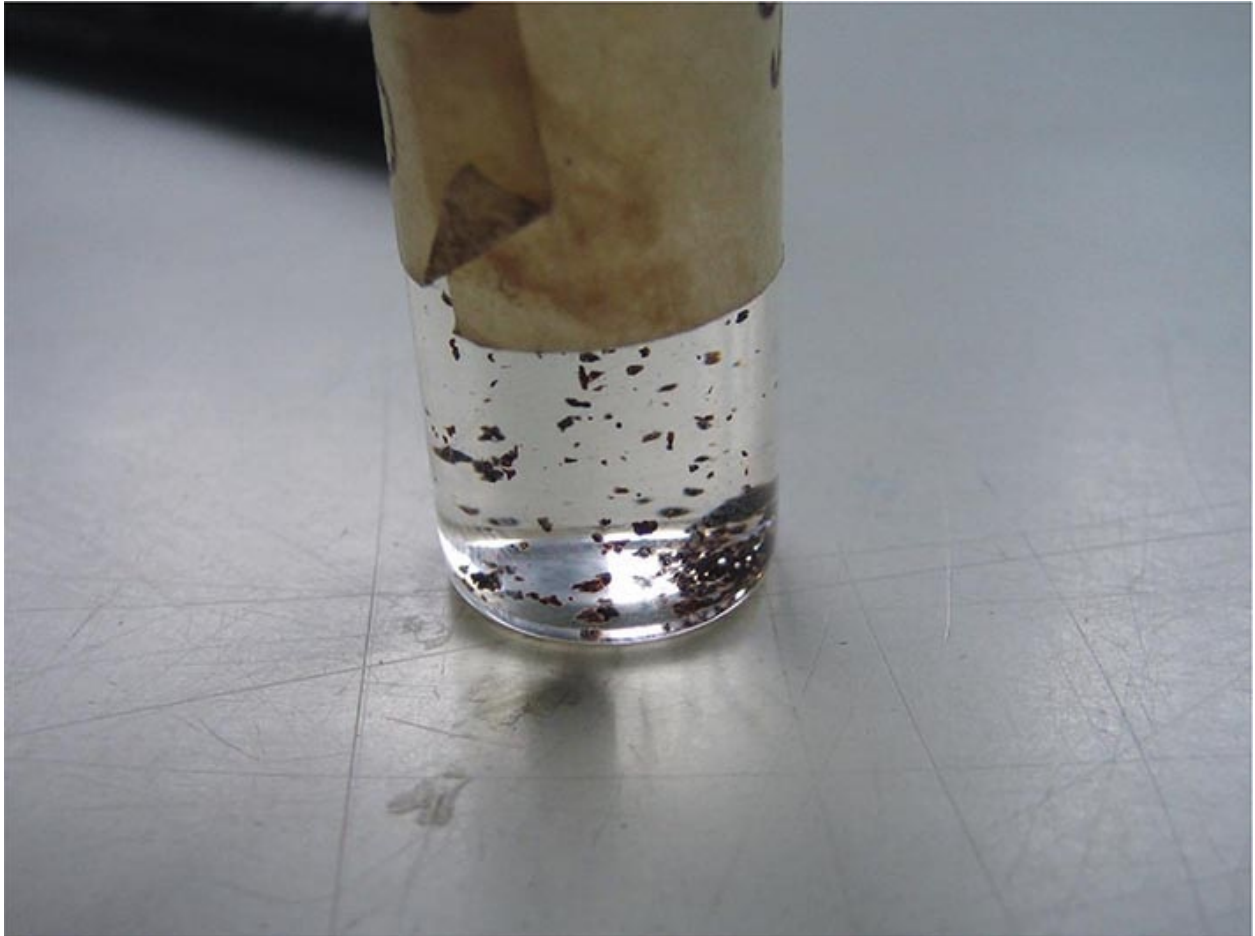


FIGURE 21.57 ■ Mycetoma Granules. Sinuses discharge characteristic dark granules (sclerotia) from a eumycetoma. The granules represent microcolonies of the organism. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.58 ■ Mycetoma. Mycetoma of the foot from *Nocardia* sp.: typical location with multiple draining sites. (Reproduced with permission from Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, Wolff K. *Fitzpatrick's Dermatology in General Medicine*. 8th ed. New York, NY: McGraw Hill; 2012: Fig. 185-6, p. 2249.)

MYIASIS

Clinical Summary

Myiasis is the invasion of living human or animal tissue by maggots, the larvae of flies. Infection is most commonly sub-cutaneous, but may be seen in wounds and body cavities. The human botfly (*Dermatobia hominis*) is a major cause of furuncular myiasis in the New World; mosquitoes courier eggs to the host. A papule develops as the larvae feed, followed by a pruritic furuncle, often with drainage from a central punctum. After maturation, the larva emerges and falls to the ground, where it pupates in the soil and evolves into an adult fly.

The tumbu fly, found in Africa, also causes furuncular myiasis and is spread from eggs deposited in soil, sand, or clothes. Screwworm species are found in

the Old and New Worlds. These worms are notorious for direct deposition of eggs after flying into the nasal cavity, leading to nasal cavity myiasis. Feeding maggots can cause extensive tissue damage. Wound myiasis, caused by numerous fly species, is seen in open sores and gangrenous tissues. Wearing protective clothing, using insect repellent, and covering wounds are preventative measures.

Management and Disposition

Lidocaine injection at the lesion base, or application of suffocating occlusive substances such as Vaseline, may cause the larva to surface and facilitate removal in furuncular myiasis. Incisional extraction is challenging due to the larva's tapered shape and many rows of spines and hooks that it uses to grip tissue. Wound myiasis is treated with thorough debridement. Secondary infection is the only indication for antibiotics; it is uncommon due to bacteriostatic activity in the gut of the larvae, but may occur by leaving portions of them after removal.

Pearls

1. Patients with lesions may have the sensation of something moving under the skin.
2. Myiasis is self-limiting and usually not harmful, but it does cause psychological distress to the host, especially when the larvae surface. Although rare, deaths from meningitis have occurred after tissue penetration from infection in the eye, nose, or ear canal.
3. Screwworms are major pests of livestock. A sterile male release program has eliminated the screwworm from the United States.
4. Some feel that fly maggots are a useful method of wound debridement. Packets of fly maggots have been commercially developed for this purpose.



FIGURE 21.59 ■ Myiasis. Preserved specimens of *D. hominis*. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.60 ■ Wound Myiasis. Wound myiasis in an elderly patient following dressing removal. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.61 ■ Myiasis. Wound on the arm of a woman who had visited the Amazon jungle. A *D hominis* larva was extracted from the wound. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.62 ■ Tumbu Fly Lesions. Multiple lesions from tumbu fly infestation on the skin of a 14-month-old American boy living in Botswana. The child had been wearing clothes dried on infected ground. (Photo contributor: Anonymous.)

NONTUBERCULOSIS MYCOBACTERIA INFECTIONS

Clinical Summary

Mycobacteria other than tuberculosis and leprosy cause disorders primarily seen in tropical countries. As opposed to *Mycobacterium tuberculosis* and *M leprae*, they are usually environmental saprophytes, not obligate human/animal pathogens. Human exposure may be via inhalation, contaminated water, or preexisting wound contamination. Overall, nontuberculosis mycobacteria can cause four different clinical syndromes: pulmonary disease, regional lymphadenitis, disseminated disease in immunocompromised patients, and skin/soft-tissue infections.

Buruli ulcer is the most important disorder caused by infection with *Mycobacterium ulcerans*. They are seen throughout the tropics but are particularly prevalent in Africa. The ulcer is slow growing with extensive necrosis and deep undermining of wound edges. Serious complications can occur, including osteomyelitis, contractures, and scarring.

A variety of other cutaneous forms of mycobacterial diseases exist. Fish tank granuloma (from *Mycobacterium marinum*) is seen following handling of tropical fish tanks and occasionally in fishermen. Posttraumatic abscesses are caused by the rapidly growing mycobacteria, *Mycobacterium fortuitum* and *Mycobacterium chelonae*.

Disseminated disease from *Mycobacterium avium* complex is a late-stage opportunistic infection in HIV/AIDS but is occasionally seen in immunocompetent individuals.

Management and Disposition

Providers should consider these illnesses in the differential diagnosis when patients present with typical skin lesions and in severely immunocompromised AIDS patients. Buruli ulcer is typically treated for 8 weeks with rifampin and either streptomycin or clarithromycin. Surgical excision with grafting may be needed. Other infections are treated with macrolides or other antibiotics.

Pearls

1. Buruli ulcer is the 3rd most common mycobacterial disease worldwide, after tuberculosis and leprosy.
2. Consider *M marinum* infection in patients with a hand lesion following tropical fish tank manipulation.
3. Fast-growing mycobacteria such as *M chelonae* cause “cold abscesses,” often seen after nonsterile injections that are given for cosmetic purposes. A recent

outbreak of this organism was also seen following tattooing.

4. Some postulate that Crohn disease and sarcoidosis may be caused by yet unidentified mycobacteria.
5. *Mycobacterium haemophilum* is considered the 2nd most common cause of cervical lymphadenitis in children.



FIGURE 21.63 ■ Buruli Ulcer. Characteristic shallow base with undermined edges involving the knee. (Photo contributors: Shannon Langston, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.64 ■ Cold Abscess. A cold abscess following a cosmetic injection for weight loss. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.65 ■ Cold Abscesses. A patient with multiple recurrent cold abscesses from *M chelonae* following nonsterile weight loss injections in Peru. (Photo contributors: Rob Greidanus, MD, and

PROTEIN-ENERGY MALNUTRITION— KWASHIORKOR AND MARASMUS

Clinical Summary

Protein-energy malnutrition (PEM) applies to a group of disorders including kwashiorkor and marasmus. These are characterized by an imbalance between the body's supply and demand of energy and nutrients. Kwashiorkor means “the sickness of weaning” as it is often seen following weaning after birth. It usually occurs between the ages of 1 and 4 with a deficiency of dietary protein in the presence of normal to high carbohydrate intake. The etiology is complex, but there is decreased synthesis of proteins resulting in hypoalbuminemia.

Kwashiorkor is an acute illness manifested by edema secondary to fluid and sodium retention. Patients have peripheral edema, anasarca, moon facies, apathy, and a protuberant abdomen secondary to hepatomegaly. The skin can become hyperkeratotic and may split open in pressure-prone areas.

Marasmus is often seen under the age of 1 and is associated with inadequate intake of both protein and calories. The body's own energy stores are utilized, resulting in emaciation. These individuals do not have edema, but rather a loss of subcutaneous fat, muscle wasting, and wrinkled loose skin.

Marasmickwashiorkor refers to the combination of both forms simultaneously. Laboratory studies are useful, but often are not available. Patients with PEM may be left with permanent neurologic and physical deficits due to lack of calories, vitamins, and essential amino acids.

Management and Disposition

These conditions are rarely seen or treated in developed countries, so acute treatment is often in settings with limited resources. These children often have other acute illnesses and are also at greater risk for infection, specifically pneumonia, gastroenteritis, and sepsis. IV fluids are limited to those with shock as they are prone to congestive heart failure; WHO oral rehydration salt solution is usually used for the first 6 to 10 hours. Low-protein milk formula is then started with a goal of 100 kcal/kg/day. A normal diet is gradually started over a

few weeks. Inpatient admission for evaluation, intervention, and arrangement of long-term care is advised when possible.



FIGURE 21.66 ■ Kwashiorkor. A Zambian child with typical light hair, moon facies, peripheral edema, and dry skin of kwashiorkor. (Photo contributor: Meg Jack, MD.)



FIGURE 21.67 ■ Marasmus. An African infant with severe marasmus due to poor feeding following maternal death. The infant is severely underweight with loose skin and little subcutaneous fat. (Photo contributor: Meg Jack, MD.)



FIGURE 21.68 ■ Kwashiorkor. A Ugandan boy with edema typical of acute kwashiorkor. The “enamel paint peeling” rash is occasionally seen. The patient died several days after the photo was taken. (Photo contributor: Seth W. Wright, MD.)

Pearls

1. PEM is one of the leading causes of death among children younger than 5 years of age worldwide.
2. Depigmentation of dark hair causes it to turn reddish, and curly hair may become straight and brittle. Intermittent periods of proper nutrition may lead to alternating bands of light and dark hair known as the “flag sign.”
3. Kwashiorkor is an acute illness, often of only several days’ duration. It is commonly precipitated by acute infections.
4. The mid upper arm circumference (MUAC) is often used for rapid assessment of nutritional status in developing countries.
5. Newer nutritional products, such as “Plumpy’nut,” have been developed and allow for outpatient management.



FIGURE 21.69 ■ Marasmus. An HIV-positive Ugandan girl recovering from marasmus. She is eating Plumpy'nut, a commonly used ready-to-use therapeutic food. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.70 ■ **Marasmus.** An African boy with marasmus. (Photo contributor: Seth W. Wright, MD.)

RABIES

Clinical Summary

Rabies is a viral zoonotic disease typically acquired through the bite of an

infected animal. Transmission via aerosolized virus has been documented in bat-infested caves and in laboratory workers. Human-to-human transmission has only been documented in transplant recipients. Infection can be prevented by preexposure or postexposure vaccination.

Rabies has the dubious distinction of having the highest case fatality rate of any infectious disease. It is a severe progressive encephalitis and presents in one of two forms. In 80% of cases, patients will develop the encephalitic (furious) form, with typical symptoms of fever, agitation, laryngeal spasms, severe pain on swallowing, hydrophobia, confusion, and a host of other neurologic signs and symptoms. Patients with the paralytic (dumb) form (20%) present with an ascending paralysis like Guillain-Barré syndrome. Patients with either form often have preceding paresthesias and pain at the exposure site, as well as a nonspecific prodromal phase lasting up to 1 week. Autonomic instability is seen in all patients prior to death. The diagnosis of rabies in developing countries is primarily clinical. Fluorescent antibody testing, biopsy, and PCR are done in areas where available.

Management and Disposition

Management of clinical rabies is traditionally palliative as the outcome has been uniformly fatal. Vaccination after development of clinical rabies is futile. Treatment in developing countries is usually limited to sedation with benzodiazepines and other comfort measures. Survival of several patients using the “Milwaukee protocol” has been reported. This uses an induced coma (midazolam and ketamine) with continuous electroencephalography (EEG) monitoring, antiviral agents (amantadine), and aggressive diagnosis and treatment of cerebral vasospasm. This regimen is considered experimental in the United States and is not practical in most developing countries.

Pearls

1. Insect-eating bats cause most US rabies cases, but most patients are unable to recall a bite. Dog bites are the most common cause worldwide.
2. The shortest incubation periods are associated with extensive bites and bite wounds to the face and scalp.
3. Rabies vaccine and immunoglobulin are crucial in avoiding rabies following an exposure. They are, however, contraindicated after the onset of clinical rabies as they might lead to an alteration of the natural immune response.

4. Human-to-human transmission is extremely rare or nonexistent. Nevertheless, postexposure prophylaxis of close contacts is recommended as the virus is present in saliva.
5. A report from Peru has suggested the possibility of subclinical rabies disease in Amazon residents.



FIGURE 21.71 ■ Rabies. A 20-year-old Ugandan patient with furious rabies on the fourth day of clinical illness after a dog bite 2 months earlier. The neck is visibly swollen due to subcutaneous emphysema. The patient developed spontaneous pneumomediastinum due to forceful vomiting during a period of severe laryngeal spasms. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.72 ■ Rabies. Same patient on the sixth day, now unable to swallow or control secretions due to laryngeal pain and spasms. (Photo contributor: Seth W. Wright, MD.)

SCHISTOSOMIASIS

Clinical Summary

Over 200 million people worldwide are infected with schistosomiasis, with approximately 200,000 annual deaths. Transmission has been documented in over 75 tropical countries; it is considered the 2nd most devastating parasitic disease, behind malaria. Humans become infected by contact with blood flukes in contaminated freshwater, usually through bathing, swimming, fishing, or agricultural work. The infectious larval form of the worm, known as cercariae, penetrates the skin, develops into an adult, and deposits eggs causing a host immune response and tissue damage.

Acute symptoms are most often seen in nonimmune individuals such as travelers. Cercarial dermatitis, or swimmer's itch, is a pruritic papular rash discovered immediately following exposure. Katayama fever may occur 2 to 8 weeks after exposure from systemic hypersensitivity and resembles serum sickness: fever, cough, chills, myalgias/arthralgias, diarrhea, lymphadenopathy, and hepatosplenomegaly. Resolution usually occurs after a few weeks, but

patients can progress to coma or death.

Chronic symptoms are related to parasite load and involved organs. Intestinal symptoms (caused by *Schistosoma mansoni*) include abdominal pain, poor appetite, bloody diarrhea, with possible hepatosplenomegaly, periportal fibrosis, ascites, and portal hypertension. Urogenital disease (caused by *Schistosoma haematobium*) typically causes hematuria and dysuria, as well as genital lesions and vaginal bleeding in women. It can progress to fibrosis and calcification of the bladder and ureters, leading to hydronephrosis/nephrosis, infection, glomerular disease, and bladder cancer. Schistosomiasis is also associated with neurologic and pulmonary complications, including transverse myelitis, seizures, and pulmonary hypertension. Diagnosis is made through egg detection via stool or urine microscopy. Anemia and eosinophilia can be found in up to two-thirds of patients. Serology is useful for diagnosis in travelers.

Management and Disposition

Cercarial dermatitis usually resolves within 1 week; topical or systemic glucocorticoids may be considered. Therapy for Katayama fever is supportive, although glucocorticoids might be a useful adjunct. Praziquantel is effective against all forms of chronic disease but is not active on immature worms. Thus, patients with acute Katayama fever require delayed treatment in order to eliminate matured worms. Central nervous system disease is treated with a combination of praziquantel and steroids.

Pearls

1. Hematuria is a common presenting symptom in areas endemic for *S mansoni*. These patients are often treated empirically with praziquantel.
2. Schistosomiasis can impact other infections, leading to more severe disease in patients with hepatitis B or C virus, HIV, and malaria.
3. Prevention includes avoiding high-risk waterways, wearing protective boots, and vigorous toweling/drying following contact/exposure.
4. Swimmer's itch can occur as an initial manifestation of human schistosomal infection but is most often caused by avian schistosomes and is not otherwise infectious.



FIGURE 21.73 ■ Schistosomiasis. Bladder thickening and calcification in a patient with *S haematobium* infection. (Used with permission from the J.D. MacLean Centre for Tropical Diseases at McGill University.)



FIGURE 21.74 ■ Schistosome Cercarial Dermatitis. Rash due to avian schistosomes in a man following freshwater exposure in a Guyana river. Cercarial dermatitis usually follows freshwater exposure, but occasionally is seen with salt water. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.75 ■ Schistosome Cercarial Dermatitis. A highly pruritic papulovesicular eruption on the knee acquired after wading through a slow-flowing creek. (Reproduced with permission from Wolff K, Johnson RA, Saavedra AP (eds). *Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology*. 7th ed. New York, NY: McGraw Hill; 2013: Fig. 28-26.)

TETANUS

Clinical Summary

The infective spores of *Clostridium tetani* are widely distributed in soil and

resistant to heat and disinfectants. Disease occurs when toxin forms after organism growth in wounds; it is characterized by acute onset of skeletal muscle rigidity and convulsive spasm.

The toxin affects inhibitory γ -aminobutyric acid and glycine receptors, leading to unopposed contraction and spasm of skeletal muscle. Initial symptoms involve the facial musculature producing trismus (lockjaw) and risus sardonicus (sneering grin). As larger muscles are involved, one may see opisthotonos, arm flexion and abduction, fist clenching against the thorax, abdominal rigidity, and lower extremity extension. Other symptoms include laryngeal spasm resulting in asphyxia, seizures, hyperthermia, hypertension, diaphoresis, and tachycardia. Reflex spasms may be triggered by minimal external stimuli. Fractures, dislocations, and rhabdomyolysis may occur due to forceful sustained muscle contractions.

Management and Disposition

Diagnosis is primarily clinical. Treatment includes airway protection, metronidazole or penicillin, active immunization with tetanus vaccine, tetanus immune globulin, benzodiazepines, and supportive therapy. Wounds should be cleansed and debrided to eliminate further toxin production. Tetanus immune globulin facilitates removal of unbound tetanus toxin but does not affect toxin attached to nerve endings. Recovery of nerve function requires sprouting of new terminals and formation of new synapses, which may take months to occur.

Pearls

1. There are four clinical forms of tetanus: local, cephalic, neonatal, and generalized disease (most common).
2. Neonatal tetanus occurs in infants born without passive immunity 3 to 21 days after birth. Inoculation usually occurs through infection of the umbilical stump and is common in developing countries.
3. Magnesium sulfate decreases spasms and cardiovascular instability.
4. In developed countries, tetanus is primarily a disease of the elderly, diabetics, and inadequately vaccinated immigrants.
5. Tetanus disease does not always lead to immunity.



FIGURE 21.76 ■ Tetanus. Fatal generalized tetanus in an 8-year-old Haitian child resulting from an infected puncture wound. Severe opisthotonic posturing and rigid jaw clenching are evident. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.77 ■ Tetanus. Abdominal wall contractions in a 12-year-old Ugandan girl with tetanus. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.78 ■ Tetanus. Severe facial tetany. The distorted grin and raised eyebrows are known as risus sardonicus. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.79 ■ Neonatal Tetanus. Neonatal tetanus in a 11-day-old infant. Opisthotonic posturing, muscular rigidity, and inability to swallow are seen. (Photo contributor: Seth W. Wright, MD.)

TRACHOMA

Clinical Summary

Trachoma is the leading cause of infectious blindness in the world. It is endemic in areas of Africa, Asia, Latin America, the Middle East, and aboriginal communities in Australia. It is a chronic follicular conjunctivitis caused by *Chlamydia trachomatis*. Trachoma is prevalent in populations with limited access to adequate sanitation and clean water; it is spread through ocular and respiratory secretions as well as flies.

The distinct *active* phase produces mild itching, irritation, and eye discharge, as well as conjunctival inflammation, particularly of the superior tarsal plate. This may progress to marked photophobia, blurred vision, and eye pain. The *scarring*, or cicatricial, phase occurs after repeated or severe infection; chronic inflammation causes the upper lid to shorten (entropion), with subsequent eyelash inversion (trichiasis). Trichiasis may cause painful corneal abrasions and subsequent corneal edema, ulceration, scarring, opacities, and, ultimately,

blindness. Lacrimal gland involvement leads to dryness and increased irritation. Trachoma usually affects both eyes. Diagnosis is largely clinical but may be confirmed by culture. Community-based efforts on hygiene education and behavior modification can decrease the incidence.

Management and Disposition

For acute and subacute infections, the most effective treatment is a single dose of azithromycin; tetracycline ophthalmic ointment is an alternative. Eyelid surgery to correct trichiasis and entropion may prevent blindness.

Pearls

1. Trachoma was once endemic to North America and Europe, but has disappeared with improved sanitation and living conditions.
2. It is extremely contagious and may be spread through direct contact with eye, nose, or throat secretions.
3. Young children are particularly susceptible, but the disease progresses slowly and the more painful symptoms may not emerge until adulthood.
4. Adult women are at much greater risk due to their close contact with small children, who are the main reservoir of infection.
5. The WHO is attempting to eradicate trachoma by 2020 through their public health program known as SAFE. This multifaceted approach involves surgery for advanced disease, antibiotics, facial cleanliness, and environmental/sanitation improvements.



FIGURE 21.80 ■ Trachoma. Extensive inflammatory response with trichiasis. (Photo contributor: Seth W.

Wright, MD.)



FIGURE 21.81 ■ Trachoma. Chronic corneal scarring with excessive tearing in an African patient. (Photo contributor: Meg Jack, MD.)

TRADITIONAL MEDICINE IN THE TROPICS

Clinical Summary

According to WHO, traditional medicine is the sum of the knowledge, skills, and practices based on the theories, beliefs, and experiences indigenous to different cultures, whether explicable or not, used in the maintenance of health as well as in the prevention, diagnosis, improvement, or treatment of physical and mental illness. Such practices are common worldwide and, while frequently used in developed countries, they are even more prevalent in the tropics. As many as 80% of people in some countries rely upon traditional medical practices as their primary form of health care. Poverty, lack of education, decreased access to standard medical care, and mistrust of Western medical concepts all play a role.

Traditional medicine practices are as varied as the societies of the world. Local practices for acute and chronic illnesses might include prayer, meditation, diets, fasting, massage, exercise, herbal remedies, acupuncture, skin scraping, and scarification. Underlying principles of traditional therapies vary greatly; in some societies, they are related to the balance or homeostasis between negative (bad, dark, devil, etc) and positive (good, light, angels, etc) forces. Ayurvedic medicine is practiced throughout South Asia and roughly translates to “the science of life.” This common practice seeks to promote spiritual harmony based

upon the theory that health exists when there is a balance between body, mind, and spirit. Other concepts, such as witchcraft or the “evil eye,” are prominent in dozens of countries. The evil eye, or *ocularis sinister*, is thought to be the cause of a curse, spell, misfortune, or disease, and various amulets, decorations, or procedures may be used to ward off the unwanted effects.



FIGURE 21.82 ■ Coin Rubbing. Typical coin-rubbing marks on the back of a Southeast Asian immigrant with a minor illness. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.83 ■ Evil Eye Make-Up. Make-up on a girl in Bangladesh used to ward off the “evil eye,” a sickness transmitted by someone who is envious, jealous, or covetous. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.84 ■ Traditional Healing Practice. Razor marks placed by a traditional healer in a Zambian patient with fever and cough. A chest x-ray showed an infiltrate corresponding to the razor marks. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.85 ■ Fire Cupping. Fire cupping is used in traditional Chinese medicine for a variety of ailments including musculoskeletal pain and various respiratory, digestive, and gynecologic diseases. The distinctive temporary cupping marks develop as a result of vacuum formation within the cups as the heated air cools. (Photo contributor: Allison Bollinger, MD.)



FIGURE 21.86 ■ Traditional Healing Practice. Use of plant material for healing of an otherwise untreated open ankle fracture following the 2010 Haiti earthquake. (Photo contributor: Seth W. Wright, MD.)

Pearls

1. More than 70% of Americans using traditional therapies will not inform their physician of their use.
2. The majority of pediatric fevers from malaria are treated with herbal medicines at home in many African countries.
3. Coin rubbing (*cao gio*) is a common treatment for minor illnesses in Southeast Asia and is frequently seen in US immigrants. This should not be mistaken for child abuse.
4. Many common medications, including important antimalarial agents, are derived from traditional herbal remedies.
5. Ayurvedic and Unani systems have a recognized place in national health programs in India and have hundreds of thousands of registered practitioners.

TROPICAL SPOROTRICHOSIS

Clinical Summary

Sporotrichosis is a subcutaneous infection caused by the fungus *Sporothrix schenckii* and is acquired from moss, decaying vegetation, hay, and soil. It is usually seen in those whose vocation brings them into contact with the environment. The two primary manifestations involve the cutaneous and lymphocutaneous systems; however, osteoarticular, pulmonary, and disseminated forms (primarily in immunocompromised patients) may be seen from direct inoculation or through hematologic seeding.

The extremities are usually involved with the initial lesion at the site of injury from a thorn, barb, pine needle, or wire. After approximately 1 to 10 weeks, a localized red, purple, or pink papule develops, often resembling an insect bite. The papule evolves into one or more nodules that form painless chronic ulcers with a nonpurulent, clear discharge. In the lymphocutaneous form, the nodules will progress proximally along lymphatic tracts and blood vessels. Many strains do not grow at temperatures above 35°C, decreasing their ability to spread and commonly resulting in a localized lesion. It is not communicable from person to person, although it may be acquired through exposure to infected animals, with cats being the most infectious.

Wearing gloves and long sleeves while working in the outdoors and avoidance of skin contact with sphagnum moss are the mainstays of prevention. Confirmatory diagnosis is made with biopsy or culture.

Management and Disposition

Treatment with oral itraconazole for 3 to 6 months, or alternatively potassium iodide or terbinafine, is effective, with complete recovery in cutaneous and lymphocutaneous forms. Variable response to treatment, often initially with amphotericin B, is seen in patients with severe disease or systemic involvement.

Pearls

1. This diagnosis should be considered when a cutaneous lesion is found on a patient involved with landscaping, rose gardening, Christmas tree farming, berry picking, or baling of hay, and in veterinarians.

2. Lesions typically are noted on the distal upper extremity, and the patient may have already failed multiple treatments with antibacterial agents.
3. While first described in the southern and central United States, it is most common in Mexico, Central/South America, Japan, Australia, and Africa. In Peru, the incidence is approximately 1 in 1000 people.
4. Sporotrichosis is easily confused with leishmaniasis, and the two illnesses coexist in many locations. Lymphatic spread is more characteristic of sporotrichosis, but possible with either. Other diseases or causes on the differential include *Nocardia*, tularemia, and mycobacteria.



FIGURE 21.87 ■ Sporotrichosis. Typical sporotrichoid spread up the arm from an inoculation of the hand. Note the ulcers that resemble pyoderma gangrenosum in this Panamanian child. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. *The Color Atlas of Family Medicine*. 2nd ed. New York, NY: McGraw Hill; 2013: Fig. 174-13.)



FIGURE 21.88 ■ Sporotrichosis. Typical lymphatic spread of sporotrichosis along the lateral aspect of the foot in a 30-year-old resident of the Peruvian Amazon. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

TUBERCULOSIS

Clinical Summary

Tuberculosis (TB) is a chronic bacterial infection spread from human to human through respiratory droplets containing *M tuberculosis*, an acid-fast bacillus. Upon inhalation, the organisms are transported to regional lymph nodes where the immune system forms granulomas or “tubercles.” Most people undergo complete healing following exposure with only a positive purified protein derivative (PPD) test. However, it may lie dormant as latent disease until the immune system is suppressed, leading to organism release. TB occurs worldwide but is more common in impoverished nations.

The organisms are dependent on high oxygen content and are typically found in the upper lobe or superior segment of the lower lobes of the lungs. Although it is primarily a respiratory illness, 15% will exhibit extrapulmonary manifestations involving the adrenal glands, long bones, vertebrae, GI tract, GU tract, skin, lymph nodes, meninges, pericardium, or peritoneum. Patients with HIV infection have a much higher overall prevalence and rate of extrapulmonary

disease. Common symptoms of active TB are fever, night sweats, malaise, weight loss, cough, hemoptysis, and pleuritic chest pain. Diagnosis is made by the PPD and chest x-ray but is confirmed through acid-fast bacillus (AFB) smears and sputum culture. The mainstay of diagnosis in resource-limited countries is the AFB smear.



FIGURE 21.89 ■ Tuberculosis Adenopathy. Bilateral cervical adenopathy (scrofula) in a Peruvian child with documented TB. Pediatric cervical adenopathy in highly endemic areas is strongly suggestive, and the most common form, of extrapulmonary TB. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.90 ■ Tuberculosis Scarring. Chronic discharging sinuses with extensive scar formation in a Peruvian man with extra-pulmonary tuberculosis. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.91 ■ Tuberculosis Cold Abscess. Cold abscess on the back of a Haitian woman with untreated Pott disease. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.92 ■ Tuberculosis Pott Disease. Severe spinal deformity (“gibbus”) in an 8-year-old Ugandan boy with Pott disease. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

Typical first-line chemotherapy consists of isoniazid, rifampin, pyrazinamide, and ethambutol for 2 months, followed by isoniazid and rifampin for 4 months. Patients in developed countries are usually admitted with respiratory isolation. Outpatient therapy is common in developing countries. After 2 weeks of treatment, the patient is usually no longer contagious. Poor long-term adherence

is a major contributing factor to the development of multidrug-resistant strains. Directly observed therapy (DOT) where patients are observed taking the medication may be required to ensure compliance and is routine in most countries.



FIGURE 21.93 ■ Abdominal Tuberculosis. Severe wasting and ascites in a Ugandan boy with abdominal tuberculosis. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.94 ■ Clubbing. Clubbing of the fingers in a Ugandan man with longstanding pulmonary tuberculosis. (Photo contributor: Seth W. Wright, MD.)

Pearls

1. Any immunocompromised person with pulmonary symptoms should be placed in respiratory isolation until TB is excluded.
2. Scrofula refers to cervical lymph node involvement of TB, while vertebral involvement is called Pott disease.
3. Isoniazid combined with rifapentine for treatment of latent TB, given weekly for 12 weeks, is now an alternative to the 9-month regimen of daily isoniazid.
4. TB is the most important opportunistic infection in HIV patients in sub-Saharan Africa. The HIV epidemic has led to a massive increase in the incidence of TB in many countries.



FIGURE 21.95 ■ Pulmonary Tuberculosis. Severe wasting in a Sudanese soldier with smear-positive pulmonary tuberculosis. His initial weight was 38 kg with a body mass index (BMI) of 11. His weight increased to 52 kg and BMI to 15 by the end of the 2-month intensive phase of treatment. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Tungiasis is an ectoparasitic condition caused by the cutaneous penetration of a *Tunga penetrans* flea. These tiny fleas are commonly found in sandy beaches and areas with warm, dry soil. Risk factors for infection include household dirt floors, lack of closed footwear, and the presence of farm animals. In some poverty-stricken regions, the prevalence of tungiasis approaches 50%. Tungiasis is also common in returned travelers, usually following beach vacations.

The female flea burrows into the skin of a host, feeds on blood, matures, lays eggs, and eventually dies in the cutaneous tissue. The rear of the flea remains visible as a black spot and serves for respiration and excretion of feces. A pronounced inflammatory response is seen leading to characteristic lesions. The vast majority of lesions are seen on the feet with the area at the rim of the toenails being the preferred site.

Early-stage lesions demonstrate erythema surrounding a 0.5- to 2.0-mm black dot. A yellowish-white halo then develops around the dot with increasing erythema, pain, and pruritus. Later stage lesions develop a brownish coloration with crusting and desquamation. Secondary bacterial infection is common. Tungiasis is usually a clinical diagnosis; a small central dot representing the flea is an important clue. A biopsy will show the flea, but is not required for diagnosis.

Management and Disposition

No oral drug has been proven to be effective against an embedded flea. Occlusive ointments will kill the flea, but does not aid in removal. The flea can be removed with excision or gently with a needle. Untreated, the flea will eventually die after 4 to 6 weeks with subsequent sloughing of the flea and eventual resolution of the inflammatory lesion. Antibiotics are indicated for secondary infection.

Pearls

1. Tungiasis is known by numerous local names including sand fleas, jiggers, bicho de pie, nigua, pigue, suthi, and chigoe.
2. The causative flea is native to the West Indies and was first reported in a crewman who sailed with Christopher Columbus. They are now widely found throughout Latin America, Africa, and Asia.

3. The initial stage of penetration is manifested by a stinging sensation followed by itching that is surprisingly described as being pleasant.
4. Numerous cases of tetanus and gangrene have been reported in patients with secondarily infected *T penetrans* lesions.



FIGURE 21.96 ■ Tungiasis. Acute tungiasis lesion from an adult flea seen on the foot of a 30-year-old American aid worker in the Democratic Republic of Congo. Lesions are more obvious in patients who do not habitually walk barefoot. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.97 ■ Tungiasis. Chronic lesions in a Kenyan man. Crusted scales are common in those with chronic infections and thicker skin. (Photo contributor: Lawrence B. Stack, MD.)

ZIKA VIRUS

Clinical Summary

Zika virus is a viral disease related to dengue fever. A flavivirus, Zika is an emerging virus that has spread from its origins near equatorial Africa and Asia to the Americas. It is transmitted primarily by the bite of the *Aedes aegypti* mosquito and to a lesser extent *Aedes albopictus*. Sexual transmission has been reported, and Zika has been detected in semen for greater than 90 days following infection. In utero transmission has been reported; thus, safe sex precautions should be observed for 90 days following exposure.

Symptoms of infection are generally mild, with patients reporting a low-grade fever, a maculopapular rash that may start on the face and spread to the remainder of the body, associated arthralgia, and conjunctivitis. Headaches are common. The incubation period for Zika virus is 3 to 14 days. Previous infection with Zika usually precludes future infection due to immunity.

The virus is associated with an increased incidence of Guil-lain-Barré syndrome. It is known to cause severe congenital defects including microcephaly

and chorioretinal scarring if contracted during pregnancy. The chance of congenital birth defects is highest with first-trimester infection and diminishes for illness contracted later in pregnancy. The incidence of congenital birth defects following congenital disease infection is estimated to be 5% to 10%. Previous infections with dengue may provide some cross-immunity to Zika infection. Although laboratory abnormalities are not common, occasional leukopenia, thrombocytopenia, and mild transaminitis are sometimes identified. Diagnosis can be confirmed with a reverse transcriptase PCR (RT-PCR) in the acutely ill patient. The virus may be detected in the urine up to 14 days after onset of symptoms. Later diagnosis can be confirmed by IgG and IgM antibodies to Zika virus.



FIGURE 21.98 ■ Presentation of Zika virus infection. (A) Edema and erythema of the malar region of the face and conjunctival injection. (B) Macular rash on the abdomen. (C) Hyperemia and petechiae in the hard palate. (D) Tender, mobile, soft lymph node, about 15 mm in diameter, behind the left ear.(Reproduced with permission from Brasil P, Calvet GA, de Souza RV et al. Exanthema associated with Zika virus infection. *Lancet Infect Dis.* 2016;16(7):866. Copyright © Elsevier.)

Management and Disposition

Supportive therapies for pain, fever, and rash are the mainstays of therapy. False-

positive testing with IgM is usually observed due to cross-reaction with other flaviviruses. Aspirin and nonsteroidal anti-inflammatory drugs should be avoided as there is a risk of increased hemorrhagic complications in the event the illnesses caused by Dengue fever. Those who are planning on conceiving should wait at least 3 months after exposure to Zika or travel to endemic regions before engaging in unprotected sex. Admission is indicated for those with severe symptoms or an uncertain diagnosis.

Pearls

1. Consider Zika virus infection in recently returned travelers from the Caribbean and South and Central America.
2. Pregnant women with evidence of a possible Zika virus infection should have serial fetal ultrasounds every 3 to 4 weeks for evaluation of fetal abnormalities.
3. Although most cases of Zika in the continental United States have been imported by travelers to endemic regions, local mosquito-borne transmission has been reported in Florida and Texas.

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Chapter 22

AIRWAY

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Team Intubation. Safe and successful intubation often requires a team effort. Here the laryngoscopist is assisted by personnel providing lip retraction, thyroid manipulation, endotracheal tube balloon insufflation, and cervical spine stabilization. (Photo contributor: Lawrence B. Stack, MD.)

AIRWAY MAXIM ONE

MAXIM: Endotracheal intubation is not always the best initial intervention for respiratory failure.

Summary

Some patients in respiratory distress may benefit from other interventions, short of intubation. Patients with flash pulmonary edema may have dramatic improvement with intravenous nitroglycerin, intravenous furosemide, and bilevel positive airway pressure (BiPAP) ventilation (see [Fig. 22.1](#)). Patients with airway narrowing (edema, neoplasm, stricture, foreign body) can have significant decreased work of breathing by decreasing airway resistance to inspired gas using administration of helium-oxygen (HELIOX) mixture. HELIOX, usually as a 78%:22% helium-to-oxygen mixture, is much less dense than either air or 100% oxygen by virtue of helium replacing nitrogen or oxygen, respectively. This lowers resistance to laminar flow by as much as 25% to 20%, and the effects are immediate.



FIGURE 22.1 ■ Bi-Level Positive Airway Pressure. This patient with chronic obstructive pulmonary disease rapidly improved with the application of BiPAP. (Photo contributor: Steven J. White, MD.)

Pearl

1. Alternative ventilatory adjuncts include HELIOX (see [Fig. 22.3](#)), continuous positive airway pressure (CPAP), BiPAP (see [Fig. 22.1](#)), and Vapotherm (see [Fig. 22.2](#)). These adjuncts may prevent the need for intubation in selected patients.



FIGURE 22.2 ■ Vapotherm. Application of Vapotherm (high-flow warmed oxygen) rapidly improved this toddler with pneumonia and reactive airway disease. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.3 ■ HELIOX Delivery System. An 80% helium 20% oxygen HELIOX delivery system. Use a non-rebreather mask at 8 L/min in patients with suspected upper airway obstruction. (Photo contributor: Lawrence B. Stack, MD.)

AIRWAY MAXIM TWO

MAXIM: *The most important initial airway intervention may be to ask for help.*

Summary

Clinical scenarios where asking for help may be necessary include:

1. Laryngeal injury/tracheal disruption, for whom a nonendoscopic intubation attempt can result in tracheal disruption and fatally lost airway (see [Fig. 22.4](#))
2. Recent neck surgery, with pending loss of airway from an expanding hematoma; definitive and life-saving intervention in this case is to open up the recent incision and evacuate the hematoma.
3. Suspected epiglottitis, where an immediate operative tracheostomy or cricothyroidotomy may be required if intubation fails due to epiglottic edema.
4. Severe angioedema with tongue and oropharynx swelling where endotracheal or nasotracheal intubation may be obstructed (see [Fig. 22.5](#)).
5. Severe facial edema after burns or trauma (see [Fig. 22.6](#)).



FIGURE 22.4 ■ Laryngeal Fracture. This 17-year-old male was kicked in the neck by a bull at a rodeo causing a laryngeal fracture. (Photo contributor: Rudy Kink, MD.)



FIGURE 22.5 ■ Angioedema. This patient with ACE inhibitor angioedema and respiratory failure required awake fiber optic nasotracheal intubation by anesthesia. (Photo contributor: David Effron, MD.)



FIGURE 22.6 ■ Flash Cigarette Burns. This patient with tobacco dependence disorder and oxygen-dependent chronic obstructive pulmonary disease developed facial burns when his cigarette and nasal

canula ignited while lighting a cigarette. These patients typically have partial-thickness facial burns and no airway injury. Intubation is rarely needed. (Photo contributor: Lawrence B. Stack MD.)

Pearls

1. Emergency physicians are emergency airway experts, but know your limitations.
2. Do not let your ego get in the way of proper patient airway management and care.

AIRWAY MAXIM THREE

MAXIM: Most patients with airway/respiratory problems should be positioned for their comfort, not ours.

Summary

If mentating normally and physically able, a patient with airway difficulty will assume a position that optimizes their airway patency and gas exchange, usually sitting up and leaning forward. Such patients include those with incomplete airway obstruction, flash pulmonary edema, and massive airway bleeding from oropharyngeal trauma. Unfortunately, during preparation for intubation, such patients often are placed supine prematurely, increasing the patients' respiratory distress and anxiety, increasing the likelihood of spontaneous emesis and aspiration, and decreasing their ability to handle oropharyngeal bleeding or secretions. In these clinical situations, rethink the desire to immediately place a patient supine for endotracheal intubation. Intubation is accomplished with the patient sitting up, by either:

1. Altering the intubation technique (see [Fig. 22.7](#)); or
2. Altering the intubator position relative to the patient (see [Fig. 22.8](#)).



FIGURE 22.7 ■ Intubation Facing the Sitting Patient or “Tomahawk Technique.” Holding the laryngoscope in the right hand and displacing the mandible forward and passage of the tube with the left hand is an alternative position to supine intubation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.8 ■ Intubation Above the Sitting Patient. Standing above a patient and intubating in the conventional manner is an alternative airway management position in a patient who may be difficult to ventilate in the supine position. An assistant may be necessary to hold the patient upright if sedated and/or paralyzed. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Keep patients in optimal position for spontaneous ventilation until they are sedated just prior to intubation; then, place them supine.
2. Consider intubating a patient sitting upright if you feel the supine position will compromise the patient's ability to be ventilated.
3. Titrating ketamine in small doses (10 mg every 1-2 minutes) can facilitate

awake intubation in a patient sitting upright.

AIRWAY MAXIM FOUR

MAXIM: *Think before you paralyze.*

Summary

Before committing to rapid-sequence induction (RSI) for direct laryngoscopy, consider the following:

1. Are any planned medications contraindicated?
2. Can rescue ventilation be achieved?
3. Is direct laryngoscopy possible? (See [Fig. 22.9.](#))



FIGURE 22.9 ■ Wired Mandible. This 33-year-old male had recent mandibular wire fixation of his mandibular fracture. A nasal or neck approach would be the only options for an emergency airway in this patient if no wire cutters were available. (Photo contributor: David Effron, MD.)

4. What are my secondary and tertiary backup plans in the event of primary plan failure (can't intubate, can't ventilate)?
5. Are my equipment and personnel ready for RSI? (See [Figs. 22.12](#) to [22.14.](#))
6. Is patient resuscitation required prior to RSI? Does the patient require

crystalloid, blood, or vasopressors prior to intubation to avoid cardiovascular collapse?

Pearls

1. A good backup plan for direct laryngoscopy should have at least one alternative intubation technique, one alternative ventilation technique, and one surgical airway technique.
2. Prepared equipment, correct patient position ([Fig. 22.15](#)), proper drug dosing, having a backup plan, effective communication, and good technique will promote first-pass intubation (see [Figs. 22.10](#) and [22.11](#)).



FIGURE 22.10 ■ Difficult Intubation. This morbidly obese man with no identifiable neck landmarks and bushy beard was successfully intubated after the third attempt. Ventilation was extremely difficult after paralysis. (Photo contributor: Lawrence E. Heiskell, MD.)



FIGURE 22.11 ■ Difficult Intubation. Anticipation of difficulty ventilating this patient if paralyzed prompted planning to optimize intubation success, which required three attempts. (Photo contributor: Lawrence E. Heiskell, MD.)

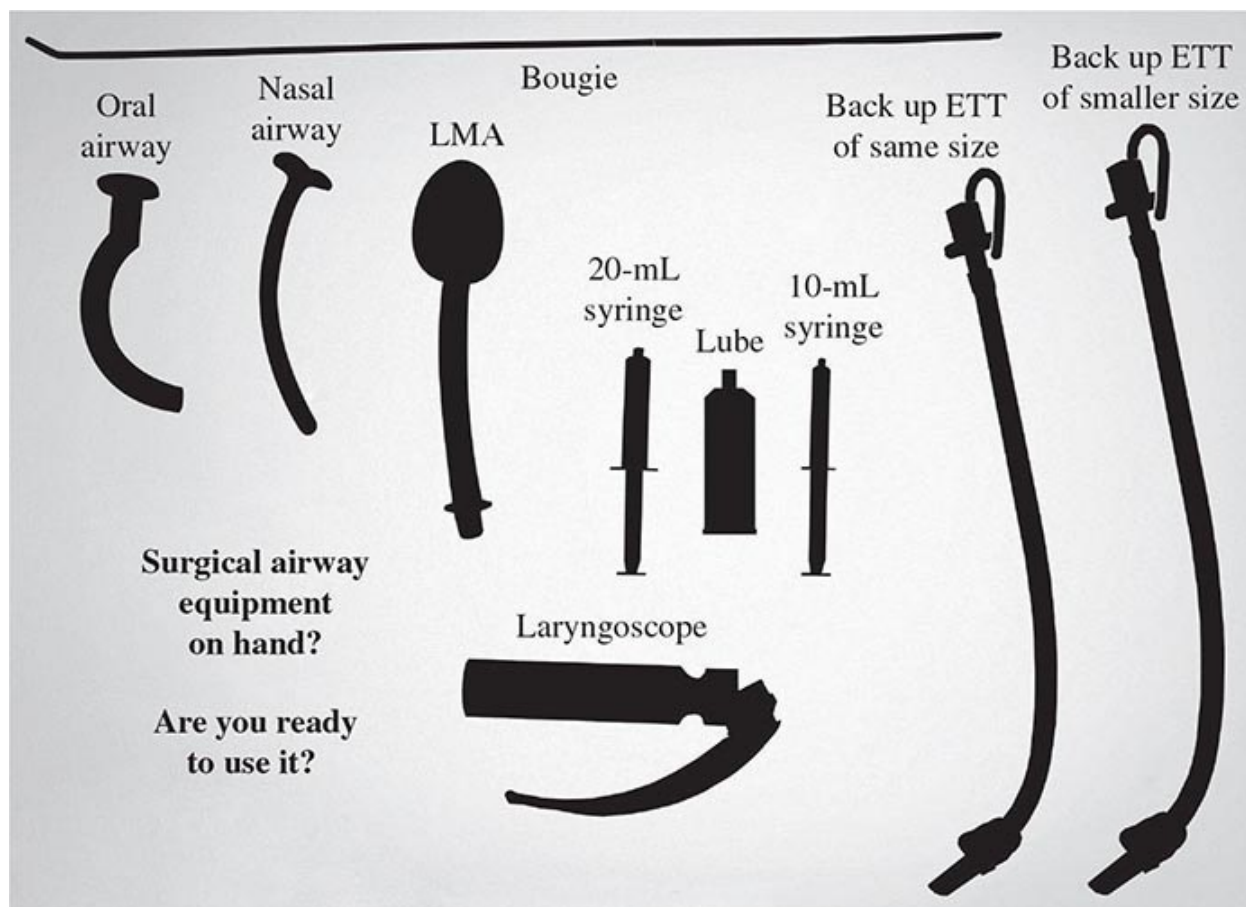


FIGURE 22.12 ■ Airway Equipment Place Map. An equipment place map is a quick visual cue to ensure all my equipment is present. Each item is placed over the silhouette. ETT, endotracheal tube; LMA, laryngeal mask airway. (Photo Contributor: Kevin High, RN.)

Prior to Patient Arrival
All items must be verified by EM resident and attending
Oxygen mask and NC available with flow
Oral airway available
Suction available and on
Blade/handle available and checked
Tube/stylet available and shaped appropriately
Extra tubes/stylet available
Bougie available
Backup devices available (LMA, King, crich kit)
Monitors/Storz screen positioned appropriately
BVM with ETCO ₂ attachment available
IVF available
Individual designated to hold inline stabilization
Airway plan verbalized

FIGURE 22.13 ■ Prearrival Airway Checklist. Preintubation checklists are associated with a reduction in intubation-related complications and a decrease in paralysis-to-intubation time. BVM, bag-valve-mask; EM, emergency medicine; ETCO₂, end-tidal carbon dioxide; IVF, intravenous fluid; LMA, laryngeal mask airway; NC, nasal cannula.

Preinduction Time Out
All items must be verified by scribe and EM attending
Prearrival checklist completed
Airway plan confirmed between trauma and ED attendings (including bougie, LMA, surgical airway)
IV functioning
RSI drugs/doses confirmed and drawn up
Inline stabilization designated
Preoxygenation (NC = mask) underway
Patient positioning optimized
BP cuff on opposite arm than IV

FIGURE 22.14 ■ Preinduction Timeout Checklist. Checklists for a critical procedure like intubation improve procedural success and patient safety. BP, blood pressure; ED, emergency department; EM, emergency medicine; IV, intravenous; LMA, laryngeal mask airway; NC, nasal cannula; RSI, rapid-sequence induction.

AIRWAY MAXIM FIVE

MAXIM: *Patients will not die if they are not intubated; they will die if their lungs are not ventilated and their blood is not oxygenated.*

Summary

The goal for airway management in any patient must be to maintain adequate ventilation and oxygenation. This does not necessarily mean intubation. Correct bag-valve-mask (BVM) ventilation/oxygenation technique is an underrated skill that will buy you time in the patient with a difficult airway. Proper steps for optimal two-person BVM ventilation include the following:

1. Positioning—ear-to-sternal notch alignment (when clinical scenario permits). Neck slightly flexed and head slightly extended. (See [Fig. 22.15](#).)



FIGURE 22.15 ■ Optimal Ear-to-Sternal Notch Alignment. Optimal position for ventilation and laryngoscopy occurs when the external auditory canal and the sternal notch are aligned in the horizontal plane (B) and the face is parallel to the ceiling (C). This position optimizes airway patency and ventilation mechanics. (Photo contributor: Lawrence B. Stack, MD.)

2. Jaw thrust—displace mandible anteriorly with pressure from long, ring, and small fingers on mandible, not soft tissues.
3. Mask compression—thumb and index fingers should apply firm pressure to face and nasal bridge. (See [Figs. 22.16](#) and [22.17](#).)



FIGURE 22.16 ■ Bag-Valve-Mask Ventilation. Correct positioning and forces during BVM ventilation are demonstrated for the one- and two-person (thumbs) techniques. Upward force on the mandible, not soft tissue, is key to effective jaw-thrust technique. (Photo contributor: Lawrence B. Stack, MD.)

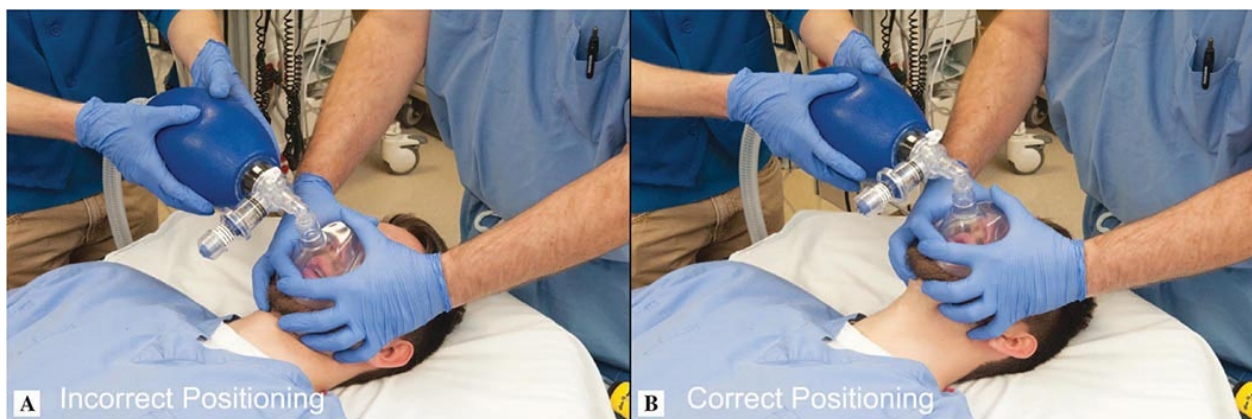


FIGURE 22.17 ■ Bag-Valve-Mask Ventilation. Incorrect (A) and correct (B) positioning and forces during

BVM ventilation are demonstrated for the two-person techniques. Lack of jaw thrust during BVM will allow the tongue to block air passage. (Photo contributor: Lawrence B. Stack, MD.)

4. Oral/nasal airways—may help maintain airway patency during BVM ventilation. (See [Fig. 22.18](#).)



FIGURE 22.18 ■ Nasal and Oral Airways. Appropriately sized and placed nasal and oral airways maximize upper airway patency during BVM ventilation. (Photo contributor: Lawrence B. Stack, MD.)

5. Use 7 mg/kg tidal volume, over 1 to 2 seconds at 12 breaths/min.

Pearls

1. Be an expert at BMV ventilation.
2. Ear-to-sternal notch positioning is most beneficial in obese patients and those with obstructive sleep apnea.
3. Keeping dentures in place facilitates BVM, whereas removing them facilitates orotracheal intubation.
4. Mid-face and mandibular disfiguration from whatever cause will interfere with optimal BVM ventilation.
5. Consider laryngeal mask airway (LMA), King LT, or Air-Q if unable to intubate or obtain adequate seal for BVM ventilation.

LARYNGOSCOPIC OROTRACHEAL INTUBATION

Clinical Summary

Optimizing First-Pass Intubation Success

Laryngoscopic orotracheal intubation is divided into two distinct processes:

glottic visualization and endotracheal tube (ETT) delivery, with the majority of the effort spent on visualization. An intubation attempt is defined as any time a laryngoscope blade enters the mouth. The probability of successful intubation (tube in the trachea without hypoxia) diminishes with successive attempts. Optimizing controllable variables increases first-pass intubation success rates. As in the airline industry, preprocedural checklists and “time outs” provide a consistent process to complex and dangerous procedures, such as landing a plane or endotracheal intubation. Checklists communicate to all involved and allow others to speak out if a process is violated. A prearrival checklist (see [Fig. 22.13](#)) ensures equipment availability and confirms function. An intubation equipment place map (see [Fig. 22.12](#)) provides a quick visual aid to determine if all necessary equipment is present. A pre-induction time out checklist (see [Fig. 22.14](#)) confirms that all the tasks prior to medication administration are in order. Team members are encouraged to speak out if the preparatory tasks are incomplete.

Patient Positioning

The optimal position to maximize laryngoscopic visualization of the larynx is:

1. The head is extended.
2. The neck is flexed.
3. The base of the ear is aligned with the sternal notch (see [Fig. 22.15](#)).
4. The facial plane is horizontal, parallel to the ceiling (see [Fig. 22.15](#)).
5. The facial plane should be positioned between the laryngoscopist’s xyphoid process and umbilicus (see [Figs. 22.19A](#) and [22.19B](#)).



FIGURE 22.19A ■ Incorrect Intubating Height. An incorrect working height creates poor body mechanics, making glottic visualization and tube passage more difficult, especially for prolonged intubation times. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.19B ■ Correct Intubating Height. Optimal height for intubation is typically when the plane of the patient's face is between the laryngoscopist's umbilicus and xyphoid process. (Photo Contributor: Lawrence B. Stack, MD.)

This position most closely replicates in a supine posture that position which the patient would assume sitting up. Large individuals or those with morbid obesity often require creation of a textile ramp of blankets, sheets, or towels to raise the head and shoulders to achieve ear-to-sternal notch alignment.

Preoxygenation and Passive Apneic Oxygenation (See Fig. 22.20)



FIGURE 22.20 ■ Preoxygenation and Passive Oxygenation. In preparation for intubation, simultaneously placing a non-rebreather mask (preoxygenation before induction) and nasal cannula (passive oxygenation after paralysis) at 15 L/min will help prolong tissue oxygenation during the apneic phase of intubation. (Photo contributor: Lawrence B. Stack, MD.)

Preoxygenation, when possible, is used to create an oxygen surplus in the blood and tissue, which permits a period of apnea to occur without arterial oxygen desaturation. This process hopefully begins in the prehospital setting with the placement of a non-rebreather mask with high-flow oxygen on the patient. This continues in the emergency department (ED) in the event intubation is required. In addition, a nasal cannula on high flow (15 L/min) is placed when the patient arrives in the ED if there is a high likelihood of intubation. High-flow oxygen delivered by nasal cannula provides passive apneic oxygenation during the apneic phase of endotracheal intubation.

Technique

Stylet Shape (See Fig. 22.21)

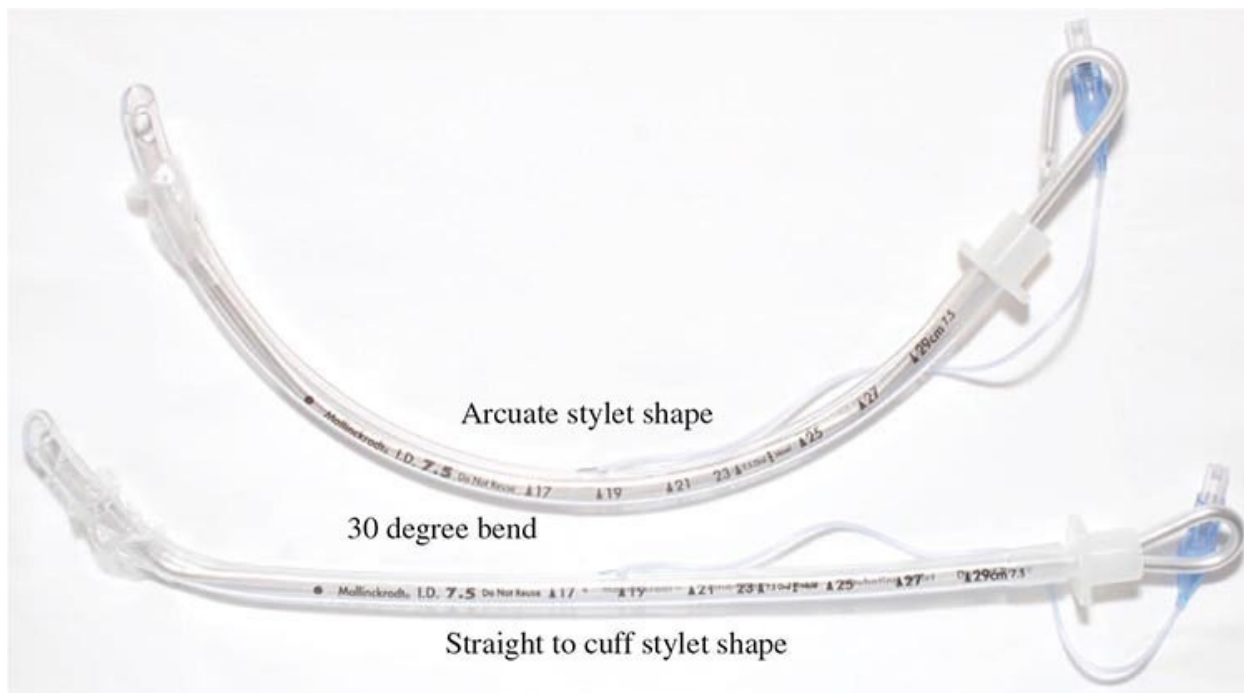


FIGURE 22.21 ■ Stylet Shape. Straight-to-cuff with a 30-degree bend is the optimal stylet shape as this offers the most control of ETT tip and best view of the glottic opening during tube passage. (Photo contributor: Lawrence B. Stack, MD.)

A stylet or bougie should be used with all oral intubations in which a laryngoscope is employed. The laryngoscopist will typically fashion an inexpensive stylet, essentially a malleable wire, into a shape of their preference, to control the distal ETT tip. One should take care not to place too much bend or curvature to the stylet because such a configuration can actually impair the glottic view and control of the ETT tip. Optimal stylet configuration has been described as “straight to the cuff” and then a gentle anterior bend of 30 degrees.

Even with a correct bend on the stylet, it may be difficult to advance the styletied ETT into the trachea. The tip of the left-sided beveled tube can impact and catch on the anterior tracheal cartilages, preventing advancement. This can often be remedied with a generous clockwise or rightward rotation of tube and stylet, which acts to rotate the bevel anteriorly and depress the ETT tip. Other strategies for difficult tube delivery are provided in [Table 22.1](#).

TABLE 22.1 ■ TUBE DELIVERY DIFFICULTY STRATEGIES

Difficulty	Strategy	Video
Visualization is good but stylet and tube will not pass	<ol style="list-style-type: none"> 1. Consider bougie first followed by tube. 2. Consider smaller tube. 	<ol style="list-style-type: none"> 1. Video 22.2 2. Video 22.3
Visualization is good, but stylet and tube tip are through the cords but will not advance any further	<ol style="list-style-type: none"> 1. Consider right turn (clockwise of the tube) and attempt to advance. 2. Consider maintaining tube position, retracting stylet 5 cm, and attempt to advance the tube. 3. Consider maintaining tube position, replace stylet with a bougie, and attempt to advance the tube over the bougie. 4. Consider smaller tube. 	
Visualization is good; tube over bougie gets bevel hung up on the posterior cartilage	<ol style="list-style-type: none"> 1. Perform the “bougie left turn” maneuver, which displaces the bevel from the posterior cartilage. 	<ol style="list-style-type: none"> 1. Video 22.4 2. Video 22.5

Optimizing Biomechanics (See **Figs. 22.2A, 22.2B, and 22.23**)



FIGURE 22.22A ■ Two-Finger Laryngoscope Grip. The initial grip is with the proximal handle held between the index and long fingers and the thumb. This grip provides the best “feel” of the tissues as the blade tip is inserted into the vallecula. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.22B ■ Four-Finger Laryngoscope Grip. The addition of the ring and small fingers provides power to lift structures and visualize the glottis. The extended thumb is now placed parallel to the axis of the handle. This makes a natural extension of the forearm. (Photo contributor: Lawrence B. Stack, MD.)

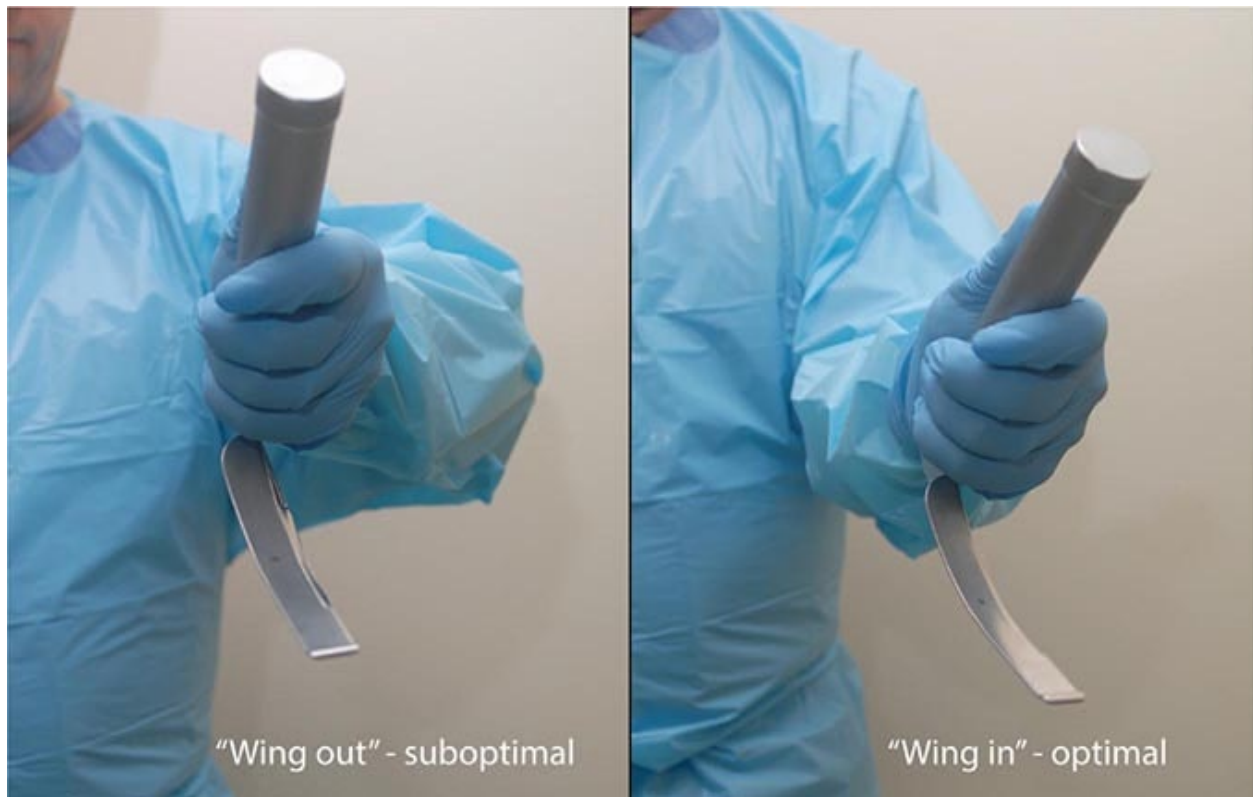


FIGURE 22.23 ■ Laryngoscope Biomechanics. Keeping the elbow close to the body requires less effort and more control during laryngoscopy. (Photo contributor: Lawrence B. Stack, MD.)

The initial grip of the laryngoscope is with the proximal handle held between the index and long fingers and thumb (see [Fig. 22.22A](#)). These digits provide the most control and “feel” of the tissues. Fine scooping movements are used to ultimately advance the blade tip into the vallecula. Once the blade tip is in the vallecula, all fingers encircle the handle and the thumb runs parallel to the axis of the handle (see [Fig. 22.22B](#)). This grip creates a natural extension of the forearm and provides fine control of the blade tip and, with the small and ring fingers, the power to lift tissues. Placing the elbow close to the body (see [Fig. 22.23](#)) requires less effort and more mechanical control than if the elbow abducted from the body. A memory aid for these concepts is “thumb up-wing in.”

Mouth Opening Techniques (See [Fig. 22.24](#))

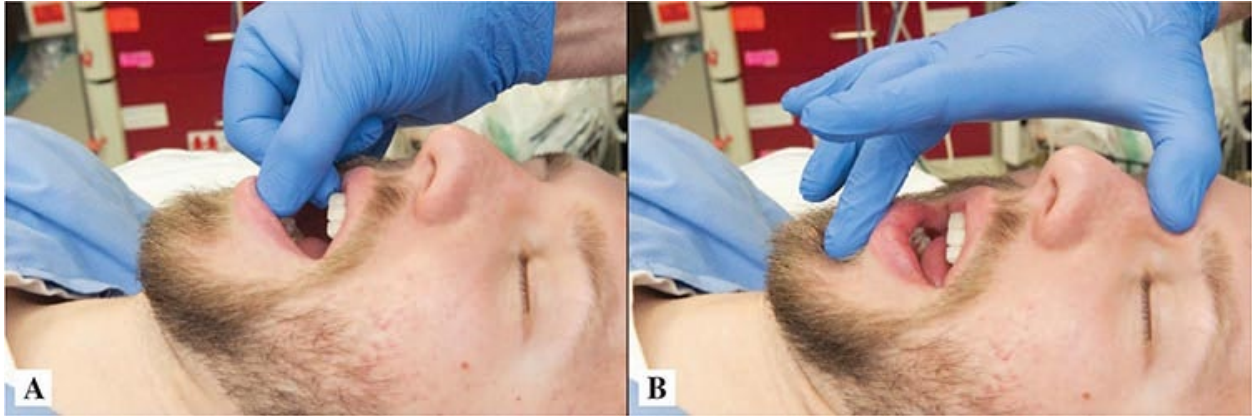


FIGURE 22.24 ■ Mouth Opening Techniques. The traditional “scissor” technique (A) and the “clean hand” technique (B) for opening the mouth during endotracheal intubation. (Photo contributor: Lawrence B. Stack, MD.)

The scissor technique places the laryngoscopist’s right thumb on the occlusive surface of the patient’s lateral mandibular teeth or gum and long or index finger on the maxillary teeth or gum (see [Fig. 22.24A](#)). A scissoring motion opens the mouth, allowing blade insertion. The fingers are removed once the blade is adequately inserted. An alternative technique is to place the laryngoscopist’s right thumb on the nasal bridge and long finger between the mental protuberance and alveolar margin. The thumb and long finger are moved away from each other, causing the mouth to open (see [Fig. 22.24B](#)).

Blade Insertion

The blade is initially inserted just to the right of midline with the handle pointed toward the patient’s feet. As the tip of the blade advances downward along the tongue base and into vallecula, the tongue is swept slightly to the left to increase the amount of workspace. When fully inserted into the vallecula, the angle of the handle is now approximately 40 degrees from horizontal with lifting forces directed upward and forward.

Optimal Epiglottoscopy

The key to using the laryngoscope to optimally visualize the glottic opening is to first visualize and control the epiglottis, a relatively fixed anterior structure. Pulmonary secretions, blood, and/or vomitus that pools in the posterior pharynx may obscure the posterior laryngeal structures, dependent with gravity. The epiglottis itself may be camouflaged in this pool of muck (see [Fig. 22.25](#)). Once in the mouth, the laryngoscope blade is advanced with small scooping motions until the tip is in the vallecula. The epiglottis must be visualized to and

controlled with the blade tip. Failure to do so risks inserting the laryngoscope blade too deeply and often results in displacing the larynx anteriorly to expose the esophagus, which, as a consequence of anterior-ward tension on the laryngoscope blade, may then look like a glottic opening begging for a tube. By carefully controlling the epiglottis, the laryngoscopist will be able to locate important airway landmarks. Be careful to displace the tongue to the left side as you insert the blade. If the bulk of the tongue wraps around the blade, it can both impair your view and impede tube delivery.

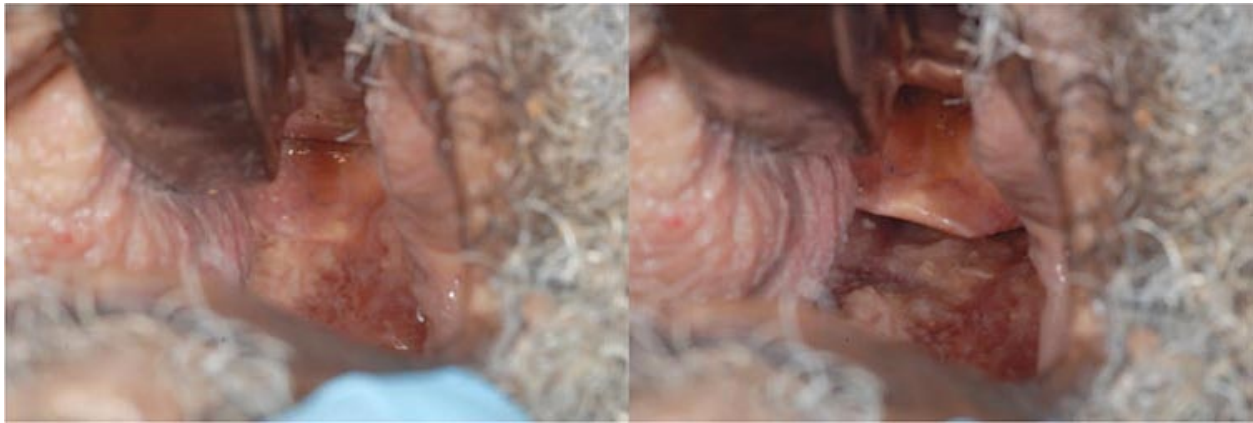


FIGURE 22.25 ■ Epiglottic Camouflage. Vomitus, blood, and pulmonary secretions may pool in the posterior pharynx and obscure the gravity-dependent epiglottis. Elevating anterior laryngeal structures will expose the epiglottis. (Photo contributor: Lawrence B. Stack, MD.)

The Cormack-Lehane classification system (see [Fig. 22.26](#)) is used to describe the structures seen during direct laryngoscopy. It is helpful for trainees during emergent intubations to quickly describe what they see during direct laryngoscopy and let assistants know how they might better assist the trainee. For example, a grade 3 view may prompt an assistant to give some laryngeal manipulation or get a bougie ready for use. In a grade 1 view, most of the glottis is seen; in grade 2 view, the posterior glottis is seen; in grade 3 view, none of the glottis is seen; and in grade 4 view, not even the epiglottis is seen.



FIGURE 22.26 ■ Cormack-Lehane Classification. In a grade 1 view, most of the glottis is seen; in grade 2 view, the posterior glottis is seen; in grade 3 view, none of the glottis is seen; and in grade 4 view, not even the epiglottis is seen. (Photo contributor: Lawrence B. Stack, MD.)

Progressive visualization of laryngeal structures is predictable:

1. Uvula and posterior pharynx (see [Fig. 22.27A](#))
2. Epiglottis (see [Fig. 22.27B](#))
3. Posterior arytenoids cartilages and interarytenoid notch (see [Fig. 22.27C](#))
4. Glottic opening (see [Fig. 22.27D](#))
5. Vocal cords (see [Fig. 22.27E](#))
6. Esophagus (see [Fig. 22.27F](#))

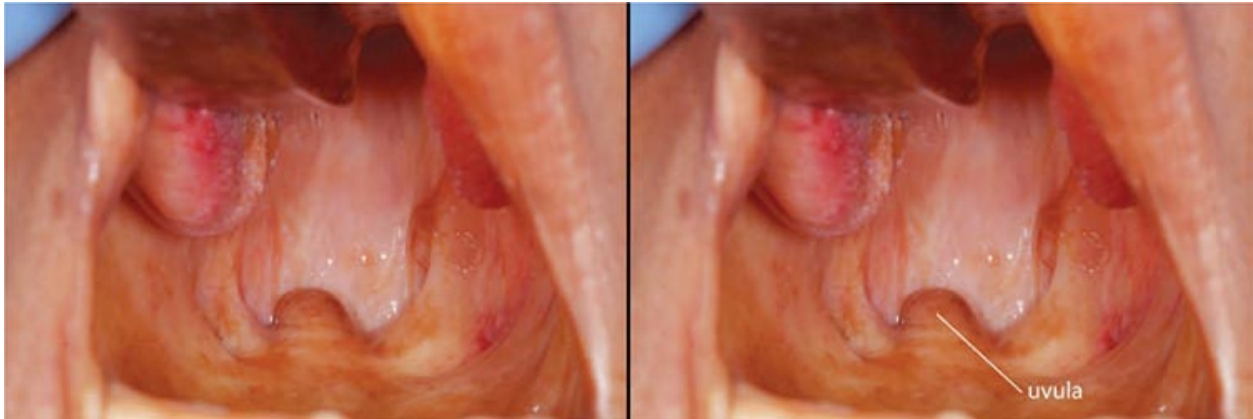


FIGURE 22.27A ■ Progressive Visualization—Uvula. The uvula and posterior pharynx are the first structures visualized during correct laryngoscopic technique. The uvula points to the way to get to the epiglottis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.27B ■ Progressive Visualization—Epiglottis. The key to first-pass intubation is finding and identifying the epiglottis. Following the epiglottis will take one to the glottic opening. (Photo contributor: Lawrence B. Stack, MD.)

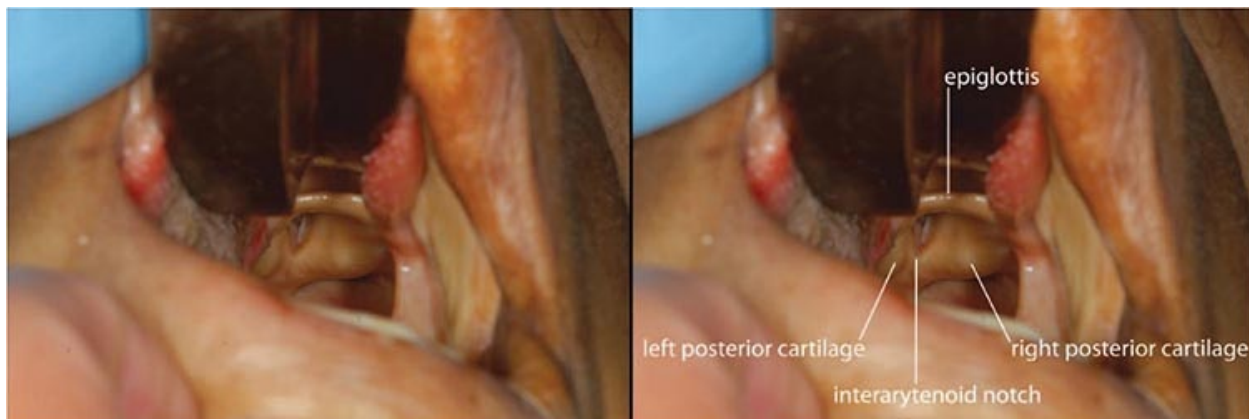


FIGURE 22.27C ■ Progressive Visualization—Interarytenoid Notch. The interarytenoid notch is a vertical cleft between the posterior cartilages. Above the interarytenoid notch lies the glottic opening. Below the interarytenoid notch is the esophagus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.27D ■ Progressive Visualization—Glottic Opening. The posterior glottic opening is seen before the vocal cords are visualized in this cadaver. Typically, cords will be seen prior to the glottic opening. (Photo contributor: Lawrence B. Stack, MD.)

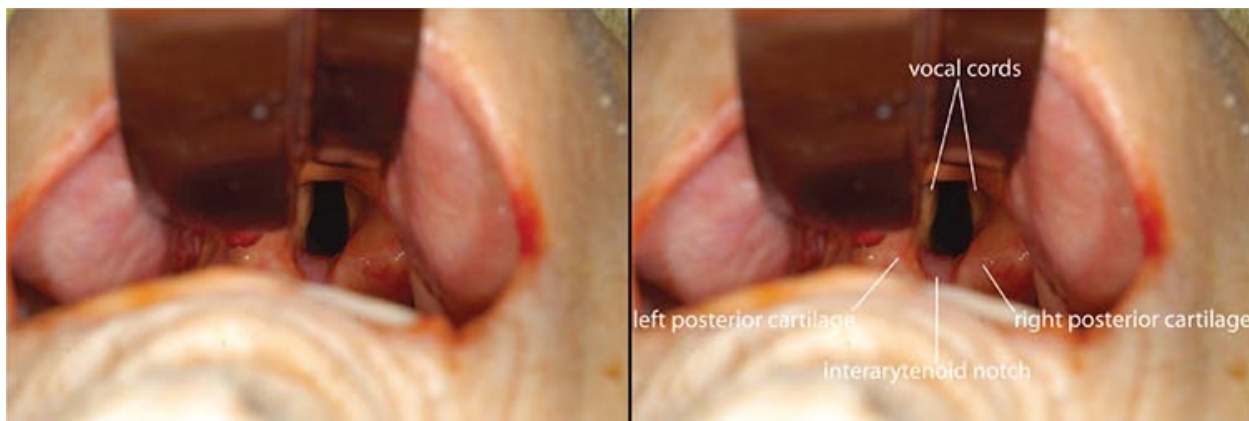


FIGURE 22.27E ■ Progressive Visualization During Laryngoscopy—Vocal Cords. The vocal cords have a distinct white appearance. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.27F ■ Progressive Visualization During Laryngoscopy—Esophagus. The esophagus lies directly below the interarytenoid notch. (Photo contributor: Lawrence B. Stack, MD.)

Bimanual Laryngoscopy (See Fig. 22.28)

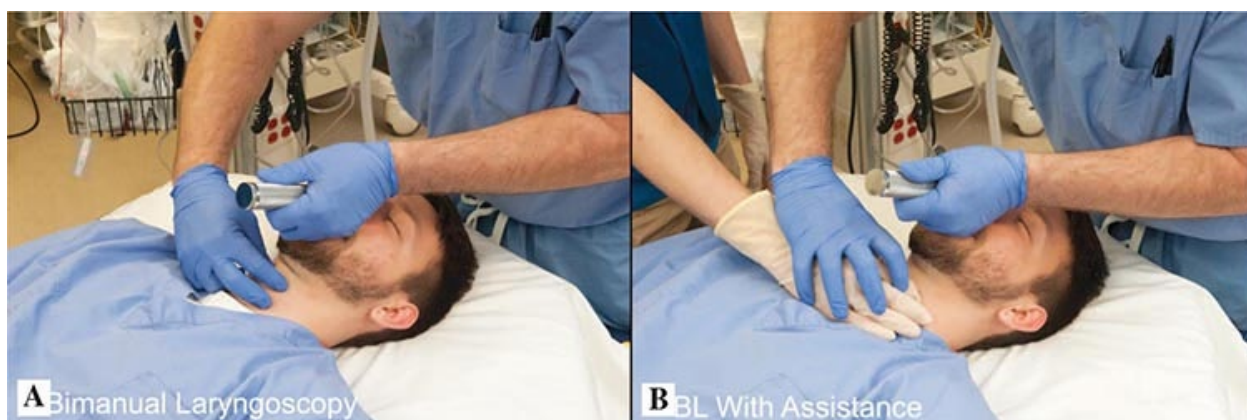


FIGURE 22.28 ■ Bimanual Laryngoscopy. Laryngoscopist uses the right hand to manipulate laryngeal structures for optimal visualization during laryngoscopy. Once optimal position is identified, an assistant maintains it during intubation. (Photo contributor: Lawrence B. Stack, MD.)

Even with well-performed laryngoscopy, adequate visualization of the laryngeal structures may be difficult. Bimanual laryngoscopy (external laryngeal manipulation by the operator), where the laryngoscopist performs laryngoscopy while simultaneously manipulating the external larynx, facilitates optimal visualization (see Fig. 22.28A). This eliminates any delay or miscommunication between an assistant and laryngoscopist. Once optimal position is found, an assistant can maintain that position. Alternatively, an assistant's hand, placed on the laryngeal structures, guided by the laryngoscopist's hand can maintain optimal position after the laryngoscopist removes his hand from the assistant's hand (see Fig. 22.28B).

Lip Commissure Retraction (See Figs. 22.29 and 22.30)

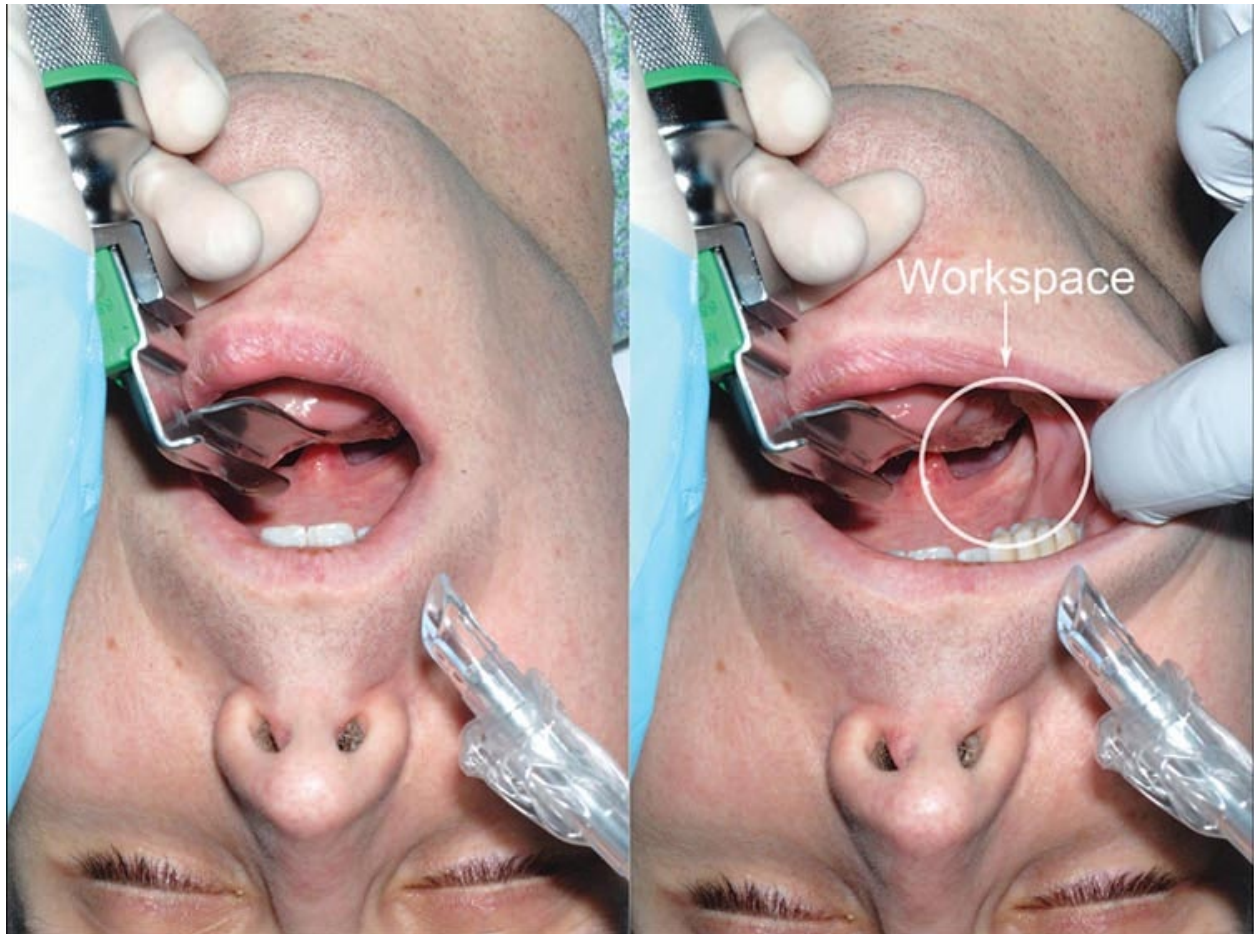


FIGURE 22.29 ■ Commissure Retraction. An assistant retracts the right commissure to create a generous “workspace” to visualize the glottis and deliver the endotracheal tube. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.30 ■ Pull on Commissure—Push on Maxilla. When pulling on the commissure with the index finger, simultaneous pushing on the maxilla with the thumb will minimize head movement during intubation. (Photo contributor: Lawrence B. Stack, MD.)

The lip commissure is the junction of the upper and lower lips. Lateral retraction of the patient's right lip commissure by an assistant facilitates visualization of oral structures and insertion of the ETT into the oral cavity. This creates an optimal workspace (see [Fig. 22.29](#)) for tube delivery. After glottis visualization is achieved, an assistant places their right index finger in the patient's right commissure and thumb against the maxilla. The index finger retracts the commissure laterally while applying an opposing force with the thumb to avoid excessive movement of the head (see [Fig. 22.30](#)). A memory aid of this concept is "pull out commissure, push on maxilla," or "POC-POM." Retraction of the lip commissure aids in keeping the ETT from blocking the view of the glottic opening during advancement of the ETT. Using the retracted lip commissure as a

fulcrum, or “toggle,” gives optimal control of the ETT tip, especially when the stylet is in the “straight to cuff” shape. Retraction may also facilitate intubation by allowing clockwise rotation of the tube if the ETT tip becomes hung up on the proximal tracheal cartilages. A memory aid of this concept is “right on rings,” or rotate the tube to the right or clockwise if it hangs up on the tracheal rings.

Endotracheal Tube Handling (See Figs. 22.31 and 22.32)



FIGURE 22.31 ■ Correct ETT Grip. Using the thumb, index, and long fingers to grip the mid-endotracheal tube like a “throwing dart” will provide the most precise control during tube deliver. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.32 ■ Incorrect ETT Grip. These ETT grips will provide a powerful force on the tube but will lack precise handling of the tube, critical to tube delivery. (Photo contributor: Lawrence B. Stack, MD.)

The thumb, index, and long fingers provide the fine dexterity for the hand and should be used to control the tube during endotracheal intubation. The ETT should be held like a “throwing dart” between the thumb, index, and long fingers midway along the tube for precise control during the procedure. A trained assistant should hand the tube to the laryngoscopist in this manner.

Endotracheal Tube Delivery (See Fig. 22.33)



FIGURE 22.33 ■ Tube Delivery. Obtain the best glottic view. Introduce the ETT at the 3 o'clock position of the mouth. Advance the tube posteriorly in a manner not to obstruct the view of the “target.” Advance the tube through the glottis. (Photo contributor: Lawrence B. Stack, MD.)

Obtain the best glottic view and have an assistant provide right lip commissure retraction. Introduce the ETT at the 3 o'clock position of the mouth. Advance the tube *posteriorly* in a manner that will not obstruct the direct view of the “target.” Advance the tube through the glottis. The last thing you should see is the tube going through cords. If resistance is met, it is most likely due to the bevel being caught on a tracheal ring. Turn the tube to the right or clockwise and attempt to advance the tube. Advance the tube to 22 cm at the lip, inflate the cuff, and confirm placement. Secure the tube in place ([Table 22.2](#)).

TABLE 22.2 ■ ENDOTRACHEAL TUBE (ETT) DELIVERY STEPS

- Obtain best possible glottic view.
- Introduce ETT at 3 o'clock position of the mouth.
- Advance ETT posteriorly to avoid obstruction of “target” view.
- Advance ETT through glottis (last thing to see is ETT through cords).
- If resistance is met (bevel against rings), rotate tube clockwise, then advance.
- Inflate cuff.
- Confirm tube placement (end-tidal carbon dioxide, breath sounds, oxygen saturation monitor).
- Secure tube.

Pearls

1. The key to glottic visualization during direct or video laryngoscopy is identification and control of the epiglottis.
2. The glottic opening lies between the epiglottis and interarytenoid notch. Identification and passing the tube between these structures will improve first-pass success.
3. While most intubations are performed without difficulty, if a difficult airway is anticipated and there is time to prepare, optimize the patient’s ear-to-sternal notch position.
4. Approximately 250 mL/min of oxygen will move from the alveoli into the blood during apnea, which may extend the period of adequate arterial oxygenation during intubation.
5. Anticipate and have a strategy for tube delivery difficulties (see [Table 22.1](#)).

OROPHARYNX AND AIRWAY DECONTAMINATION STRATEGIES

Clinical Summary

Hematemesis (see Video 22.6), emesis (see Videos 22.7 and 22.8), and

oropharyngeal bleeding (see Videos 22.9 and 22.10) during intubation increase the probability of aspiration and its complications. Having a strategy for active vomiting and bleeding during intubations is prudent to prevent or lessen the volume of aspirated vomitus.

Possible Strategies

1. Place the patient in reverse Trendelenburg position to use gravity to prevent further vomiting (see [Fig. 22.34](#)).



FIGURE 22.34 ■ Reverse Trendelenburg. Continued soilage may benefit from this feet-down position. It may improve the ear-to-sternal notch positioning also. (Photo contributor: Lawrence B. Stack, MD.)

2. Perform deliberate esophageal intubation to direct emesis away from the oral cavity (see [Fig. 22.35](#)).

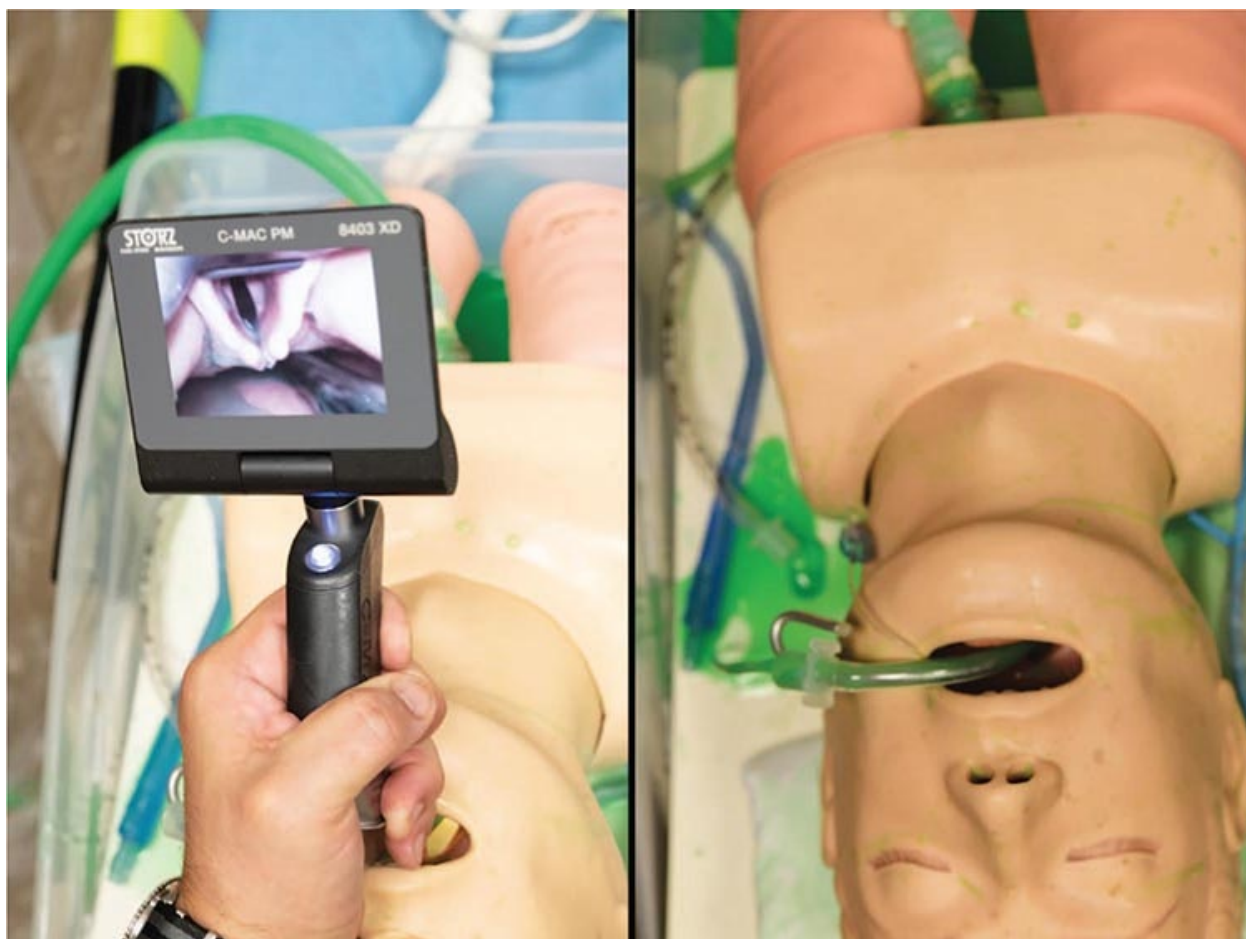


FIGURE 22.35 ■ Deliberate Esophageal Intubation. This strategy is used with continuous soilage of the oropharynx. Soilage is directed away from the working space. (Photo contributor: Lawrence B. Stack, MD.)

3. Use a large-bore DuCanto suction catheter rather than a Yankauer (see [Fig. 22.36](#)).

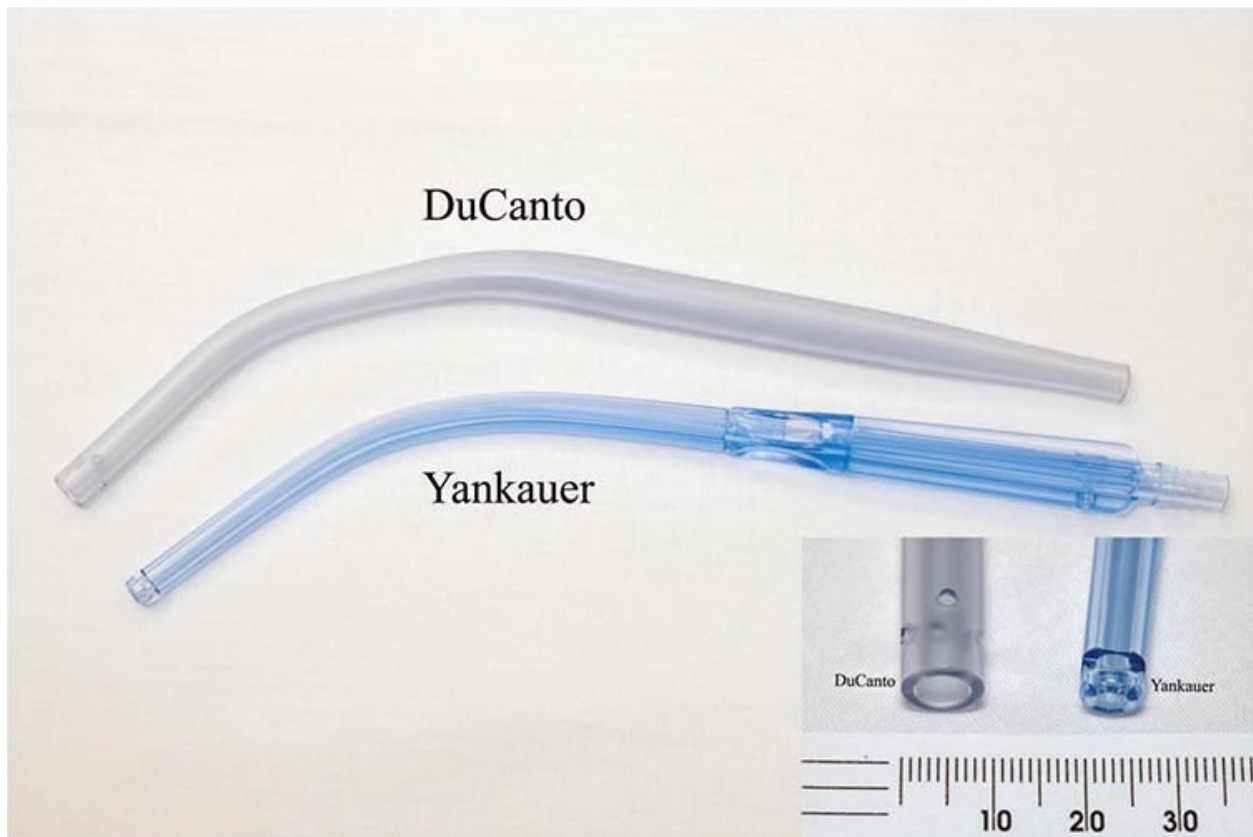


FIGURE 22.36 ■ DuCanto and Yankauer Suction Catheters. The DuCanto catheter offers a 6.6-mm internal diameter (ID) at the tip compared to the 3-mm ID tip of the Yankauer. (Photo contributor: Lawrence B. Stack, MD.)

4. Use the suction-assisted laryngoscopy and airway decontamination (SALAD) technique.
 - A. Lead with suction during laryngoscopy (see [Fig. 22.37](#)).

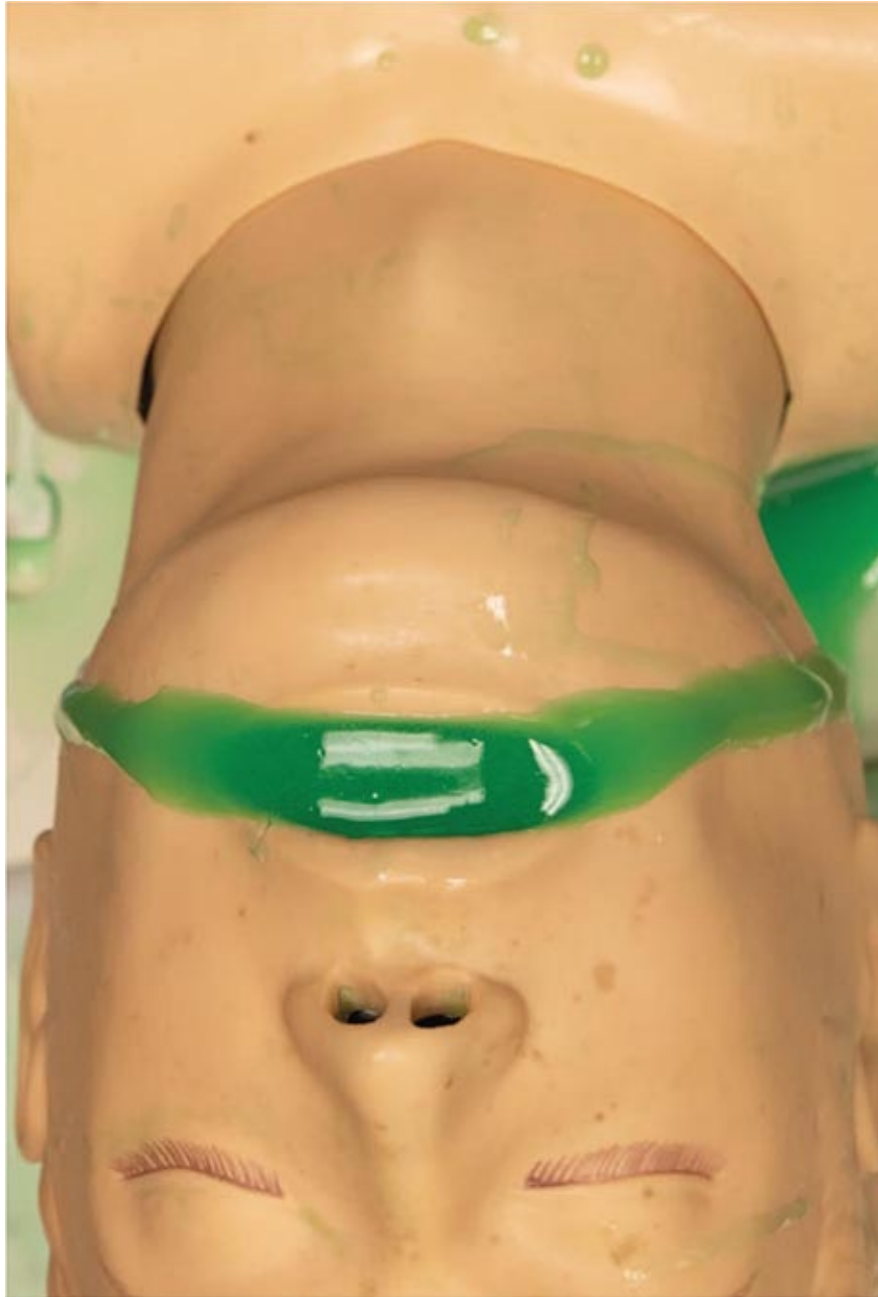


FIGURE 22.37 ■ Lead with Suction. Real or simulated airway soil-age should prompt the laryngoscopist to lead with suction. (Photo contributor: Lawrence B. Stack, MD.)

B. Decontaminate the oropharynx (see [Fig. 22.38](#)).



FIGURE 22.38 ■ Decontaminate the Oropharynx. Do this prior to entering the oral cavity with a video laryngoscope. (Photo contributor: Lawrence B. Stack, MD.)

C. Follow the suction with direct laryngoscopy or video laryngoscopy (see [Fig. 22.39](#)).



FIGURE 22.39 ■ Follow the Suction with Video Laryngoscopy or Direct Laryngoscopy. Be careful not to get ahead of the suction. Have a mechanism to clean the light and lens of the video laryngoscope if needed. (Photo contributor: Lawrence B. Stack, MD.)

D. Visualize the epiglottis and vallecula (see [Fig. 22.40](#)).



FIGURE 22.40 ■ Visualize the Epiglottis and Vallecula. Decontaminate the laryngeal structures once visualized. (Photo contributor: Lawrence B. Stack, MD.)

- E. Decontaminate the larynx.
- F. If repeated soilage, place the suction catheter to the left of the laryngoscope for continuous decontamination during tube passage (see [Fig. 22.41](#)).



FIGURE 22.41 ■ Suction Catheter to the Left. If soilage continues, place the catheter to the left of the laryngoscope and toward the esophagus. This is known as the SALAD Park Technique. (Photo contributor: Lawrence B. Stack, MD.)

- G. Place ETT and remove stylet.
- H. Suction the ETT (see [Fig. 22.42](#)).



FIGURE 22.42 ■ Suction the Endotracheal Tube. Suction the ETT with flexible suction catheter or a meconium aspiration setup when aspiration of blood or soilage is suspected. (Photo contributor: Lawrence B. Stack, MD.)

I. Begin ventilation.

BOUGIE-ASSISTED INTUBATION

Technique

The Eschmann Stylet, also known as a “gum elastic bougie” or simply “bougie,” is a 60-cm long, flexible introducer that is designed to assist intubation of the

anterior larynx, especially those with an “epiglottis-only” view. The tip of the bougie has an anterior fixed flexion with an angle of 40 degrees to facilitate entering an anterior glottic opening (see [Fig. 22.43](#)). Using a laryngoscope, one obtains the best view possible of the glottic opening. In the case of limited glottic view (see [Fig. 22.44](#)), the bougie is inserted such that the tip is introduced just under the epiglottis and probes for the glottic opening (see [Fig. 22.45](#)). The intubator should feel a tactile “pop” as the bougie enters the trachea, and he or she may also have a tactile sensation of “speed bumps” as the bougie is advanced and tracks across the tracheal cartilaginous rings (see [Fig. 22.46](#)). However, the more sensitive indicator of tracheal bougie position is the resistance encountered as the tip abuts against the carina, at approximately 27 to 30 cm. Should the bougie be inserted into the esophagus, no such endpoint is encountered.



FIGURE 22.43 ■ Bougie Tip Shape. The bougie tip has a 30- to 35-degree bend to help facilitate entering the glottic opening when the glottis is not adequately visualized. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.44 ■ Epiglottis-Only View. In this view, despite optimal laryngoscopy, the epiglottis may be all that is seen. The bougie may be a reasonable alternative in this situation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.45 ■ Bougie Under the Epiglottis. Here the bougie is hugging the underside of the epiglottis. Tracheal rings can be felt as “tactile speed bumps” confirming correct placement. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.46 ■ Passing the Bougie. The two-person technique for using the bougie. Standard direct laryngoscopy is preformed, and the bougie is used like a stylet, but hugging the underside of the epiglottis. (Photo contributor: Lawrence B. Stack, MD.)

While continuing to hold anterior traction with the laryngoscope, the laryngoscopist should direct an assistant to thread the ETT over the bougie (see [Fig. 22.47](#)) again while maintaining anterior traction; the intubator should then advance the ETT over the bougie to the appropriate insertion depth. Resistance to tube advancement may indicate “arytenoid arrest” (see [Fig. 22.48](#)) and can be remedied by rotating the tube counter clockwise 90 degrees (see [Fig. 22.49](#)) followed by attempts to advance the ETT tip past the arytenoid cartilages.



FIGURE 22.47 ■ Tube Over the Bougie. An assistant places the bougie over the tube while direct laryngoscopy is maintained. (Photo contributor: Lawrence B. Stack, MD.)

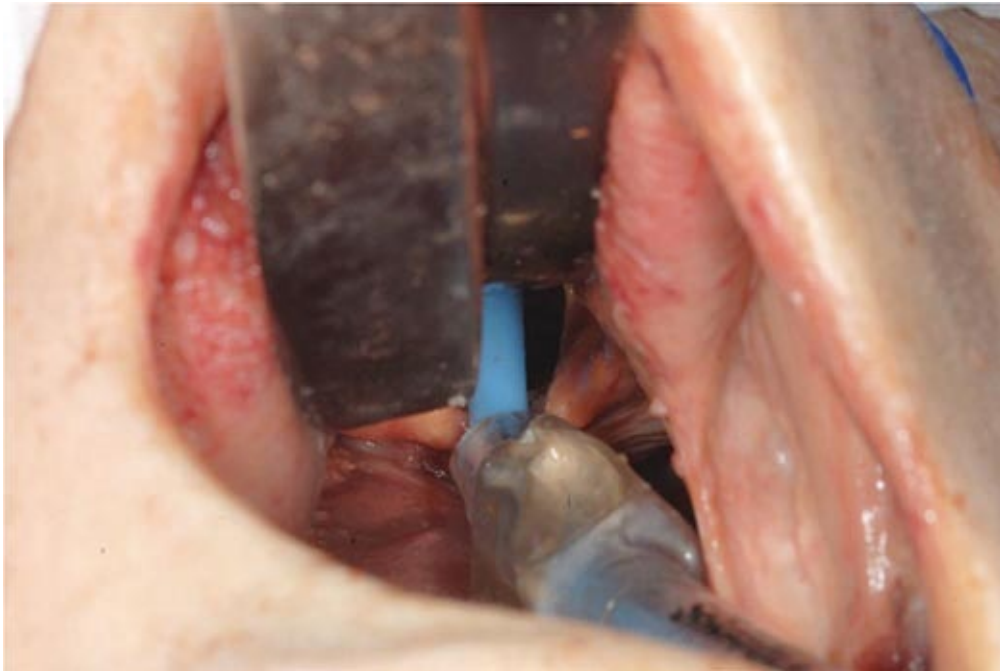


FIGURE 22.48 ■ Tube Advancement Stall. Stalling of tube advancement while using the bougie is most likely due to catching the right arytenoids cartilage with the bevel of the ETT. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.49 ■ Arytenoid “Escape.” Turning the tube counterclockwise, as shown here, will release the bevel of the ETT from the arytenoid cartilage and allow the tube to advance. (Photo contributor: Lawrence B. Stack, MD.)

A one-handed technique can be employed in which the ETT is preloaded onto the bougie, which is then curled in the right hand with approximately 20 cm of bougie protruding through the distal end (see [Fig. 22.50](#)). The laryngoscopist performs laryngoscopy as before, maintains anterior traction on the mandible, and inserts the bougie through the glottic opening as above. The laryngoscopist then uncoils the ETT/bougie and advances the pair until resistance of the carina is met, confirming intratracheal position (see [Fig. 22.51](#)). The ETT is then advanced to the proper depth and the bougie is removed.



FIGURE 22.50 ■ One-Handed Bougie Configurations. Holding the bougie in one of these configurations will allow one person to pass the bougie. Some of the tactile “feel” may be lost in the one-handed bougie technique. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.51 ■ One-Handed Bougie Technique. Passing the bougie using the one-handed technique. See text for details. (Photo contributor: Lawrence B. Stack, MD.)

Use of the bougie does not have to be limited to the anticipated difficult airway. In fact, to gain proficiency, we recommend that practitioners use it as their routine stylet, as it has come to be used in the United Kingdom. The practitioner will then be better prepared for the unanticipated anterior larynx/limited view airway.

Pearls

1. The bougie is an excellent adjunct in the limited view or epiglottis-only view airway.
2. Bougie-assisted intubations is best deployed as a two-person technique
3. Stall of ETT advancement when using the bougie is most frequently due to catching the bevel on the right arytenoid cartilage and can be relieved by rotating the tube counterclockwise.

VIDEO-ASSISTED INTUBATION—STORZ C-MAC

Equipment

The Storz C-MAC video laryngoscope system consists of stainless steel, reusable blades that house a CMOS camera chip and high-output LED light, an interchangeable powered coupling, video screen, and mobile tower (see [Fig. 22.52](#)). Available blades are similar in size and shape to the Mac 2, Mac 3, Mac 4, Miller 0, Miller 1, and a D-blade designed for patients with difficult airway anatomy. The near-standard shape of the blade allows direct laryngoscopy for glottic visualization as well as video laryngoscopy.



FIGURE 22.52 ■ Storz C-MAC Tower. Storz video laryngo-scope system including tower and adult blades. (Photo contributor: Lawrence B. Stack, MD.)

Technique (See Fig. 22.53)

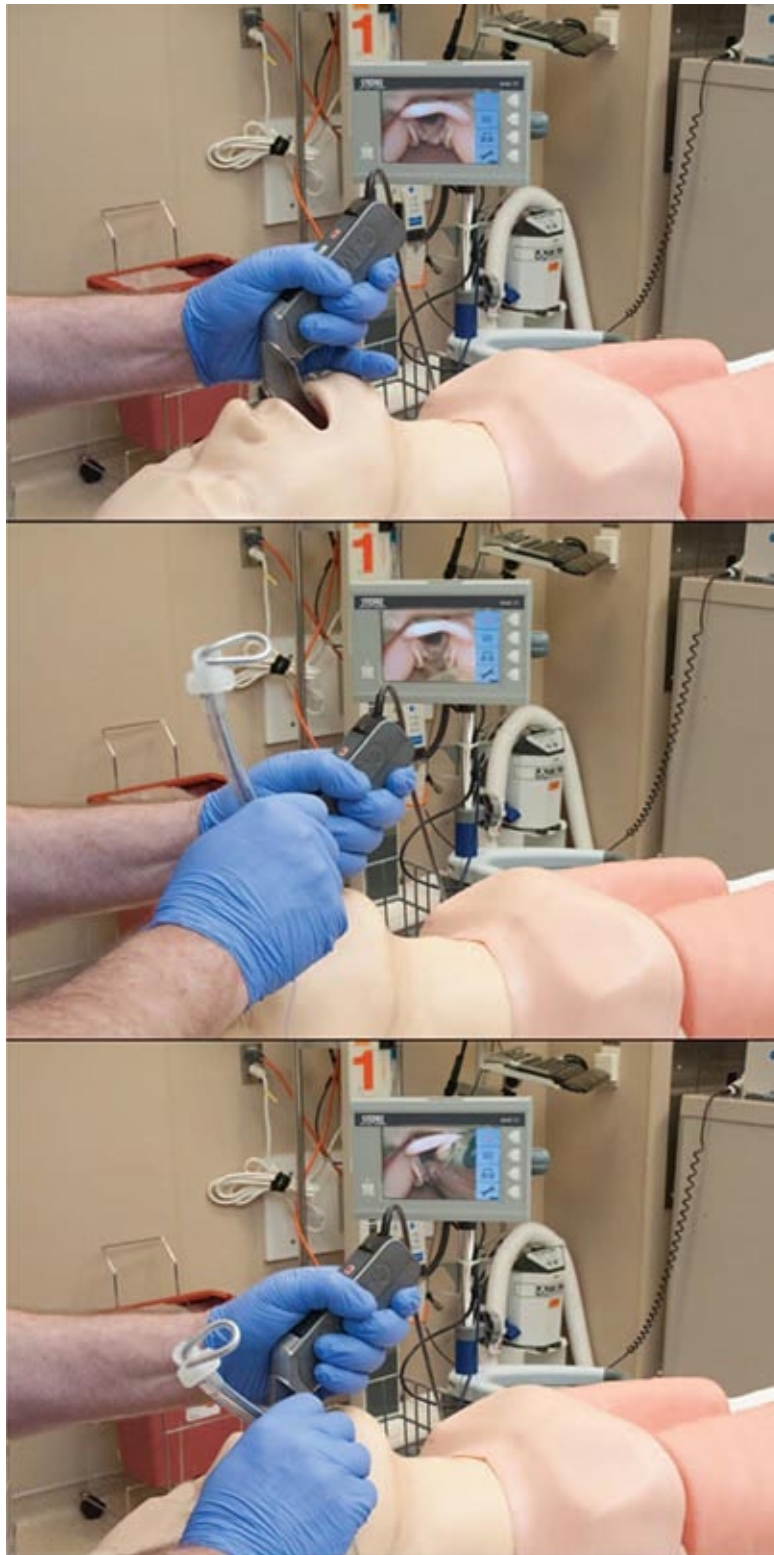


FIGURE 22.53 ■ Storz C-MAC In Use. The Storz C-MAC in action demonstrating progressive images of passage of an ETT. (Photo contributor: Lawrence B. Stack, MD.)

The blade connected to the power coupling should be handled like a standard

laryngoscope blade. It should be inserted just to the right of midline of the tongue with the handle pointing toward the feet. As the tip of the blade advances downward along the tongue base and into vallecula, the tongue is swept slightly to the left to increase the amount of workspace. When fully inserted into the vallecula, the angle of the handle is now approximately 40 degrees from horizontal with lifting forces directed upward and forward. At any time, the laryngoscopist can switch from a standard direct laryngoscopy to a video laryngoscopy; however, once changed to video laryngoscopy, it is best not to switch back and forth from video to direct laryngoscopy.

VIDEO-ASSISTED INTUBATION—McGRATH MAC EDL (FIGS. 22.54-22.57)



FIGURE 22.54 ■ McGrath MAC Enhanced Direct Laryngoscope. Compact video laryngoscope system shown here with a Mac 3 disposable blade. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.55 ■ McGrath MAC EDL. Cord visualization using the McGrath MAC, which can also be used as a direct laryngoscope. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.56 ■ McGrath MAC EDL. Vocal cord visualization and ETT delivery are demonstrated here. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.57A ■ King Vision Video Laryngoscope. The King Vision laryngoscope is a disposable, battery-powered video laryngoscope with robust chip technology and a high blade angulation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.57B ■ i-View Video Laryngoscope. The i-View is a single use, fully disposable video laryngoscope available in standard geometry Macintosh design. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.57C ■ Storz C-MAC PM. The Storz C-MAC Pocket Monitor is a portable video laryngoscope that offers both disposable and non-disposable versions combined with high-contrast imaging and compact design. The device can be configured for both adult and pediatric use. (Photo contributor: Lawrence B. Stack, MD.)

The McGrath MAC enhanced direct laryngoscope is a combination direct and video laryngoscope in a compact, battery-powered device that uses disposable standard geometry curved Macintosh blades and has an adjustable LED screen. The King Vision, the iView, and the Storz C-MAC PM (pocket monitor) are additional versions of portable handheld video laryngoscopes currently available commercially.

Technique

The disposable plastic Macintosh blade is placed onto the McGrath MAC EDL, and the device is powered on. The blade is inserted just to the right of midline of the tongue with the handle pointing toward the feet. As the tip of the blade advances downward along the tongue base and into vallecula, the tongue is swept slightly to the left to increase the amount of workspace. When fully inserted into the vallecula, the angle of the handle is now approximately 40 degrees from horizontal with lifting forces directed upward and forward. At any time, the laryngoscopist can switch from a standard direct laryngoscopy to a video laryngoscopy; however, once changed to video laryngoscopy, it is best not to switch back and forth from video to direct laryngoscopy.

LARYNGEAL MASK AIRWAY (LMA)

The LMA was originally designed to facilitate ventilation during anesthesia for short operating room procedures. It has been shown to provide improved ventilation in cardiac arrest and failed airway cases and has a shallow learning curve, promoting its use by relative novices.

Equipment

The LMA consists of a short curved tube connected to a small mask with inflatable cuff. The shape promotes blind insertion with an endpoint detected as resistance as the leading edge of the cuff just enters and obstructs the esophageal inlet. The inflated cuff then seals around the laryngeal inlet. The distal “mask” incorporates a small grate to prevent prolapse of the epiglottis within the mask. The LMA has several configurations, including a disposable model, the LMA Unique (see [Figs. 22.58](#) and [22.59](#)), and a model that facilitates blind oral intubation, the intubating LMA, or I-LMA (see [Fig. 22.64](#) in next section). LMAs are available in three adult sizes: #3 (30-50 kg), #4 (50-70 kg), and #5 (>70 kg). They are also available in pediatric sizes from size 0 to 2.5 for infants through toddlers.



FIGURE 22.58 ■ LMA Unique. Adult sizes 3, 4, and 5 and pediatric size 2.5 are seen here. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.59 ■ LMA Unique. The laryngeal surface of the LMA Unique with inflated cuff. Note the “grate,” which prevents epiglottic prolapse into the mask. (Photo contributor: Lawrence B. Stack, MD.)

Technique

The LMA is inserted into the mouth and held against the hard palate while being advanced into the hypopharynx (see [Fig. 22.60](#)). The LMA is advanced until resistance is met, and the cuff is then inflated with 20 to 40 mL of air to effect a seal (see [Fig. 22.61](#)). The mask should be lubricated prior to insertion.

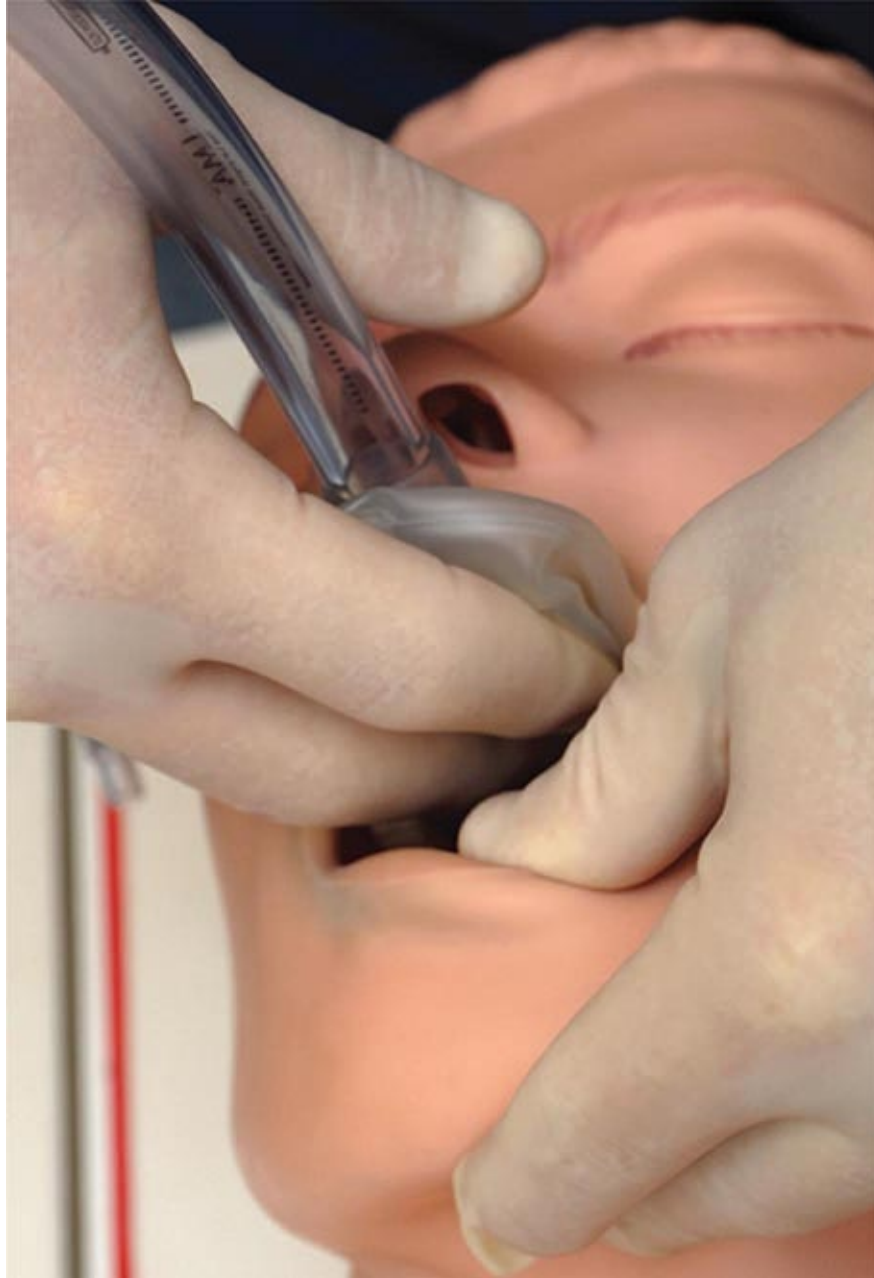


FIGURE 22.60 ■ LMA Insertion. LMA with deflated cuff is held against the hard palate during insertion by the index and third fingers. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.61 ■ Correct LMA Placement. The LMA is correctly seated when resistance is met. Slight adjustments may be needed once seated. (Photo contributor: Lawrence B. Stack, MD.)

Because the LMA aligns with the glottic opening, it is possible to intubate the trachea through the LMA with minimal interruption of ventilation. The distal grate prevents anything larger than a 6.0 internal diameter (ID) ETT through a #3 and #4, and a 7.0 ID ETT through a #5. In addition to passing a small ETT directly, it is also possible to pass a flexible fiberoptic scope or a rigid fiberoptic scope (Levitan or Shikani) through the LMA under direct visualization. One must be sure to have an ETT, sized to pass through the LMA, loaded onto the stylet prior to passage. It is recommended to rotate the ETT counterclockwise 90 degrees to orient the bevel posteriorly, rotating clockwise as the tube is passed into the glottis.

DIGITAL (TACTILE) INTUBATION (FIGS. 22.62 AND 22.63)

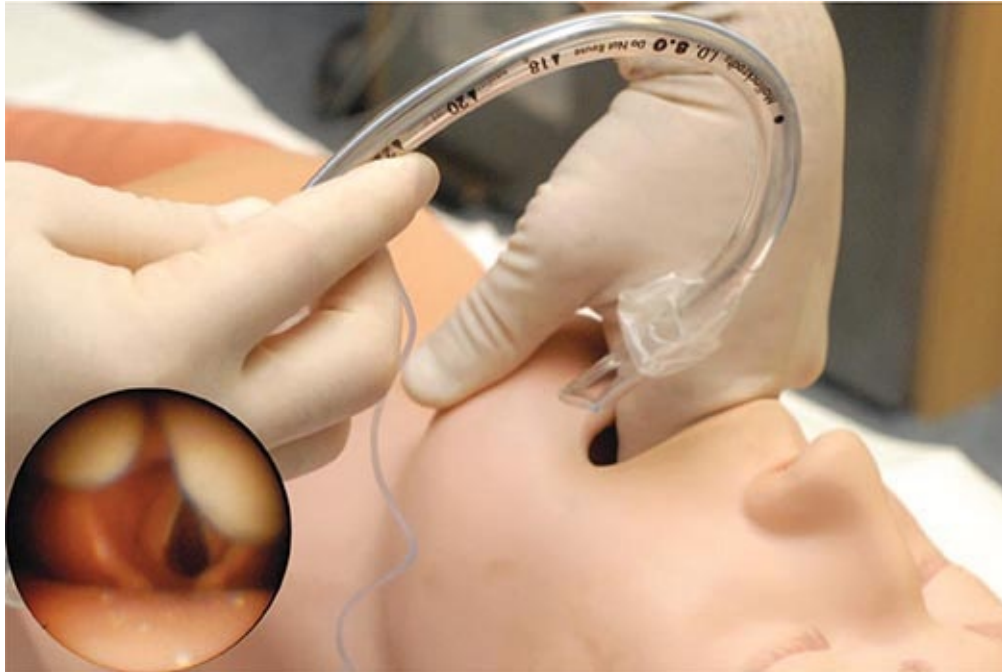


FIGURE 22.62 ■ Digital Intubation Technique. Index and third finger inserted into the mouth with “C”-shaped stylet held in left hand. The third finger holds the epiglottis anteriorly (insert). (Photo contributor: Lawrence B. Stack, MD.)

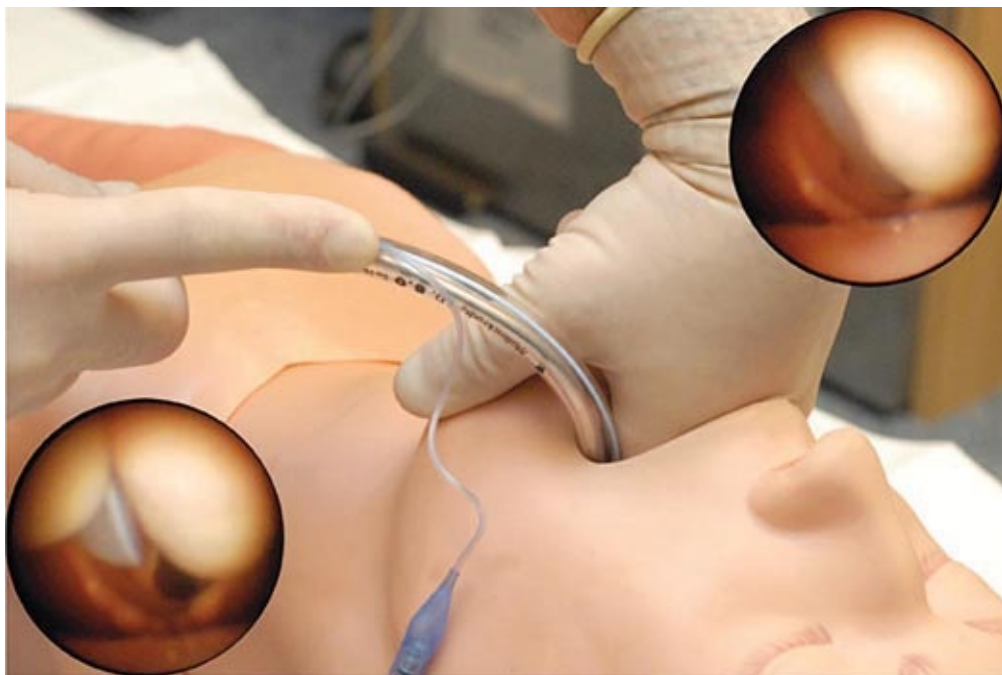


FIGURE 22.63 ■ Digital Intubation—ETT Passage. Intraoral index finger guides ETT to third finger (insert), which directs the tube into the glottic opening (insert). (Photo contributor: Lawrence B. Stack, MD.)

Digital intubation relies on tactile definition of intubation landmarks, primarily the epiglottis but often the posterior cartilages and interarytenoid notch.

Technique

Digital intubation technique is performed with the intubator facing the patient from the side. With the gloved nondominant hand, the intubator should insert the index and 3rd fingers into the patient's mouth. The intubator should next "walk" the fingers down the tongue to progressively displace the tongue anteriorly as the fingers are advanced to the epiglottis. The epiglottis will have a feel similar to the earlobe. As the 3rd finger encounters the epiglottis, the finger traps and holds the epiglottis anteriorly. The ETT, with stylet configured to an open "C," is introduced by the dominant hand into the corner of the mouth and advanced to the palmar side of the intraoral index finger. The intraoral index finger is then used to guide the ETT tip to the 3rd finger, where the ETT is slipped between the epiglottis and 3rd finger. The 3rd finger is used to keep the ETT anterior against the laryngeal surface of the epiglottis while the ETT is advanced by the dominant hand and assisted by the intraoral index finger. It is often possible to palpate the arytenoid cartilages/interarytenoid notch, in which case one need only ensure that the ETT tip remains anterior to those cartilages and is prevented from drifting posteriorly to the esophageal inlet. The posterior cartilages project cephalad, feeling like snake fangs. Correct ETT position can be confirmed by palpating the arytenoid cartilages posterior to the ETT.

INTUBATING LARYNGEAL AIRWAY (ILA) (FIGS. 22.64-22.68)



FIGURE 22.64 ■ Intubating Laryngeal Airway. Intubating LMA demonstrating an ETT through the device lumen. (Photo contributor: Lawrence B. Stack, MD.)

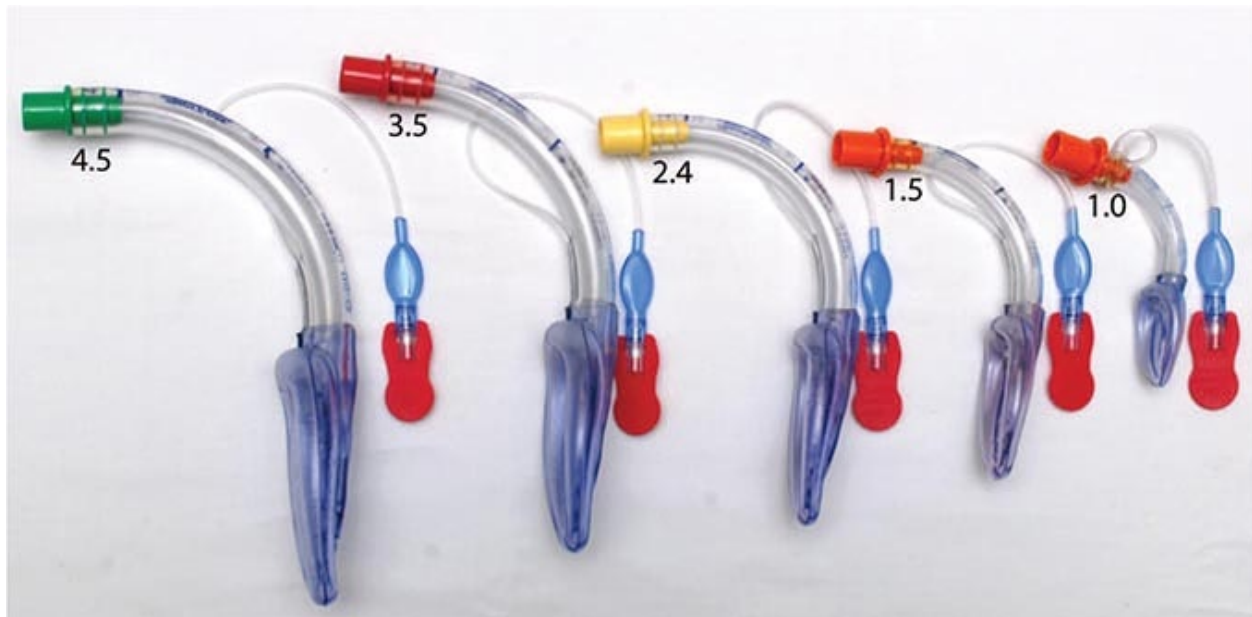


FIGURE 22.65 ■ Intubating Laryngeal Airway Sizes. ILA in five different sizes. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.66 ■ Intubating Laryngeal Airway Insertion. The ILA is inserted by displacing the tongue and jaw anteriorly and is advanced down the curvature of the oropharynx and hypopharynx. (Photo contributor: Lawrence B. Stack, MD.)

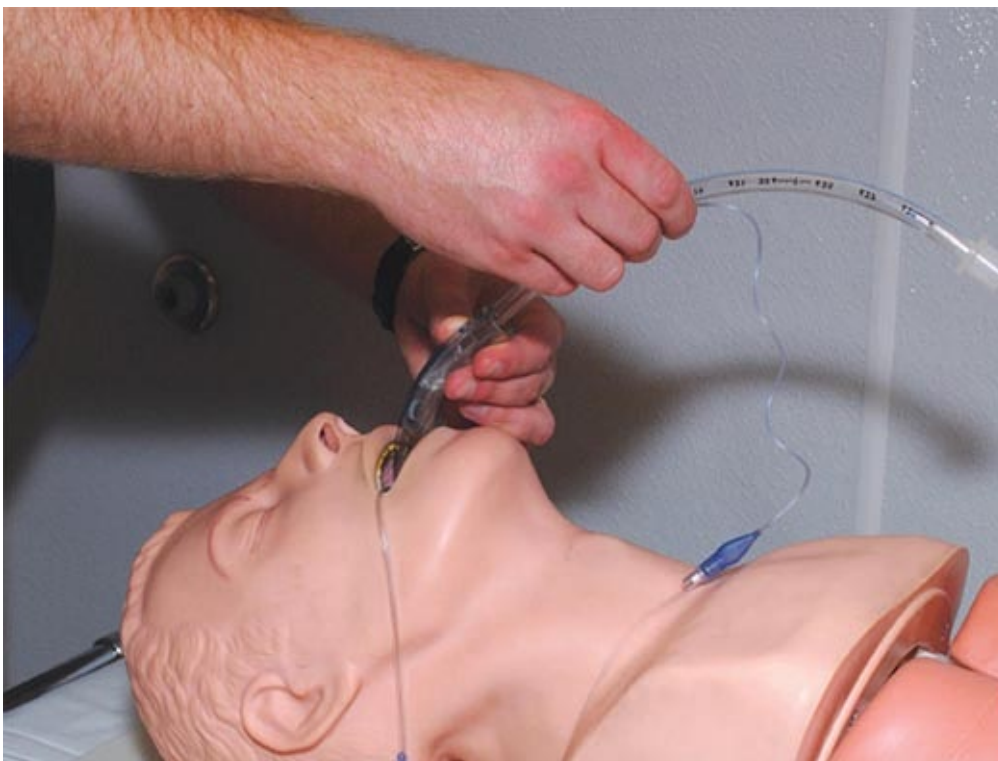


FIGURE 22.67 ■ ETT Insertion Through the Intubating Laryngeal Airway. An ETT is advanced

through the ILA. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.68 ■ Intubating Laryngeal Airway Removal. Removal of the ILA while keeping the ETT in place is accomplished by using the disposable tube stabilizer (blue). (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The reusable ILA is similar in concept to the classic LMA but has structural features that provide easier and more stable insertion with less likelihood of tube kink. In addition, it has a unique “keyhole”-like airway outlet that helps to direct an ETT to the midline at the proper glottic entry angle. Because it has a larger lumen, the ILA can accommodate standard ETT up to 8.5 mm ID. The ILA comes in five sizes—1.0 (<5 kg), 1.5 (10-20 kg), 2.4 (20-50 kg), 3.5 (50-70 kg), and 4.5 (70-100 kg)—accommodating maximum ETT sizes of 4.5, 5.5, 6.5, 7.5, and 8.5, respectively.

Technique

The mandible is displaced anteriorly with a jaw lift maneuver, bringing the tongue and epiglottis forward. The ILA is then inserted into the mouth, advancing with a downward and inward force to follow the curve of the

oropharynx/hypopharynx. The insertion endpoint is firm resistance to further insertion. The cuff is then inflated with approximately 20 mL of air or until the pilot balloon is firm. Ventilation can proceed by attaching a ventilating bag to the proximal port.

Intubation can be accomplished through the ILA in a well sedated/chemically paralyzed patient after first removing the proximal 15-mm adaptor port. A well-lubricated standard ETT can then be inserted through the ILA. This procedure can also be aided by using a lightwand or optical stylet through the ETT to facilitate optimal alignment for intubation.

Once the ETT is inserted, the ILA can be easily removed by inserting the disposable tube stabilizer/stylet.

KING LARYNGEAL TUBE (FIGS. 22.69-22.70)



FIGURE 22.69 ■ King LTS-D Airway. The King LTS-D is shown with the distal esophageal balloon and the proximal hypopharynx balloon inflated. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.70 ■ King LTS-D Airway Technique. The King LTS-D is passed down the anterior surface of the tongue while the tongue and mandible are pulled upward. The tube is advanced until resistance is met or the base of the connector is at the level of the lip or gum. The balloon is inflated with 45 to 90 mL of air depending on the size of the tube. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The King laryngeal tube (King LT) consists of an airway tube with two cuffs. The distal cuff seals the esophagus, and the proximal cuff seals the oro- and nasopharynx when inflated. Between the cuffs are multiple ventilation channels. Inflation of the cuffs occurs through one inflation line. A separate gastric drainage channel is present in the disposable model (King LTS-D).

Technique

A lubricated King LTS-D is inserted behind the tongue while performing a jaw lift, placing the patient in a sniffing position. The tube is advanced until resistance is met or until the connector base is aligned with the teeth or gums. The cuffs are inflated with 60 to 90 mL of air, depending on the size of the King LTS-D. Some “jiggling” may be required to properly seat the tube.

NASAL FLEXIBLE FIBEROPTIC INTUBATION (FIGS. 22.71 AND 22.72)



FIGURE 22.71 ■ Scope-First Method. The ETT is placed at the proximal extreme. The endoscope is then guided to the glottic opening, and the tube is then advanced over the endoscope. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.72 ■ Tube-First Method. The ETT is advanced to the hypopharynx. The endoscope is then advanced through the tube and maneuvered to the glottic opening. The tube is then advanced over the endoscope. (Photo contributor: Lawrence B. Stack, MD.)

There are two methods of nasal flexible fiberoptic technique: (1) scope first and (2) tube first.

Patient Preparation

The nares should be inspected for patency and the side deemed most hospitable to tube passage should be anesthetized and vasoconstricted, additionally, it is

helpful to confirm nasal patency by inserting a well lubricated nasal trumpet. Lidocaine jelly can be used as the lubricant to provide additional topical anesthesia. We favor the use of disposable atomizers to instill 2 ml of a mixture of tetracaine 1% phenylephrine 0.5%, which yields excellent mucosal anesthesia and vasoconstriction. Favorable intubation conditions can be achieved in about 5 minutes.

In preparation for nasal intubation, the ETT can be conformed into a circle by inserting the tip into the 15 mm proximal adaptor, and soaked in warm water to soften the tube for nasal passage.

Technique: Scope First

The largest possible ETT, preferably 7.0-mm ID, is inserted over the endoscope to the most proximal position of the endo-scope. The fiberoptic endoscope is then inserted through the selected naris. Under direct visual guidance, the endoscope is advanced along the floor of the nose, navigating under the inferior turbinate. The endoscope tip is manipulated by a combination of trigger deflection (up/down) and scope rotation (left/right) to keep the lumen in the center of the optical field. As the tube is advanced to the posterior nasopharynx, the scope tip is deflected downward to reveal the epiglottis and laryngeal inlet. The scope is advanced further, with the operator maneuvering the scope tip to keep the glottic opening in the center of the optical field. With the endoscope tip just above the cords, 2 mL of 1% lidocaine should be injected through the injection port of the fiberscope to provide vocal cord anesthesia prior to tube passage. The endoscope advancement continues until the endoscope tip is approximately 3 to 5 cm above the carina. The tube is then gently advanced over the fiberoptic sheath into the naris and to the trachea. If resistance is encountered at 14 to 16 cm, the tube may be impacting upon the right arytenoid. As discussed previously, this can be corrected by a severe counterclockwise rotation. The tube tip should be verified to be the correct distance from the carina.

If during endoscope navigation through the nose, the view becomes blurry because of adherent secretions, the tip can be cleared by gently deflecting the tip into “pink” mucosa. If one becomes lost or disoriented during scope advancement or if there is no obvious lumen through which to advance, the endo-scope should be withdrawn slightly and the tip maneuvered to provide a recognizable view. The endoscope should not be advanced if a lumen is not readily apparent.

Technique: Tube First

To avoid some of the technique difficulty inherent with nasal navigation, the ETT can be passed through the naris and advanced into the hypopharynx first, before scope insertion. The endoscopist then inserts the intubating fiberscope through the ETT and advances it to and through the glottic opening to the appropriate distance above the carina. Tube advancement proceeds as described earlier. The tube-first method may also facilitate endoscopic intubation in the event of epistaxis.

CRICOTHYROTOMY (SELDINGER TECHNIQUE) (FIGS. 22.73-22.81)



FIGURE 22.73 ■ Melker Cricothyroidotomy Kit. Components of the kit produced by Cook Inc. (www.cookmedical.com). (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.74 ■ Identify Landmarks. Palpate thyroid cartilage and cricothyroid cartilage caudally. Angle needle 30 degrees caudad to enter the trachea (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.75 ■ Bubbles During Aspiration. Aspiration of bubbles during needle advancement confirms tracheal placement. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.76 ■ Slide Catheter into Trachea. Carefully slide the catheter off the needle into the trachea. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.77 ■ Confirm Tracheal Placement. Again, aspirate bubbles from the catheter to confirm tracheal placement. (Photo contributor: Lawrence B. Stack, MD.)

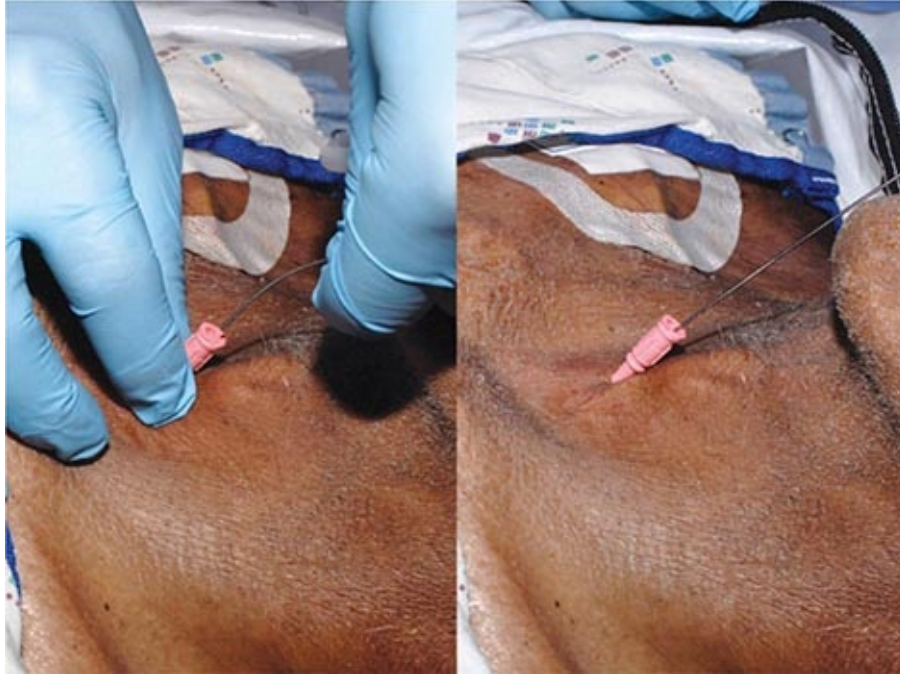


FIGURE 22.78 ■ Thread Wire. Lack of resistance during threading of Seldinger wire confirms tracheal placement. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.79 ■ Incision at Wire Entry Site. Remove catheter and make incision at the wire entry site. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.80 ■ Insert Tracheostomy Tube/Introducer. Insert both devices over the wire and into the trachea. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.81 ■ Remove Wire and Introducer. Remove these devices together as a single unit. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The Melker kit manufactured by Cook Medical includes all the equipment necessary: catheter, thin-walled needle, Seldinger wire, cuffed tracheostomy tube 5.0 mm ID, and tapered introducer.

Technique

1. Identify the thyroid cartilage and the cricothyroid membrane directly caudally.
2. Prep the neck with suitable antiseptic; anesthetize the skin overlying the cricothyroid membrane with 1% lidocaine.
3. Isolate the trachea between the thumb and 3rd finger of the sterilely gloved nondominant hand, using the index finger to palpate the cricothyroid membrane landmark. With a syringe attached, preferably containing sterile water or remaining lidocaine, insert the catheter through the cricothyroid membrane at a 30-degree angle to the horizontal, aiming toward the feet. Once through the skin, begin aspirating as the catheter is advanced. Air and bubbles entering the syringe confirm entry into the trachea.
4. Carefully slide the catheter off of the needle and into the trachea.
5. Reattach syringe and reconfirm tracheal position.
6. Thread Seldinger wire through catheter into trachea; lack of resistance to wire passage confirms correct position.
7. Remove catheter, and with the included scalpel, make a small incision at the wire-skin entry site.
8. Insert tracheostomy tube with dilator/introducer over wire into trachea. Expect significant resistance. Inability to pass dilator may indicate an inadequate skin incision.
9. Withdraw dilator/introducer and wire as one unit, inflate cuff, and initiate ventilation.
10. Secure tracheostomy tube with tracheostomy ties.

SCALPEL-FINGER-BOUGIE CRICOTHYROTOMY

Technique (See Video 22.11)

1. Cleanse and anesthetize the skin if the clinical situation allows.
2. If right hand dominant, stand on the patient's right side (see [Fig. 22.82](#)).



FIGURE 22.82 ■ Cricothyrotomy Landmarks. Stand on the side of the patient of your hand dominance. (Photo Contributor: Lawrence B. Stack, MD.)

3. With the nondominant hand, identify the thyroid cartilage and stabilize with the long finger and thumb (see [Fig. 22.83](#)).



FIGURE 22.83 ■ Stabilize the Thyroid Cartilage. Identify the thyroid and cricothyroid cartilages with the nondominant hand. (Photo contributor: Lawrence B. Stack, MD.)

4. With the nondominant index finger, identify the cricothyroid membrane and cricoid cartilage (see [Fig. 22.84](#)).



FIGURE 22.84 ■ Cricothyroid Membrane Identification. Using the index finger of the nondominant hand, palpate and identify the cricothyroid membrane. (Photo contributor: Lawrence B. Stack, MD.)

5. With the dominant hand, make a vertical (north to south) incision through the skin from the bottom of the thyroid cartilage to the top of the cricoid cartilage (see [Fig. 22.85](#)).



FIGURE 22.85 ■ Vertical (North-South) Skin Incision Over the Cricothyroid Membrane. Make a generous midline vertical incision through the skin over the cricothyroid membrane. (Photo contributor: Lawrence B. Stack, MD.)

6. With the nondominant hand index finger, through the north to south incision, reconfirm the cricothyroid membrane (see [Fig. 22.86](#)).



FIGURE 22.86 ■ Reconfirm Cricothyroid Membrane. Place non-dominant index finger into North-South incision to confirm by palpation the cricothyroid membrane. (Photo Contributor: Lawrence B. Stack, MD.)

7. Using a stab incision, incise the cricothyroid membrane horizontally (east to west) (see [Fig. 22.87](#)).

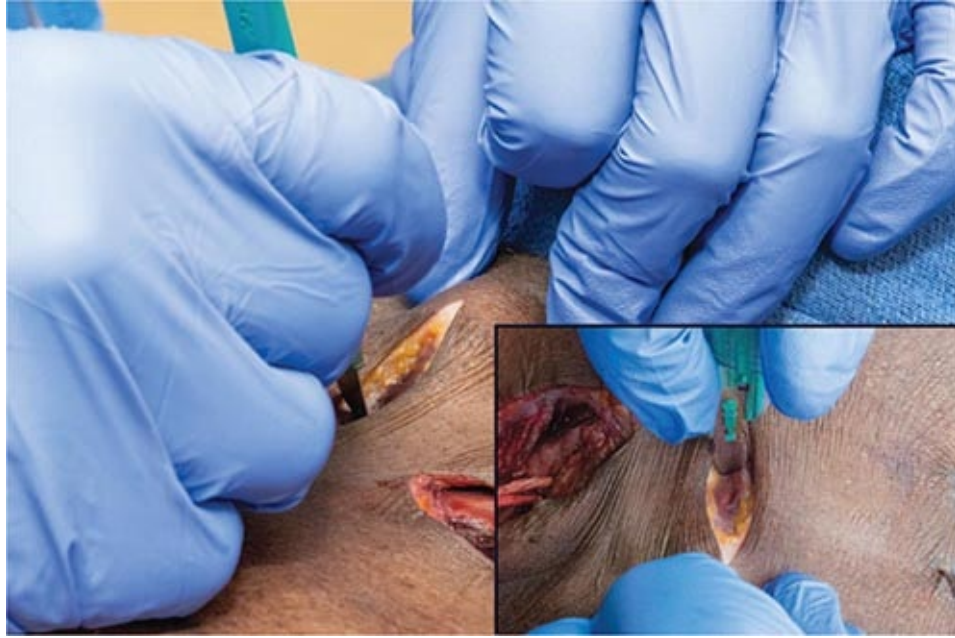


FIGURE 22.87 ■ Horizontal (East-West) Incision Through the Cricothyroid Membrane. Make a horizontal (East-West) stab incision through the cricothyroid membrane. (Photo contributor: Lawrence B. Stack, MD.)

8. Rotate the scalpel 180 degrees to fully extend the cricothyroid membrane incision (see [Fig. 22.88](#)).



FIGURE 22.88 ■ Horizontal (West-East) Incision Through the Cricothyroid Membrane. Rotate the scalpel blade 180 degrees to extend and complete the incision through the cricothyroid membrane. The hole should allow the insertion of the small finger into the trachea. (Photo contributor: Lawrence B. Stack, MD.)

9. Firmly place the dominant hand small finger into the trachea directed caudally and blunt dissect to allow passage of an ETT (see [Fig. 22.89](#)).



FIGURE 22.89 ■ Finger in the Trachea. Place the dominant hand small finger into the trachea directed caudally while awaiting the bougie. (Photo contributor: Lawrence B. Stack, MD.)

10. Place a bougie in the trachea directed caudally (see [Fig. 22.90](#)).



FIGURE 22.90 ■ Bougie in the Trachea. Place a bougie in the trachea directed caudally. (Photo

contributor: Lawrence B. Stack, MD.)

11. Insert a 6.0-mm ID ETT over the bougie and into the trachea until the top of balloon is at least 3 cm below the level of the skin (see [Fig. 22.91](#)).



FIGURE 22.91 ■ Endotracheal Tube Over Bougie. While controlling the bougie, place the endotracheal tube over the bougie. (Photo contributor: Lawrence B. Stack, MD.)

12. Inflate the ETT cuff (see [Fig. 22.92](#)).



FIGURE 22.92 ■ Endotracheal Tube in the Trachea. Place a 6.0-mm ETT over the bougie and into the trachea far enough so the balloon can be inflated in the trachea. (Photo contributor: Lawrence B. Stack, MD.)

13. Remove bougie while holding ETT in the trachea.
14. Initiate ventilation.
15. Confirm tube placement with end-tidal carbon dioxide detector.
16. Secure ETT using institutionally accepted method.

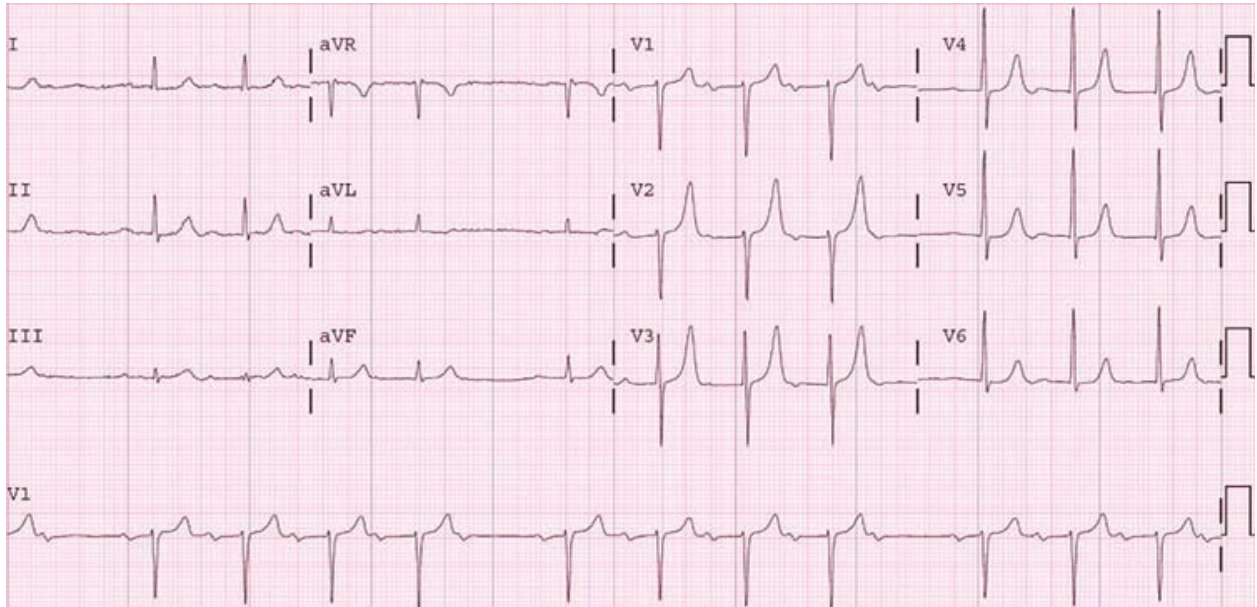
Chapter 23

ECG ABNORMALITIES

Maite A. Huis in 't Veld

Amal Mattu

R. Jason Thurman



Second-Degree AV Block (Mobitz I, Wenckebach). The PR interval gradually increases until a P wave is not followed by a QRS and a beat is “dropped.” The process then recurs. P waves occur at regular intervals, though they may be hidden by T waves. (ECG contributor: James Paul Brewer, MD.)

Part 1: ST-T Abnormalities

ACUTE ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

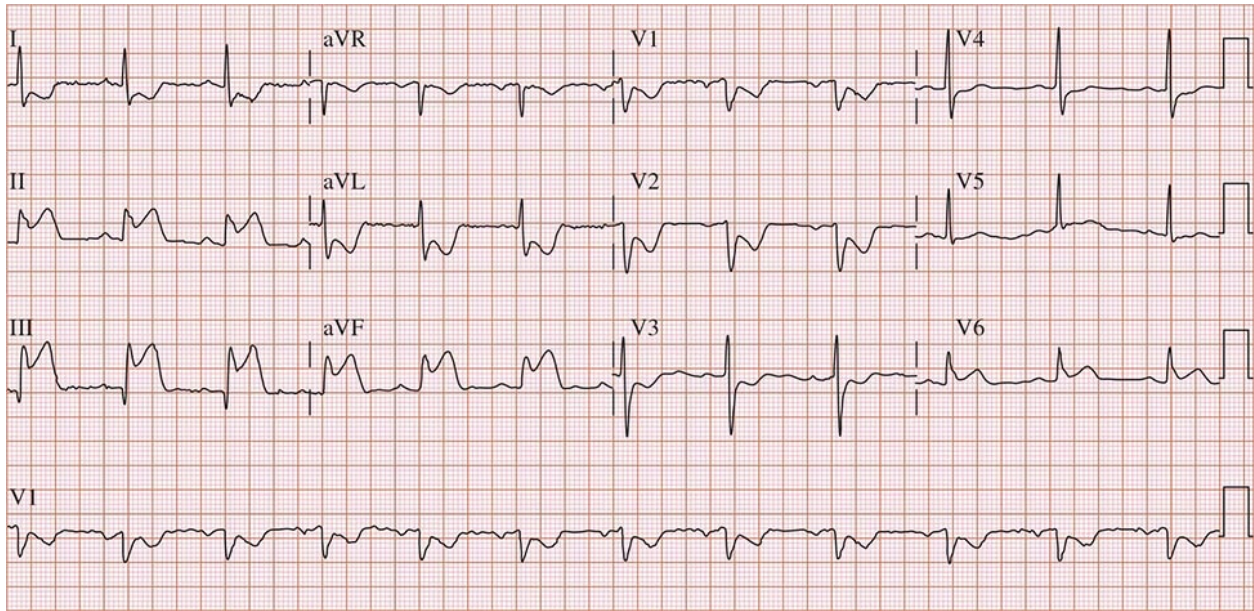


FIGURE 23.1A ■ ST-Segment Elevation Myocardial Infarction (STEMI). (ECG contributors: Clifford L. Freeman, MD, and Nicole S. McCoin, MD.)

ECG Findings

- New ST-segment elevation at the J point in at least two anatomically contiguous leads.
 - Men \geq 40 years of age: 2 mm (0.2 mV) in V_2 - V_3 and 1 mm (0.1 mV) in all other leads
 - Men < 40 years of age: 2.5 mm (0.25 mV) in V_2 - V_3 and 1 mm (0.1 mV) in all other leads
 - Women: 1.5 mm (0.15 mV) in V_2 - V_3 and 1 mm (0.1 mV) in all other leads

Pearls

1. The recommended therapy is emergent percutaneous coronary intervention (PCI). If PCI cannot be performed within 90 minutes, then thrombolysis is recommended (unless there are absolute contraindications to thrombolysis).
2. Reciprocal changes increase the specificity of ST-segment elevations for ST-segment elevation myocardial infarction (STEMI). Reciprocal changes are ST depressions in leads found at a 180-degrees vector opposite to the leads with ST elevations.

3. The development of pathologic Q waves suggests progression from myocardial injury to infarction.

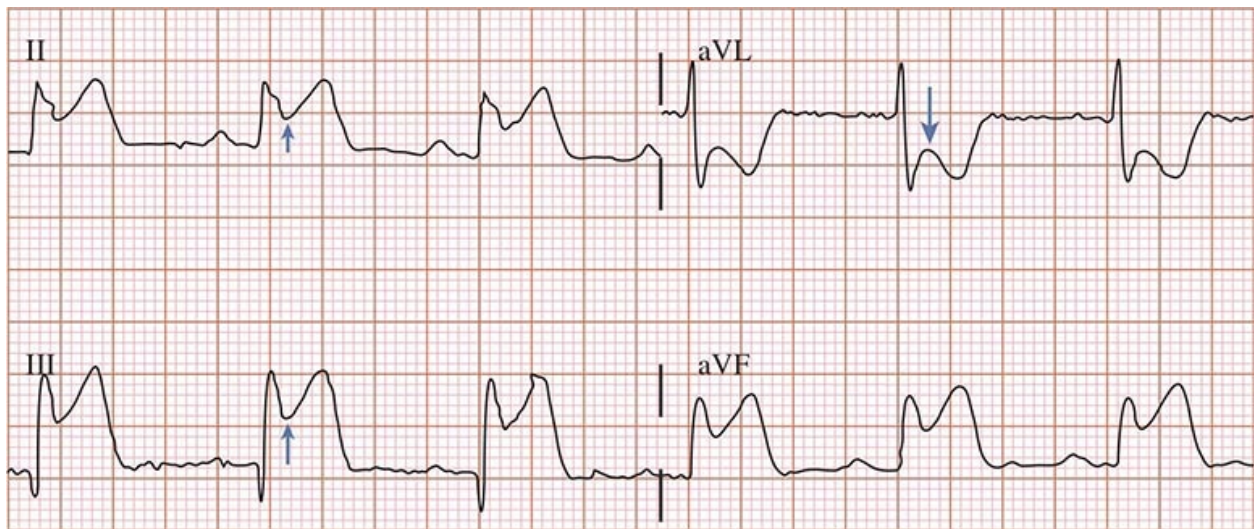


FIGURE 23.1B ■ Pathologic ST-segment elevation (upward arrows) is seen in the inferior leads (II, III, aVF) with reciprocal ST-segment depression seen here in lead aVL (downward arrow).

ACUTE ANTERIOR MYOCARDIAL INFARCTION

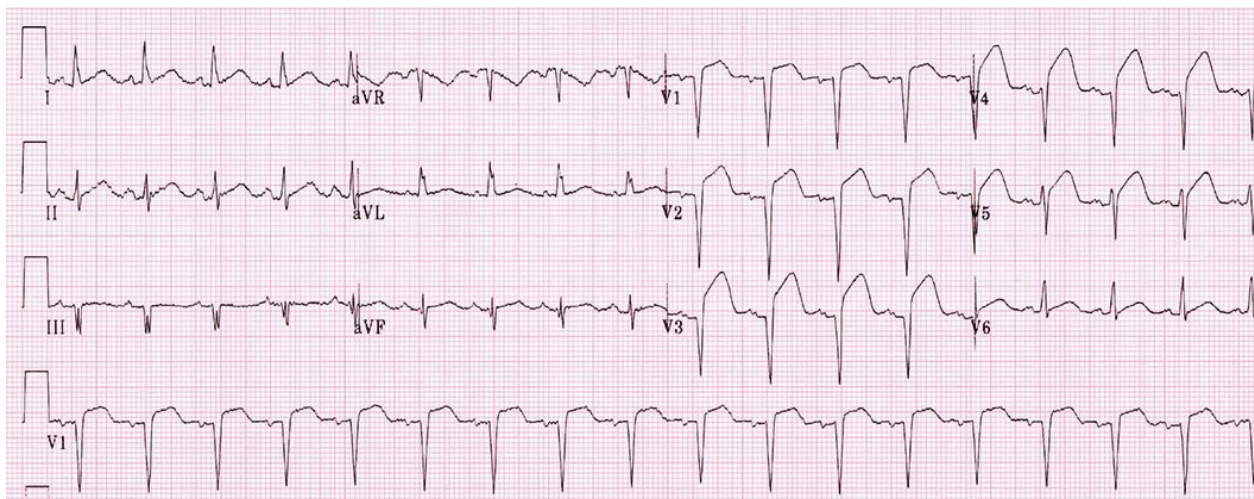


FIGURE 23.2A ■ **Acute Anteroseptal Myocardial Infarction.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST-segment elevation in the precordial leads (V₁-V₆).

- Q-wave formation in the precordial leads (V₁-V₆).
- Reciprocal ST-segment depression in the inferior leads (II, III, aVF).
- V₁-V₂: Septal injury.
- V₃-V₄: Anterior injury.
- V₅-V₆, I, and aVL: Anterolateral injury. Lateral injury may involve leads I and aVL only, and this is sometimes referred to as “high lateral” injury.

Pearls

1. Anterior STEMI results from occlusion of the left anterior descending (LAD) artery.
2. Anterior STEMI due to LAD artery occlusion has the worst prognosis of all STEMI locations. This is due to the large territory supplied by the LAD.
3. The reciprocal changes seen in the inferior leads (II, III, and aVF) are proportional to the magnitude of the ST elevations in I and aVL, and hence may be absent if the anterior STEMI does not involve the high lateral leads.
4. A “wrap around LAD” occlusion can produce ST elevations in not only the anterior precordial leads but also the inferior and lateral leads. This type of STEMI can easily be mistaken for pericarditis or early repolarization because of the ST elevations in multiple vascular territories and the absence of reciprocal ST depression.



FIGURE 23.2B ■ Pathologic ST-segment elevation beyond 1 mm (double arrow) with pathologic Q waves (arrow) in lead V₃. The ST segment demonstrates a convex upward, or “tombstone,” morphology.

ACUTE INFERIOR MYOCARDIAL INFARCTION

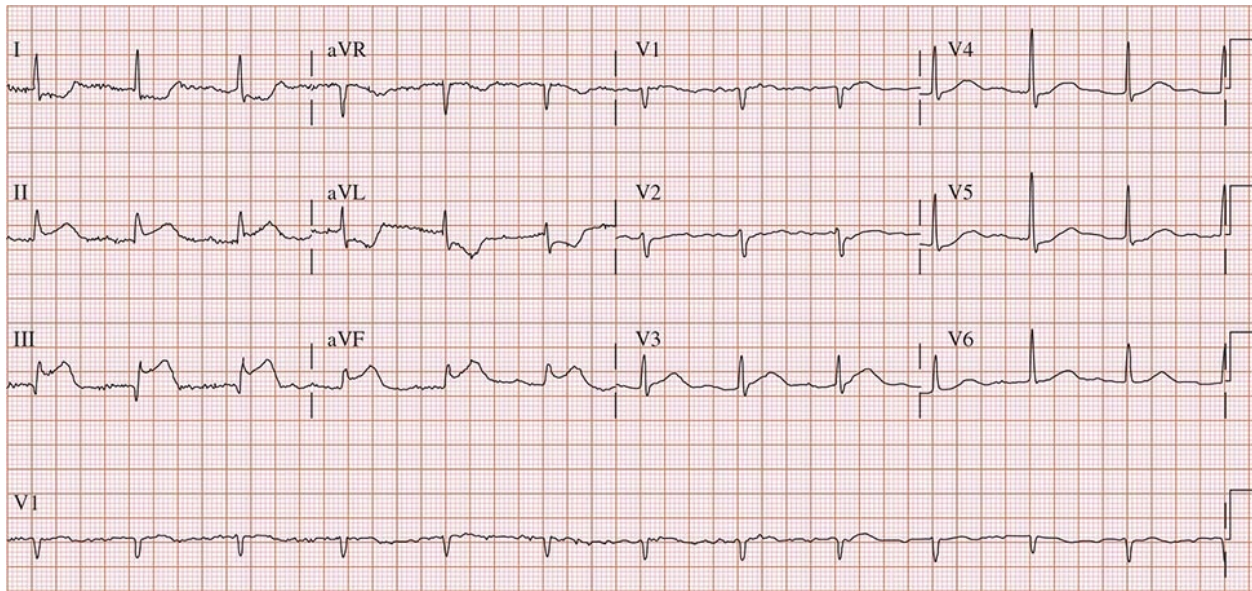


FIGURE 23.3A ■ Acute Inferior Myocardial Infarction. (ECG contributors: Clifford L. Freeman, MD, and Nicole S. McCain, MD.)

ECG Findings

- ST-segment elevation in inferior leads (II, III, aVF)
- Q-wave formation in the inferior leads (II, III, aVF)
- Reciprocal ST-segment depressions in the anterior leads (V₁-V₃) and high lateral leads (I, aVL)

Pearls

1. The right coronary artery supplies blood to the right ventricle, the sinoatrial (SA) node, the inferior portions of the left ventricle, and usually the posterior portion of the left ventricle and the atrioventricular (AV) node.
2. The left circumflex supplies blood to the lateral ventricle but also frequently supplies blood to the inferior portions of the left ventricle.
3. Infarctions involving the SA node may produce sinus dysrhythmias including tachycardias, bradycardias, and sinus arrest.
4. Infarctions involving the AV node may produce AV blocks.
5. In the presence of acute inferior injury, a right-sided ECG should be obtained

to look for right ventricular involvement.

6. Since the right coronary artery so often supplies the posterior left ventricle, look for evidence of a posterior infarction (as present in the example) and consider obtaining an ECG with posterior leads.
7. Reciprocal ST-depression is very common in cases of inferior STEMI involving right coronary artery occlusion. In fact, ST depression in aVL may precede the development of ST elevation in the inferior leads. Inferior STEMI due to left circumflex occlusion often lacks reciprocal ST depression in aVL.



FIGURE 23.3B ■ ST-segment elevation is present in the inferior leads (II, III, aVF) (upward arrow), with reciprocal ST depression in the high lateral leads (aVL seen here) (downward arrow).

ACUTE RIGHT VENTRICULAR MYOCARDIAL INFARCTION

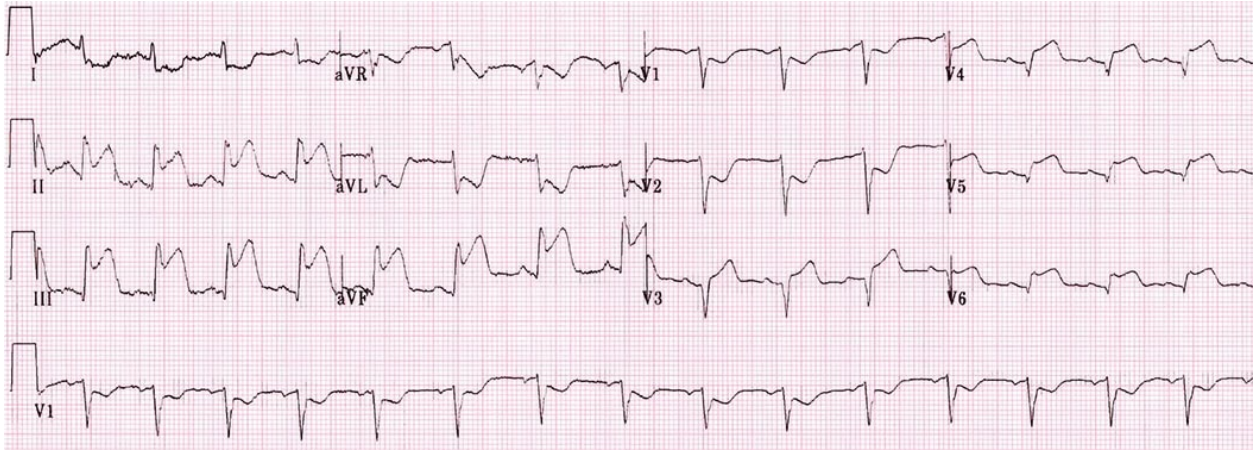


FIGURE 23.4A ■ Right Ventricular Myocardial Infarction. This ECG was obtained with right-sided lead placement. (ECG contributor: Thomas Bottoni, MD.)

ECG Findings

- Any ST elevation > 0.5 mm (0.05 mV) in right-sided leads (V_1R - V_6R).
- Simultaneous ST elevation in lead V_1 with ST-depression in lead V_2 strongly suggests right ventricular (RV) myocardial infarction (MI).
- Often associated with inferior MI and/or posterior MI.

Pearls

1. ECG changes of RV MI are subtle and easily missed. Obtain a right-sided ECG in any patient with inferior STEMI or in a patient with a significant hypotensive response to nitrates.
2. A right-sided ECG is obtained by placing right-sided V leads, with V_1 - V_6 in mirror-image locations on the right side of the chest. The ECG leads are then marked V_1R - V_6R . Alternatively, V_1 and V_2 leads can be placed in their usual position with leads V_3 - V_6 placed in mirror image (then V_3R - V_6R). V_4R is the most sensitive lead.
3. The right ventricle is very preload dependent. As such, the administration of nitroglycerin or diuretics in the presence of acute RV infarction can precipitate profound hypotension. Avoid any preload-reducing medications in these patients and give fluids liberally to maintain blood pressure as long as the lungs remain clear. Treat hypotension in a patient with acute RV MI aggressively with fluids.

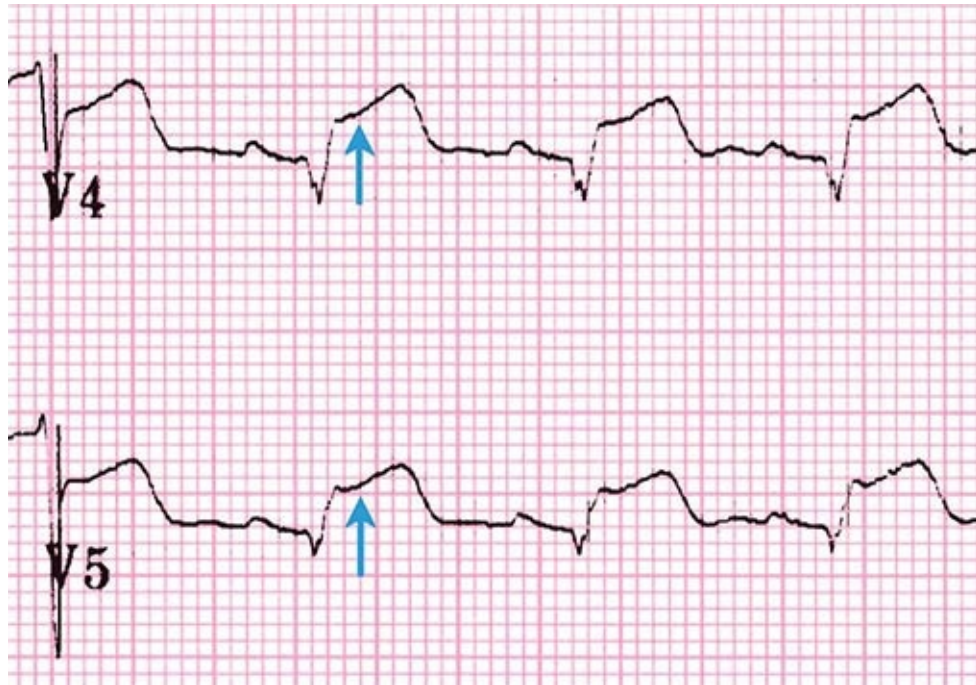


FIGURE 23.4B ■ ST elevation in V₄R and V₅R (arrows), with the V₄ and V₅ leads placed in their mirror-image locations on the right side of the chest. Any ST elevation seen in the right-sided precordial leads is significant.

ACUTE POSTERIOR MYOCARDIAL INFARCTION

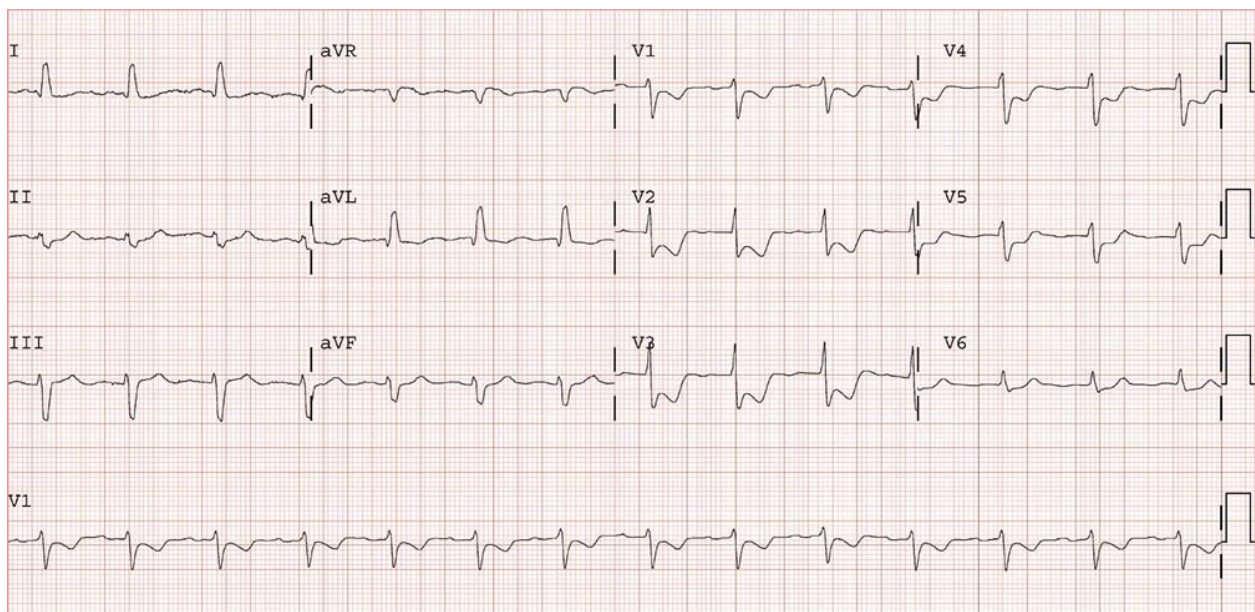


FIGURE 23.5A ■ **Acute Posterior Myocardial Infarction.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- ST-segment depression in leads V_1 - V_3
- Upright T waves in V_1 - V_3
- Tall R waves with an R-wave/S-wave ratio greater than 1 in lead V_1 - V_3 , most often seen in V_2 - V_3 , developing over the course of hours

Pearls

1. Note that some authors refer to “posterior” infarction as “inferolateral” infarction, based on echocardiographic data that show that the involved portion of the left ventricle in question is actually inferolateral rather than truly posterior in location.
2. This diagnosis is often missed as the posterior portion of the left ventricle has no ECG electrodes directly overlying it. This means that posterior MI will only reflect as reciprocal changes that are most common in V_1 - V_3 . Instead of ST elevations, one will find ST depressions, and instead of Q waves, one will see tall R waves.
3. Posterior involvement may be confirmed with posterior leads. V_7 is located in the left posterior axillary line; V_8 is located at inferior tip of left scapula; V_9 is positioned between V_8 and the spine, all in the same horizontal plane as V_6 .
4. Frequently, an inferior MI is also present with a posterior MI, since the right coronary artery serves both areas.

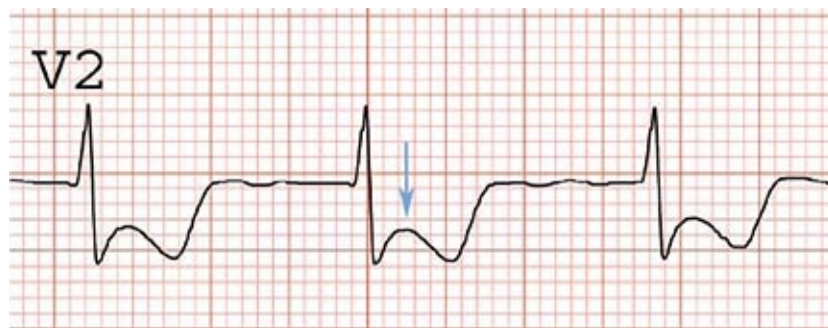


FIGURE 23.5B ■ This tracing demonstrates injury in the posterior LV, manifesting as acute ST depression in V_2 (arrow).

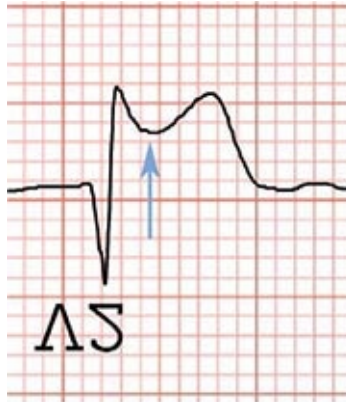


FIGURE 23.5C ■ Flipping the ECG upside down and looking through the paper with a backlight shows an ST-elevation injury pattern (arrow). A posterior ECG can help distinguish between posterior STEMI and anterior ischemia.



FIGURE 23.5D ■ The R-wave amplitude approximates that of the S wave (arrow), and the R-wave duration is significant (>4 ms). This is actually an “inverted Q wave” from a posterior infarction.

LEFT MAIN LESION

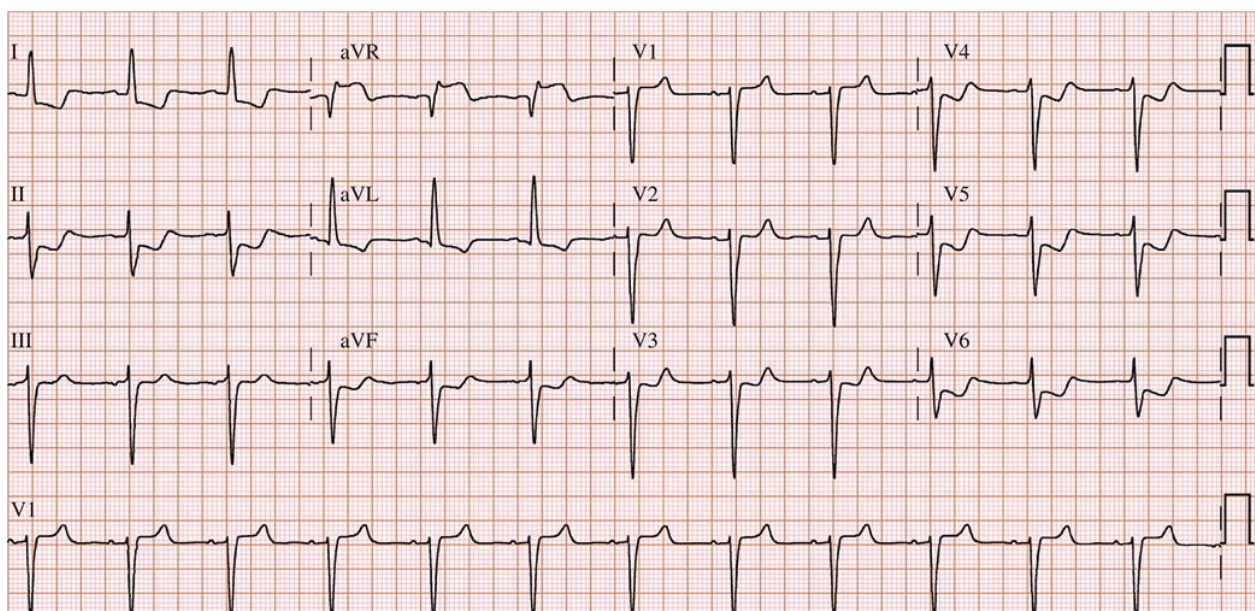


FIGURE 23.6A ■ **Left Main Coronary Artery Lesion.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- ST elevation in aVR ≥ 1 mm.
- ST elevation in $V_1 \geq 1$ mm is common as well.
- Widespread ST depression, typically in at least six to eight other leads.

Pearls

1. The left main coronary artery branches into the left anterior descending artery and the circumflex artery. It supplies blood to the ventricular septum and the anterior and lateral aspects of the left ventricle, usually sparing the posterior and inferior portion.
2. Risk of cardiogenic shock is high since so much of the left ventricle is served by the left main coronary artery. Patients with a 100% LMCA occlusion often suffer a pre-hospital cardiac arrest and may present in cardiogenic shock.
3. ST segment elevation in lead aVR may also be seen in severe triple vessel disease, proximal left anterior descending occlusion and diffuse subendocardial ischemia.
4. Other causes of diffuse myocardial ischemia, such as aortic dissection or profound anemia, can also produce this ECG pattern. ST elevation in lead aVR is not specific for left main disease, as there are many other potential causes of this finding.

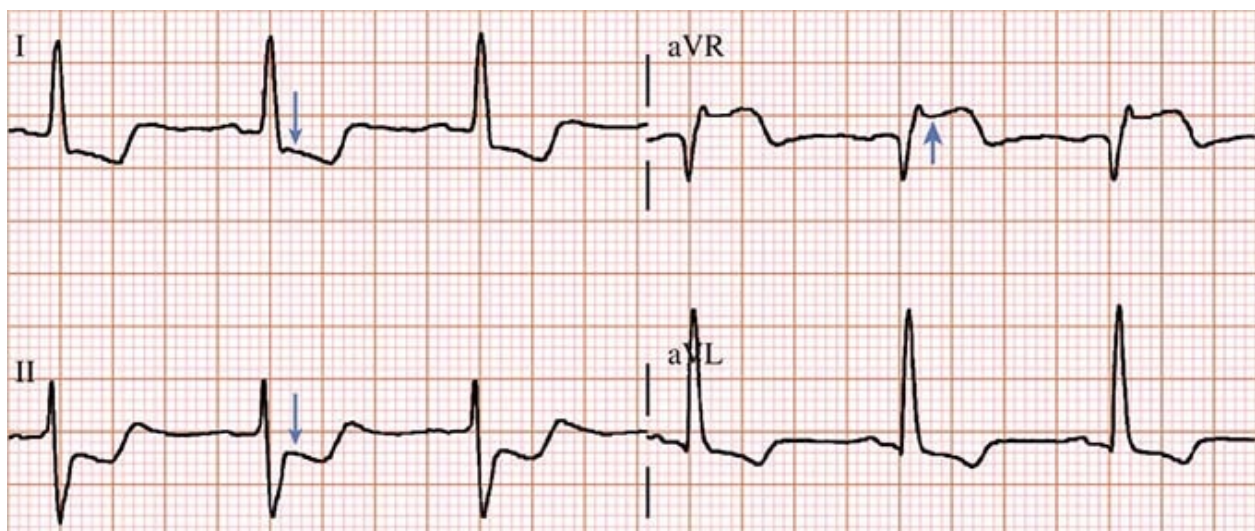


FIGURE 23.6B ■ Significant ST elevation is present in lead aVR (upward arrow), and diffuse reciprocal ST depression is seen in multiple leads (downward arrows).

SGARBOSSA CRITERIA FOR AMI IN SETTING OF LBBB

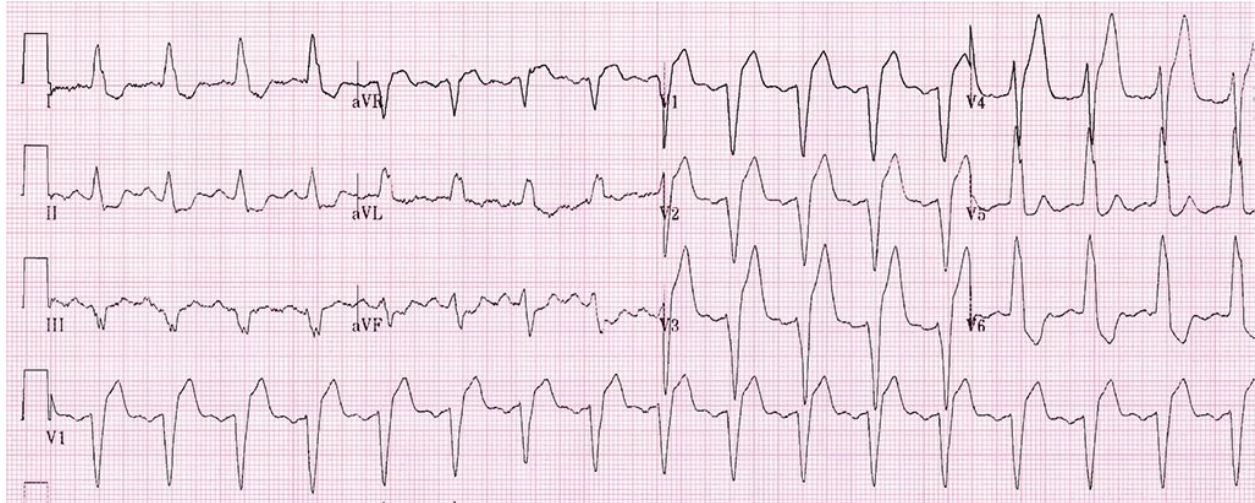


FIGURE 23.7A ■ **Acute Myocardial Infarction by Sgarbossa Criteria in the Setting of Underlying LBBB.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Concordant ST elevation ≥ 1 mm with a positive QRS complex (score = 5)
- Concordant ST depression ≥ 1 mm in leads V_1 - V_3 (score = 3)
- Discordant ST elevation ≥ 5 mm (score = 2)

Pearls

1. Diagnosing STEMI in patients with left bundle branch block (LBBB) is challenging, because a normal LBBB produces ST-segment elevations and depressions that are typically used to diagnose STEMI. The Sgarbossa criteria can be used to diagnose acute MI (AMI) in the presence of LBBB with high specificity. However, most myocardial ischemia in the setting of LBBB does not produce these changes. An absence of these findings should not be used as evidence against acute coronary syndrome.



FIGURE 23.7B ■ The ST elevation is greater than 5 mm discordant from the primary QRS deflection (arrow).

2. Score of greater than or equal to 3 gives a specificity for MI of 90%.
3. These criteria have been proposed to be used in patients with pacemakers as well. Small studies have demonstrated their utility in this setting, but larger studies are lacking. Nevertheless, the European Society of Cardiology guidelines have endorsed the use of the Sgarbossa criteria for catheterization lab activation in the setting of paced rhythms.
4. Modified Sgarbossa criteria have been proposed to increase diagnostic accuracy. In the modified criteria, the 3rd criterion (discordant elevation ≥ 5 mm) was replaced by discordant ST elevation with amplitude $> 25\%$ of the depth of the preceding S wave.



FIGURE 23.7C ■ The ST depression is greater than 1 mm concordant to the primary QRS deflection (arrow).

SUBENDOCARDIAL ISCHEMIA

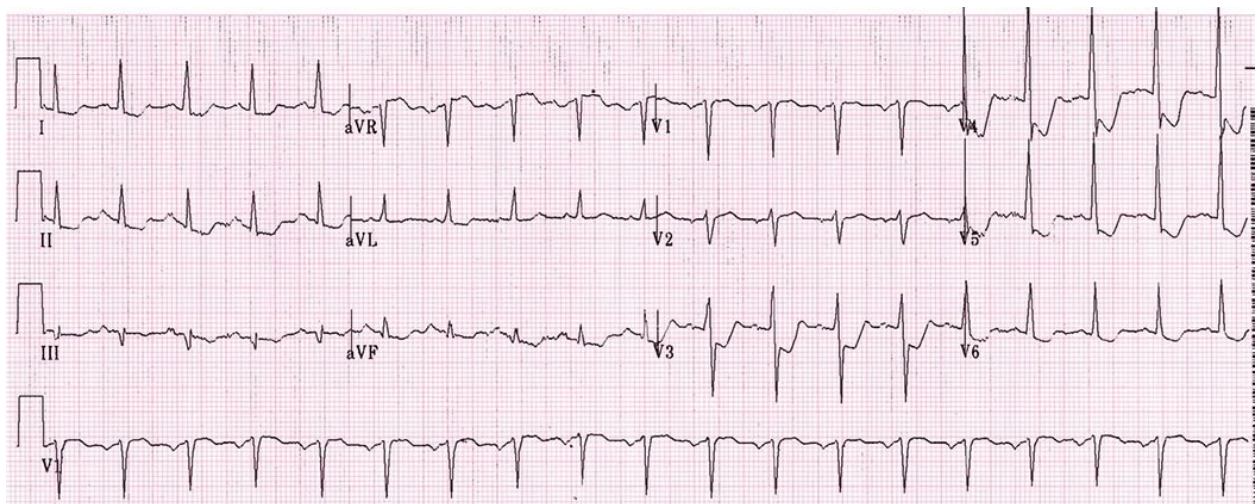


FIGURE 23.8A ■ **Subendocardial Ischemia.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment depression ≥ 1 mm in consecutive leads.
- ST segments may be horizontal or downsloping.

Pearls

1. Some ST depression in the lateral precordial leads (V_4 - V_6) is common at higher heart rates and is commonly seen during exercise treadmill tests, but such depression should not be downsloping unless ischemia is also present.
2. ST elevation in other leads suggests that the depression may represent reciprocal changes from acute injury rather than subendocardial ischemia.
3. Downsloping ST depression may also be seen in left ventricular hypertrophy (LVH), but this depression should not be dynamic and should be stable with serial ECGs. ST depression from ischemia will be dynamic, changing with time on serial ECGs.
4. Isolated ST depression in leads V_1 and V_2 may represent posterior ischemia.



FIGURE 23.8B ■ Downsloping ST segments depressed greater than 1 mm (arrow). These changes were dynamic over time. The patient sustained a nontransmural myocardial infarction.

HYPERACUTE T WAVES

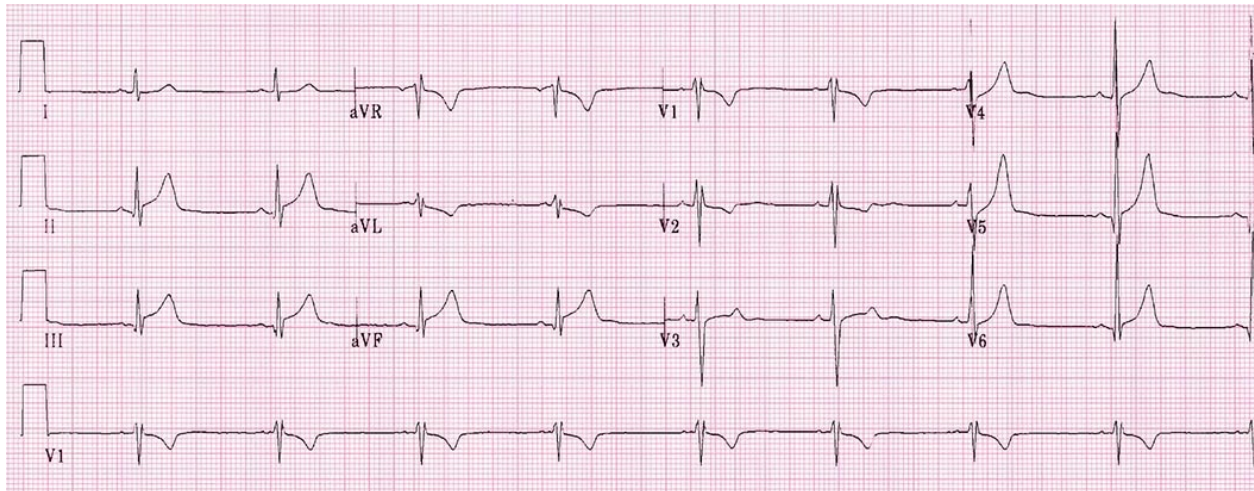


FIGURE 23.9A ■ Hyperacute T Waves. T waves in a patient with acute myocardial ischemia. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Tall, often domed T waves.
- T waves have asymmetric appearance.
- T waves will often have a broad base.

Pearls

1. Hyperacute T waves occur very early during myocardial injury and are transient.
2. The term “hyperacute T waves” is reserved for the early stages of MI.
3. The presence of prominent T waves appearing to be “hyper-acute” (ie, indicative of ischemia) is somewhat nonspecific and can also be found in patients with LVH, early repolarization, hyperkalemia, and a few other conditions. However, serial ECGs are useful in distinguishing ischemic T waves from other causes of prominent T waves. In the presence of ischemia, the ECG is likely to show evolving changes, thus confirming that the prominent T wave is a sign of ischemia. In the other conditions, evolving changes are less likely.



FIGURE 23.9B ■ T waves (double arrow) are large in proportion to the QRS complex. The height was transient and was significantly diminished in a serial tracing obtained 15 minutes later. Note also in the 12-lead ECG example above the presence of inferior ST elevation.

WELLENS WAVES

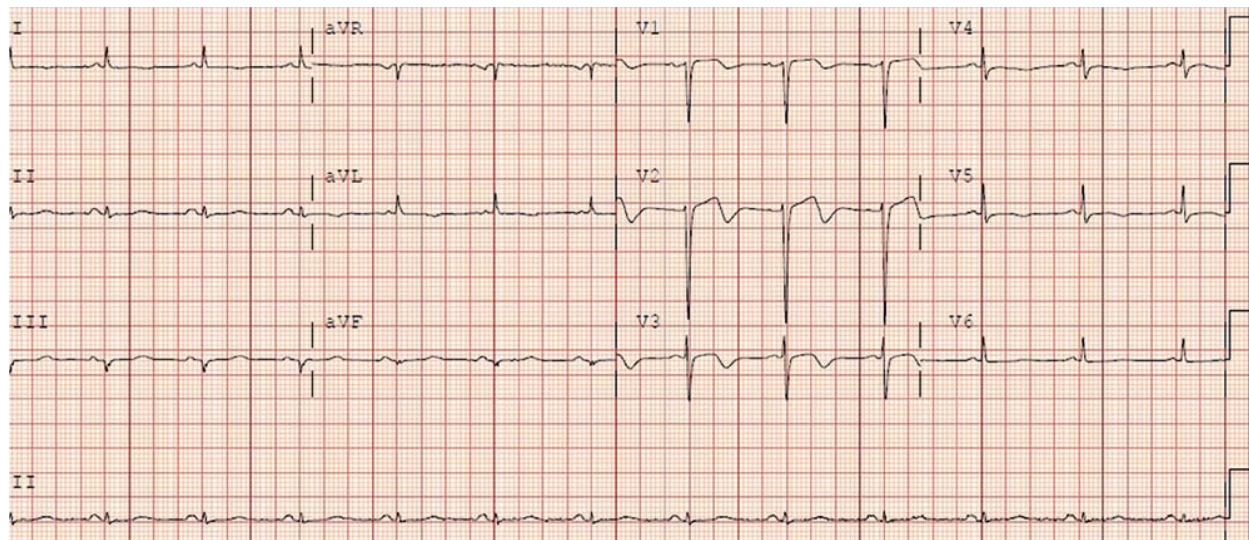


FIGURE 23.10A ■ **Wellens Waves.** Wellens waves are present in the anterior leads and are indicative of a high-grade LAD lesion. (ECG contributors: Clifford L. Freeman, MD, and Nicole S. McCain, MD.)

ECG Findings

- Biphasic (“type A”) or deeply inverted (“type B”) T waves in V_2 - V_4 .
- Isoelectric or minimal elevation of the ST segment (< 1 mm).

- Absence of precordial Q waves.
- R-wave progression is preserved.

Pearls

1. These characteristic patterns of T-wave changes are closely associated with a subacute critical LAD artery stenosis.
2. The changes are classically apparent on ECG *after* resolution of chest pain.
3. These changes are often not associated with cardiac bio-marker elevations.
4. In the absence of ongoing pain or serial ECG changes, these patients can often be treated with urgent, rather than emergent, coronary intervention.

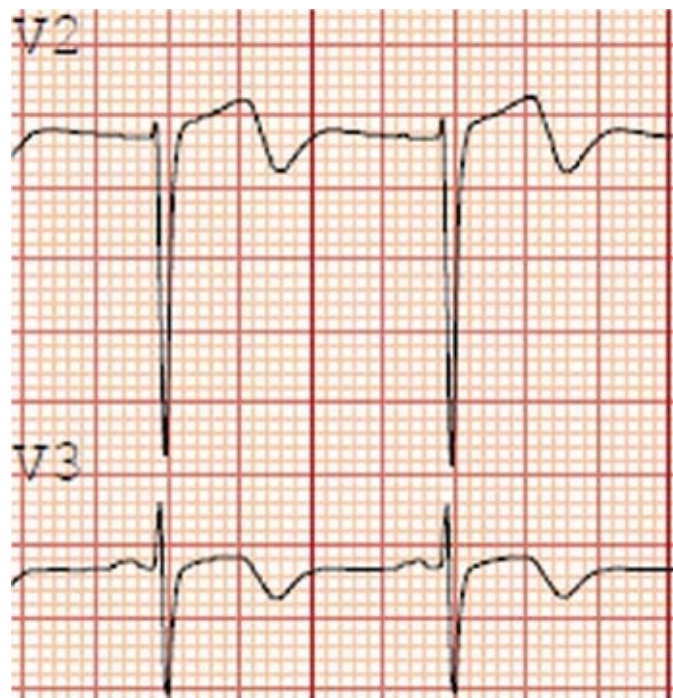


FIGURE 23.10B ■ Wellens type A, demonstrating biphasic T waves in the mid-precordial leads. These findings in the setting of suspected acute coronary syndrome strongly suggest an underlying high-grade LAD lesion.



FIGURE 23.10C ■ Wellens type B, demonstrating deeply inverted T waves in the mid-precordial leads. (ECG contributor: James V. Ritchie, MD.)

EARLY REPOLARIZATION

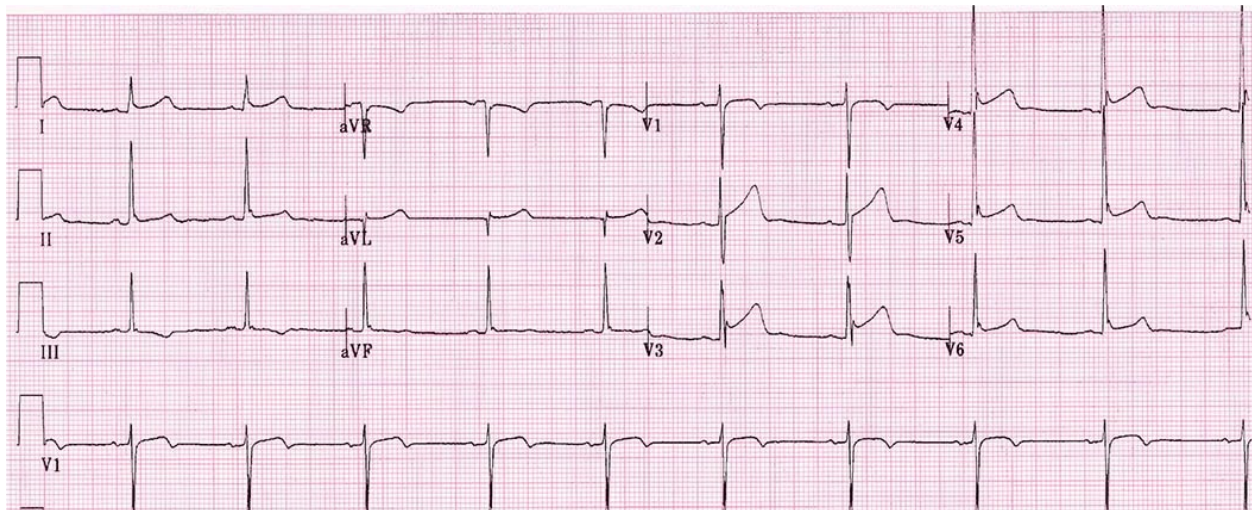


FIGURE 23.11A ■ **Early Repolarization.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST elevation, usually in the anterior leads, with a concave upward morphology
- J-point elevation, but usually less than one-third the total height of the T wave
- Terminal QRS notching
- Large, asymmetric T waves

Pearls

1. This is generally considered a normal variant and is especially common in young healthy males. However, recent studies have shown some increased susceptibility to sudden cardiac death over the long term.
2. Significant Q waves and/or reciprocal ST-segment depression in other leads should *not* accompany early repolarization. If present, they strongly suggest ischemia as the cause for the ST elevation.
3. The changes seen in early repolarization are static; hence, repeat ECG can be helpful. If dynamic changes are seen, suspect myocardial ischemia.

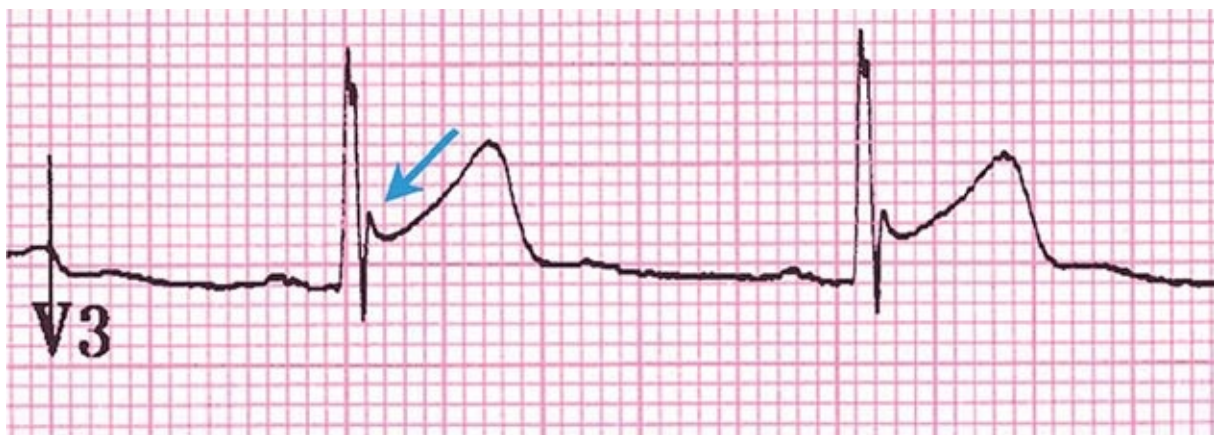


FIGURE 23.11B ■ ST elevation in precordial leads, with a concave-upward ST segment and a J-point notch (arrow).

LEFT VENTRICULAR ANEURYSM

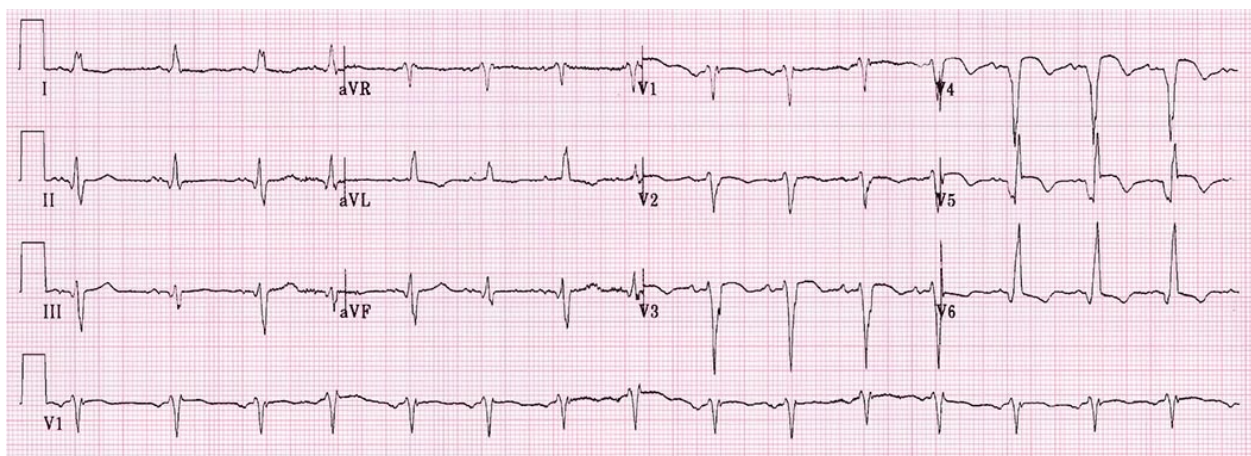


FIGURE 23.12A ■ **Left Ventricular Aneurysm.** This ECG was obtained on an asymptomatic patient with

history of MI 2 years prior. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST elevation in anterior contiguous leads
- Deep pathologic Q waves in anterior leads
- Absence of reciprocal changes
- Loss of R waves

Pearls

1. ST-segment elevation that occurs in the setting of an MI should resolve within days under normal circumstances. Persistent ST-segment elevation weeks or longer after MI is suspicious for ventricular aneurysm.
2. A left ventricular aneurysm is a localized area of dyskinetic myocardium that bulges outward during both systole and diastole.
3. Suspect an left ventricular aneurysm when these findings appear in the ECG of a patient who does not demonstrate symptoms suggesting acute coronary syndrome. However, one should also be vigilant for the presence of “silent” acute coronary syndrome.



FIGURE 23.12B ■ Persistent ST elevations (arrow) and deep, pathologic Q waves (arrowhead) in an asymptomatic patient with a history of anterior MI 2 years earlier.

ACUTE PERICARDITIS

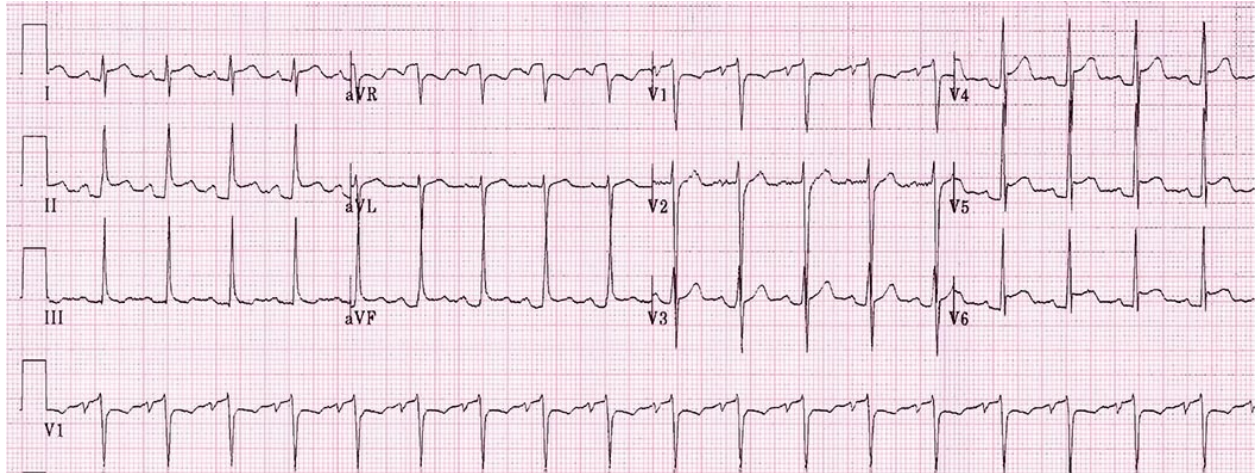


FIGURE 23.13A ■ **Acute Pericarditis.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Diffuse, concave upward ST elevation
- ST elevation typically greater in lead II than lead III
- PR depression in multiple leads (in viral pericarditis)
- PR elevation and ST depression in lead aVR common (although not specific for pericarditis)
- T-wave flattening or inversion in late stages
- Absence of reciprocal changes (with exception of aVR and V₁)

Pearls

1. Inflammation of the pericardium can cause pericarditis. As the pericardium is circumferential, the ECG changes are most often diffuse.
2. Presence of significant Q waves or reciprocal changes in any leads (with the exception of aVR or V₁) should prompt consideration of acute or old MI.
3. Pericarditis may be focal, resulting in regional rather than diffuse ECG changes.

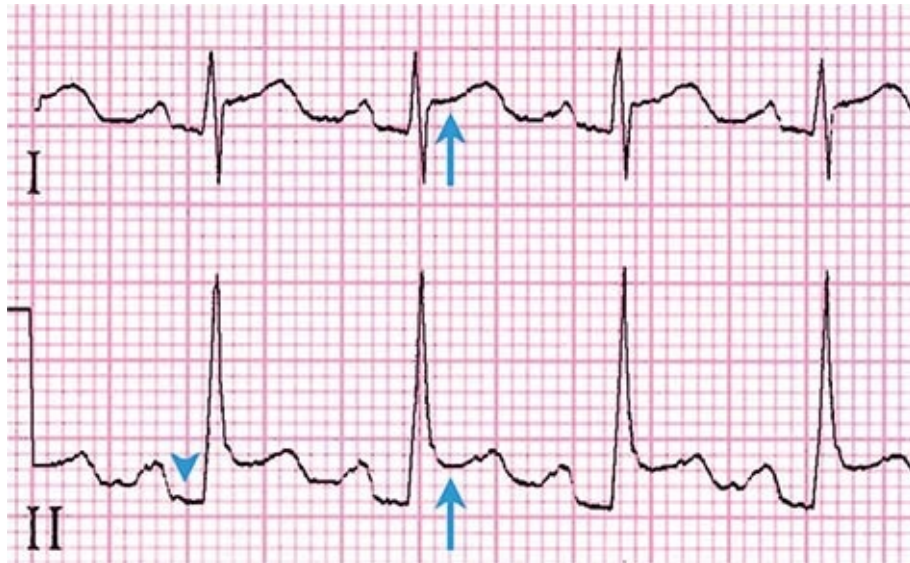


FIGURE 23.13B ■ ST elevation in noncontiguous leads I and II (arrows) with PR depression (arrowhead). No pathologic Q waves or reciprocal changes are present.

Part 2: Conduction Disturbances

FIRST-DEGREE AV BLOCK

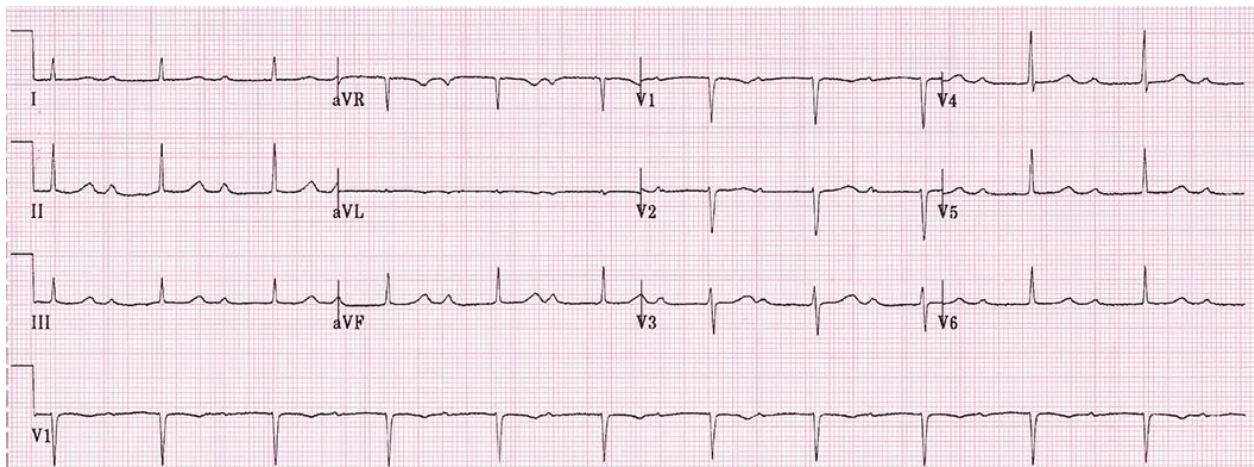


FIGURE 23.14A ■ **First-Degree AV Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- PR interval greater than 200 ms (normal 120-200 ms) with no significant variation in PR intervals between beats.
- Each P wave is followed by a QRS complex.

Pearls

1. This type of heart block usually does not affect heart function and can be considered nonpathologic (especially in athletes or patients with higher vagal tone).
2. First-degree block may also occur due to heart disease (myocarditis, rheumatic fever), drugs (digoxin, amiodarone, β -blockers, calcium channel blockers), or as a normal part of aging.

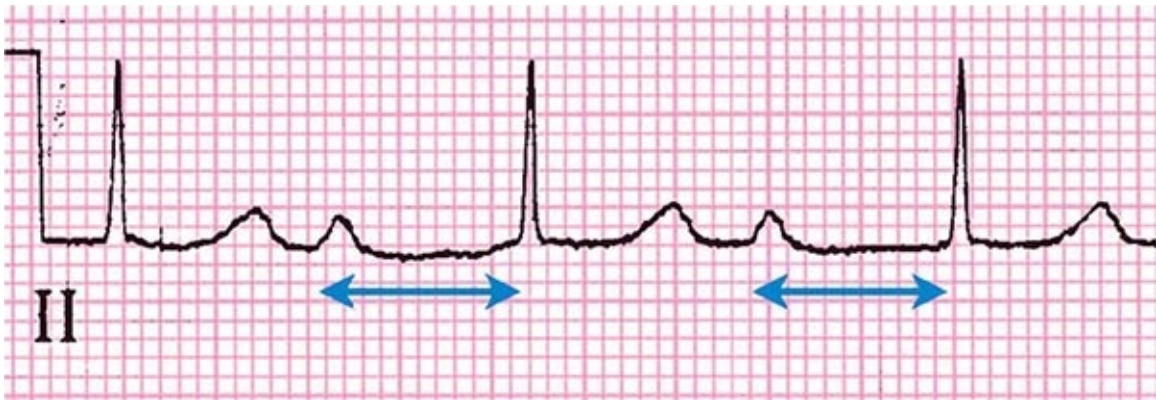


FIGURE 23.14B ■ The PR interval is fixed (double arrows) and is longer than 0.2 seconds, or five small blocks.

TYPE 1 SECOND-DEGREE AV BLOCK (MOBITZ I, WENCKEBACH)

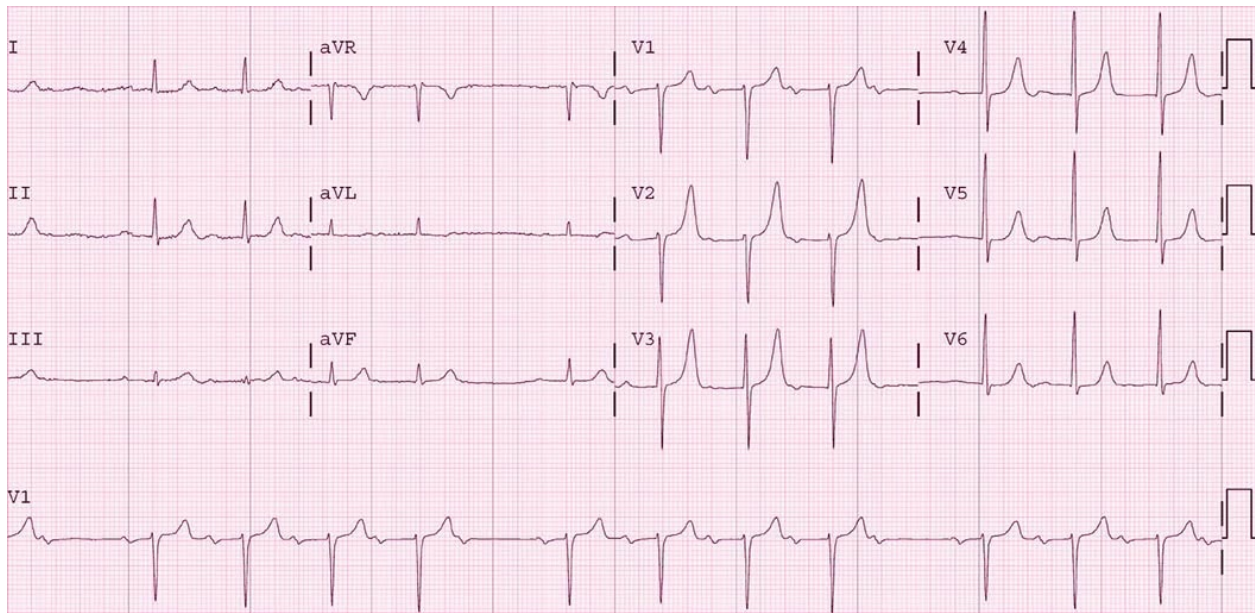


FIGURE 23.15A ■ Second-Degree AV Block (Mobitz I, Wenckebach). (ECG contributor: James Paul Brewer, MD.)

ECG Findings

- Progressive PR-interval prolongation throughout the cardiac cycle until a P wave occurs without a QRS complex (“dropped” beat).
- After the dropped QRS complex, the cycle continues again with the PR interval of the 1st beat in the cycle always shorter than the PR interval of the last beat in the previous cycle.
- P wave may be hidden by the preceding T wave.

Pearls

1. The number of P-QRS complexes prior to the “dropped” beat may vary.
2. A clue to the diagnosis of Mobitz type I AV block can be found in the appearance of grouped QRS complexes.
3. This type of block is often asymptomatic and may be seen in athletes or others with high vagal tone.
4. These patients have low risk of progression to complete heart block and usually do not require a pacemaker. However, Mobitz type I heart block may be caused by inferior MI or drugs (digoxin, amiodarone, β -blockers, calcium channel blockers).

2. Mobitz type II block can accompany MI and has a high chance of progression to a complete heart block.

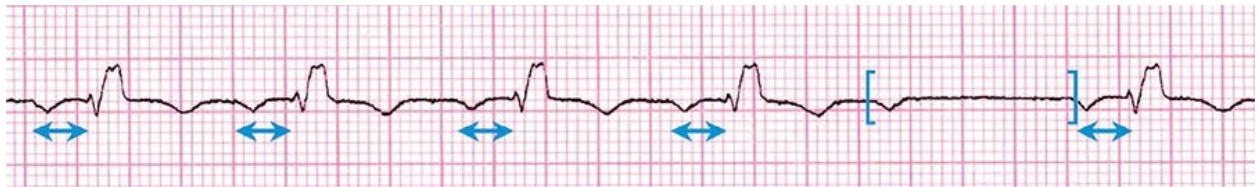


FIGURE 23.16B ■ The PR interval is constant (double arrows) until the dropped beat (brackets).

THIRD-DEGREE (COMPLETE) AV BLOCK

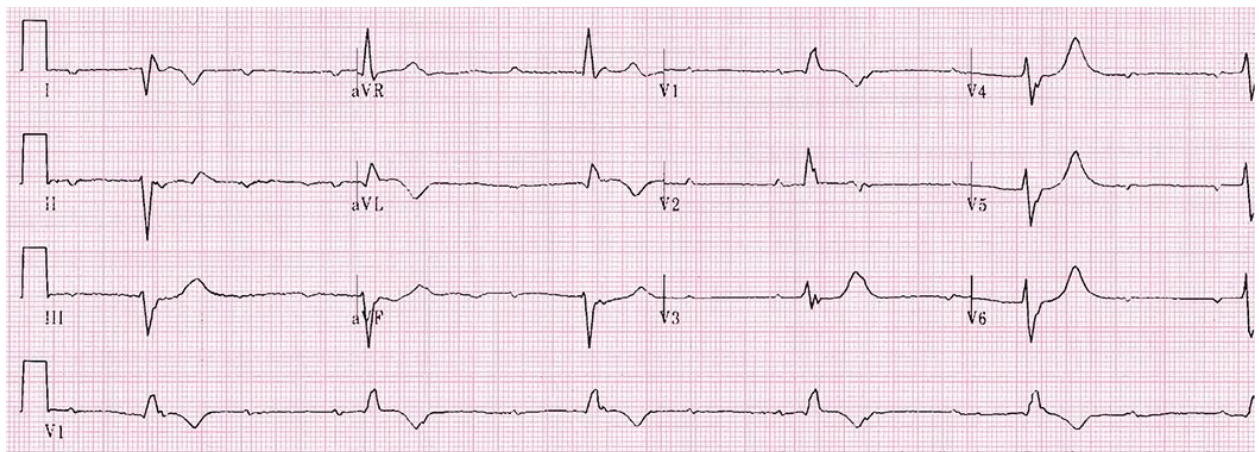


FIGURE 23.17A ■ **Third-Degree AV Block (Complete Heart Block).** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Atrial and ventricular electrical activities are entirely dissociated.
- The P-P and R-R intervals remain constant.
- P waves may be hidden in the QRS complex or may distort the shape of the T wave.
- The atrial rate is usually faster than the ventricular rate, and the ventricular rate is generally slow.

Pearls

1. Third-degree block is also called complete heart block because no impulses

are conducted from the atria to the ventricles.

2. AV rate and QRS morphology depend upon the location of the escape pacemaker.
3. A nodal escape rate is typically 40 to 60 bpm, with a narrow QRS complex.
4. Ventricular escape rate is usually 20 to 40 bpm, with a widened QRS complex.
5. Complete heart block may be caused by MI, conduction system disease, or drugs such as digoxin.
6. Complete heart block may dramatically decrease cardiac output and cause hypotension. Emergent cardiac pacing is required if the patient is unstable.



FIGURE 23.17B ■ The P-P interval is uniform (lower double arrows) and the R-R interval is uniform (upper double arrows), but the P waves and QRS complexes are disassociated.

QT INTERVAL ABNORMALITIES

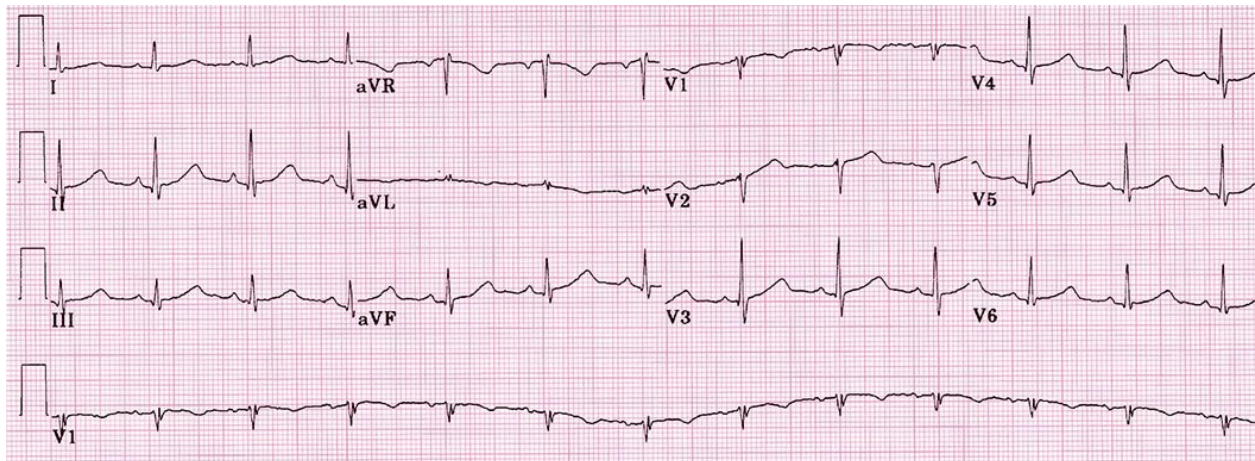


FIGURE 23.18A ■ **Prolonged QT Interval.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Normal QTc interval is gender based: < 440 ms in men or < 460 ms in women.
- The QTc interval is prolonged if it is > 440 ms in men or > 460 ms in women.

- The QTc interval is abnormally short if < 350 ms.

Pearls

1. The QT interval will increase with bradycardia and decrease with tachycardia; thus, it is important to use the corrected QT interval ($QT_c = QT/\sqrt{R-R}$ interval) for heart rates other than 60 (in which the $QT_c = QT$).
2. In most cases, QT prolongation is acquired, either due to medications or electrolyte abnormalities. In more rare instances, the QT prolongation is caused by a congenital defect.
3. Prolongation of the QT interval predisposes the heart to the R-on-T phenomenon, which causes torsades de pointes. The risk of torsades de pointes tends to be highest when the QT_c is > 500 ms.
4. Short QT can be caused by hypercalcemia, congenital short QT syndrome, or digoxin effect. A short QT predisposes the patient to atrial fibrillation, ventricular tachycardia, and ventricular fibrillation.



FIGURE 23.18B ■ QT Interval Prolongation. QT of 440 ms, QT_c of 498 (double arrow). Note the QT interval is measured from the beginning of the QRS complex to the termination point of the T wave.

RIGHT BUNDLE BRANCH BLOCK

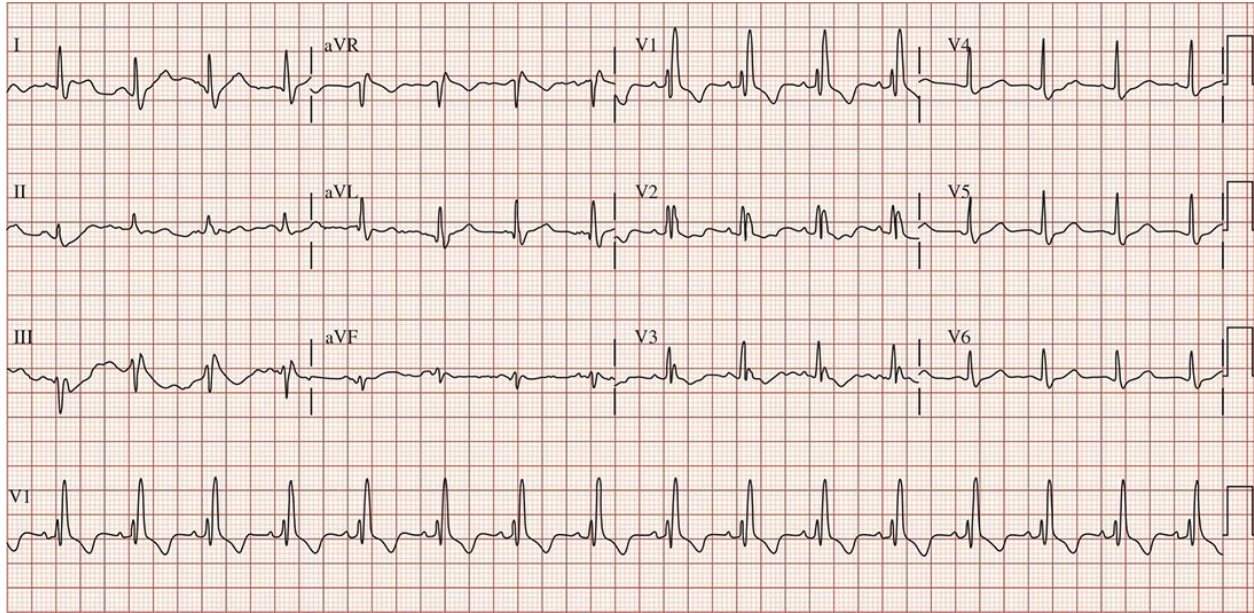


FIGURE 23.19A ■ Right Bundle Branch Block. (ECG contributors: Clifford L. Freeman, MD, and Nicole S. McCain, MD.)

ECG Findings

- Wide QRS complex > 120 ms.
- rsR' pattern in leads V_1 and V_2 .
- Slurred S wave in lateral leads (I, aVL, V_5 - V_6).
- ST depression and T-wave inversion is typical in the right precordial leads (V_1 - V_3).
- When all of these criteria are met except that the QRS duration is < 120 ms, an *incomplete* right bundle branch block (RBBB) pattern is diagnosed.

Pearls

1. In RBBB, conduction through the right bundle is delayed, whereas depolarization through the left bundle occurs at normal speed. This creates a secondary R wave (an rsR' pattern), ST depression, and T-wave inversion in the right pre-cordial leads (V_1 - V_3), as well as a slurred S wave in the lateral leads.
2. Acute right heart strain, as may occur with pulmonary embolism, may result in the appearance of an incomplete or complete RBBB pattern.



FIGURE 23.19B ■ Note the rsR' pattern in V₁ (arrowheads), the inverted T wave (arrow), and the QRS duration greater than 120 ms (double arrow).

LEFT BUNDLE BRANCH BLOCK

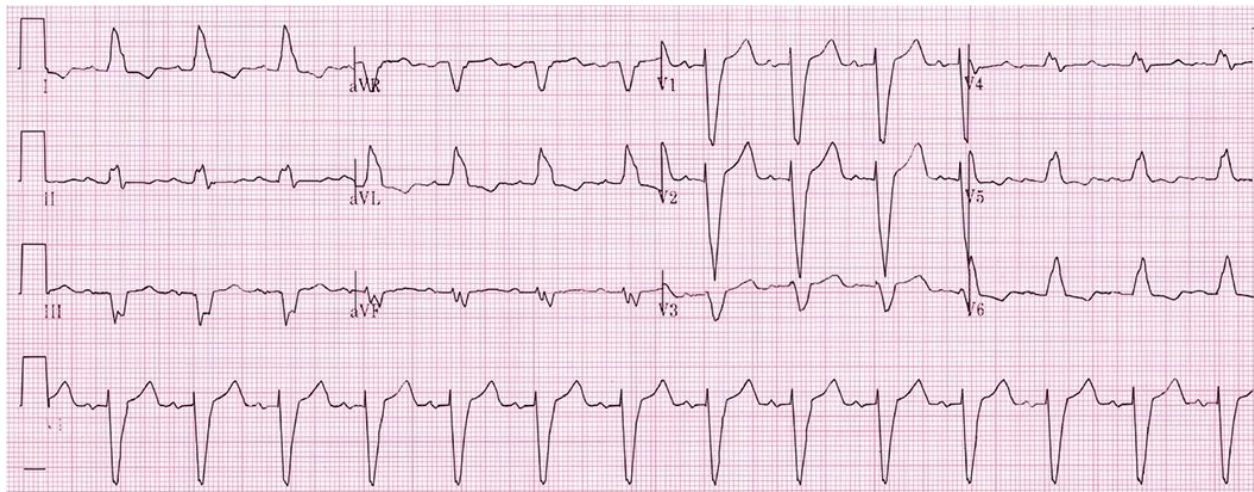


FIGURE 23.20A ■ **Left Bundle Branch Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Wide QRS complex > 120 ms
- Dominant S wave in V₁-V₃
- Broad monophasic, M-shaped or notched R wave in lateral leads (I, aVL, V₅-V₆)
- Appropriate discordance: the ST segment and T wave are in opposite direction to the main vector of the QRS complex

- Left axis deviation

Pearls

1. In left bundle branch block (LBBB), conduction through the left bundle is delayed, whereas depolarization through the right bundle occurs at normal speed. This produces tall, M-shaped or notched R waves in the lateral leads and deep S waves in the right precordial leads.
2. The presence of an LBBB makes it challenging to diagnose an STEMI, as the typical changes such as ST elevation, ST depressions, and T-wave inversions are present. In the past, a new LBBB was considered an STEMI equivalent requiring emergent treatment. More recent data have revealed that new LBBB due to an acute MI is rare in the absence of shock, and therefore, this was removed from the STEMI criteria. Instead, use the Sgarbossa criteria to help diagnose STEMI in the presence of LBBB.

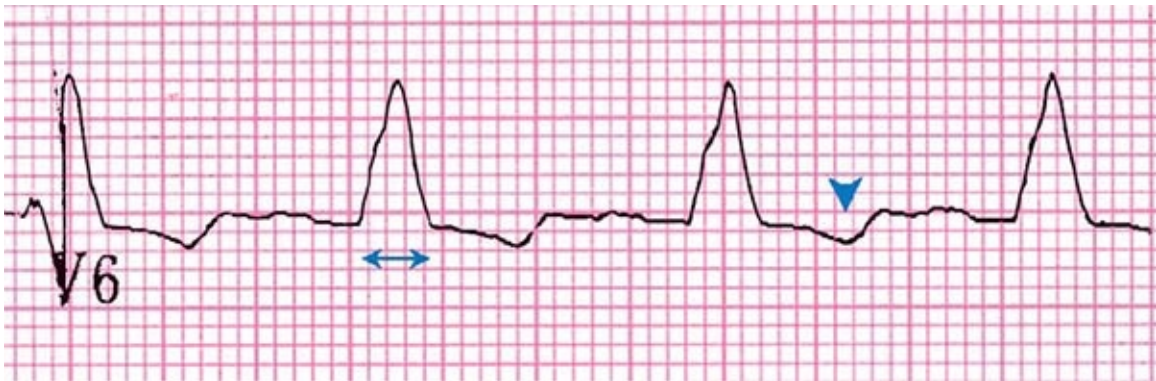


FIGURE 23.20B ■ The QRS is wider than 120 ms (double arrow). The T-wave deflection is in the opposite direction from the QRS deflection (arrowhead).

LEFT ANTERIOR FASCICULAR BLOCK

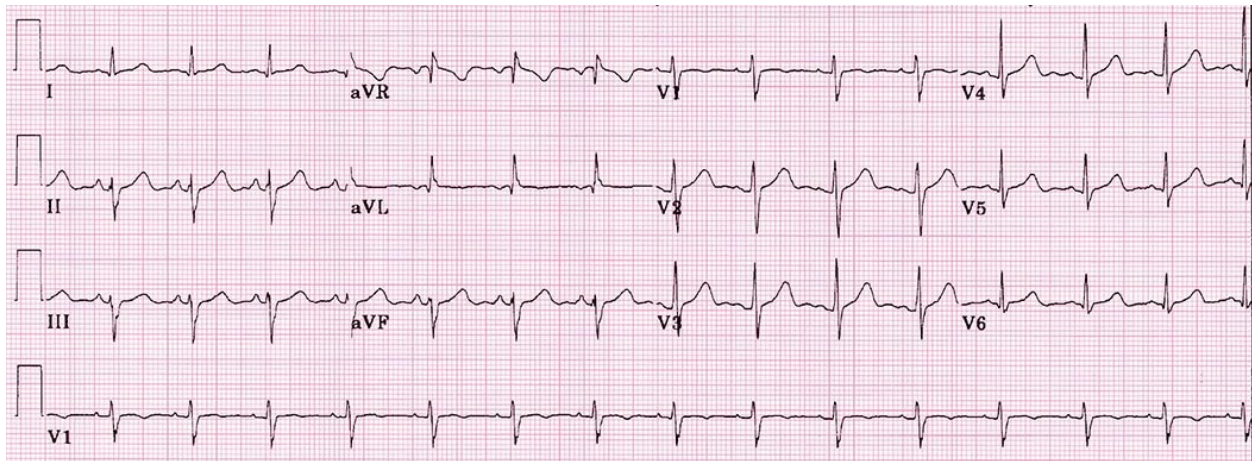


FIGURE 23.21A ■ **Left Anterior Fascicular Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- QRS duration normal or slightly prolonged
- Left axis deviation (without any other reasons for leftward axis on ECG)
- Small R wave and large S wave in the inferior leads (II, III, aVF)
- Small Q with a large R wave in the lateral leads (I, aVL)

Pearls

1. In left anterior fascicular block (LAFB) the conduction through the left anterior fascicle is blocked, which causes the conduction of the high lateral portion of the left ventricle to occur in a delayed fashion, as conduction spreads from the intact left posterior fascicle and the right bundle branch. This causes a typical leftward axis deviation.
2. This finding is more common than left posterior fascicular block and in isolation is generally considered a normal variant and is not considered a bad prognostic finding. It can be seen in anterior MI as well.

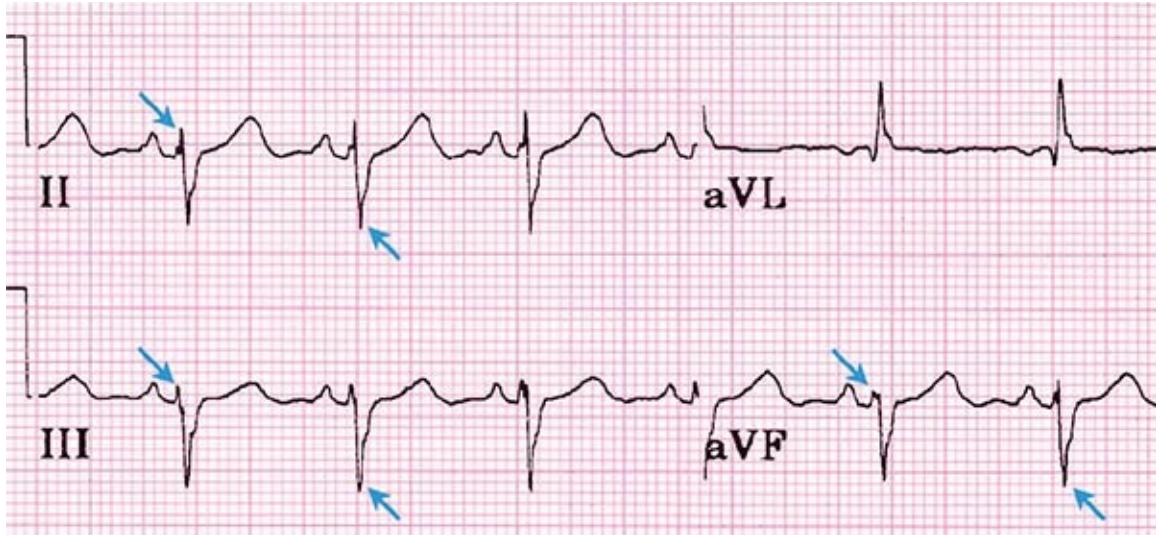


FIGURE 23.21B ■ Small R waves, large S waves in all inferior leads (arrows), with QRS axis deviated left beyond minus 45 degrees.

LEFT POSTERIOR FASCICULAR BLOCK

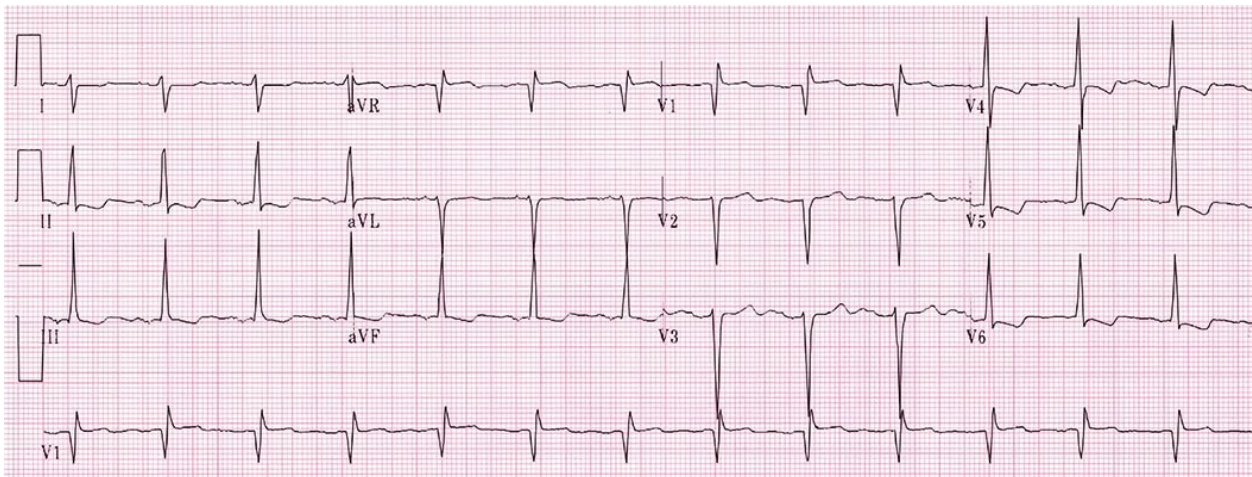


FIGURE 23.22A ■ **Left Posterior Fascicular Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- QRS duration normal or slightly prolonged
- Right axis deviation (without any other reasons for rightward axis on ECG)
- Small R wave and large S wave in the lateral leads (I, aVL)
- Small Q with a large R wave in the inferior leads (II, III, aVF)

Pearls

1. In left posterior fascicular block (LPFB) the conduction through the left posterior fascicle is blocked, which causes the conduction of the inferior portion of the left ventricle to occur in a delayed fashion, as conduction spreads from the intact left anterior fascicle and the right bundle branch. This causes a typical rightward axis deviation.
2. In contrast to the LAFB, this is rarely a normal variant. The posterior fascicle receives its blood supply from both the left coronary and right coronary arteries; hence, if an LPFB is present, this may indicate multi vessel coronary artery disease.

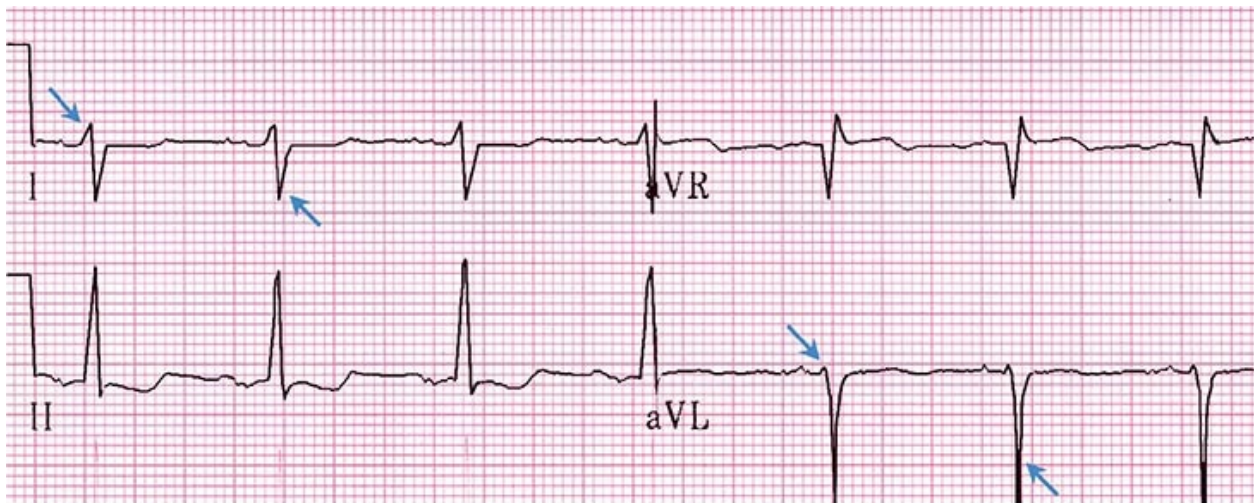


FIGURE 23.22B ■ Small R waves and large S waves in leads I and aVL (arrows).

TRIFASCICULAR BLOCK

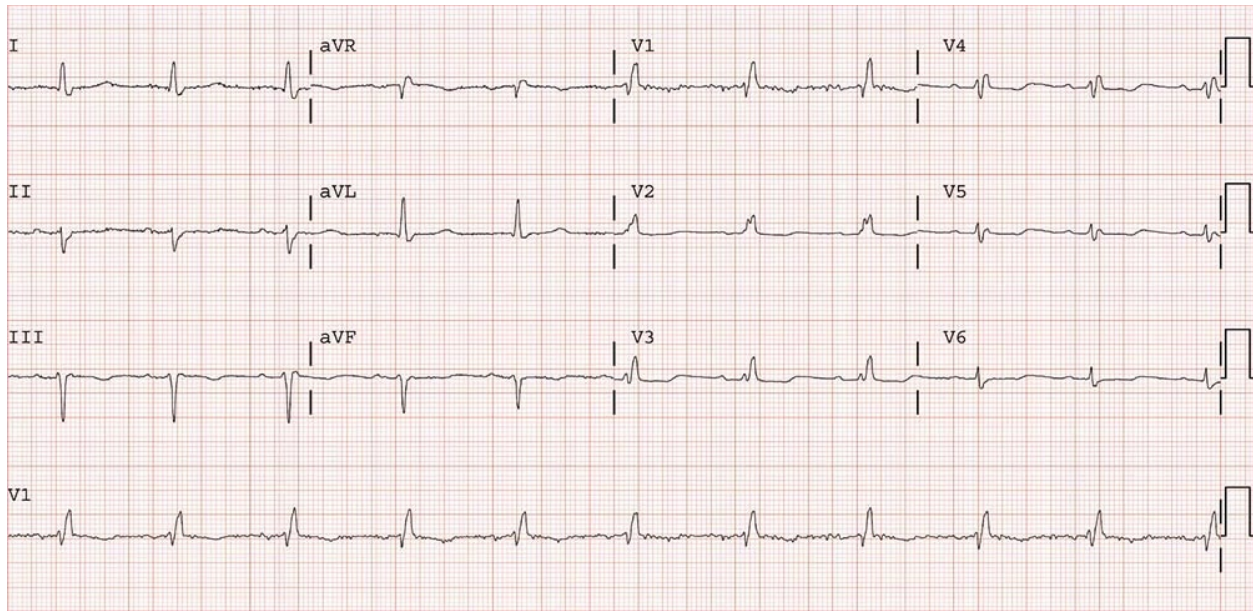


FIGURE 23.23A ■ **Trifascicular Block.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- RBBB with left anterior fascicular block and/or left posterior fascicular block and first- or second-degree AV block.
- Somewhat of a misnomer, this is not a true blocking of three fascicles; rather, two fascicles are blocked coupled with PR interval prolongation.
- LBBB plus first-degree AV block is sometimes referred to as trifascicular block as well, but again, only two fascicles are truly blocked in this scenario.

Pearls

1. A complete trifascicular block, which includes involvement of all three fascicles, is actually a complete or third-degree heart block. An incomplete trifascicular block technically is a bifascicular block (plus AV block) and may be a precursor to complete heart block. Observation and pacemaker evaluation are warranted, especially if symptomatic.
2. AV-blocking agents can potentiate degree of block.
3. Trifascicular block is usually indicative of primary advanced conduction system abnormalities secondary to coronary artery disease.

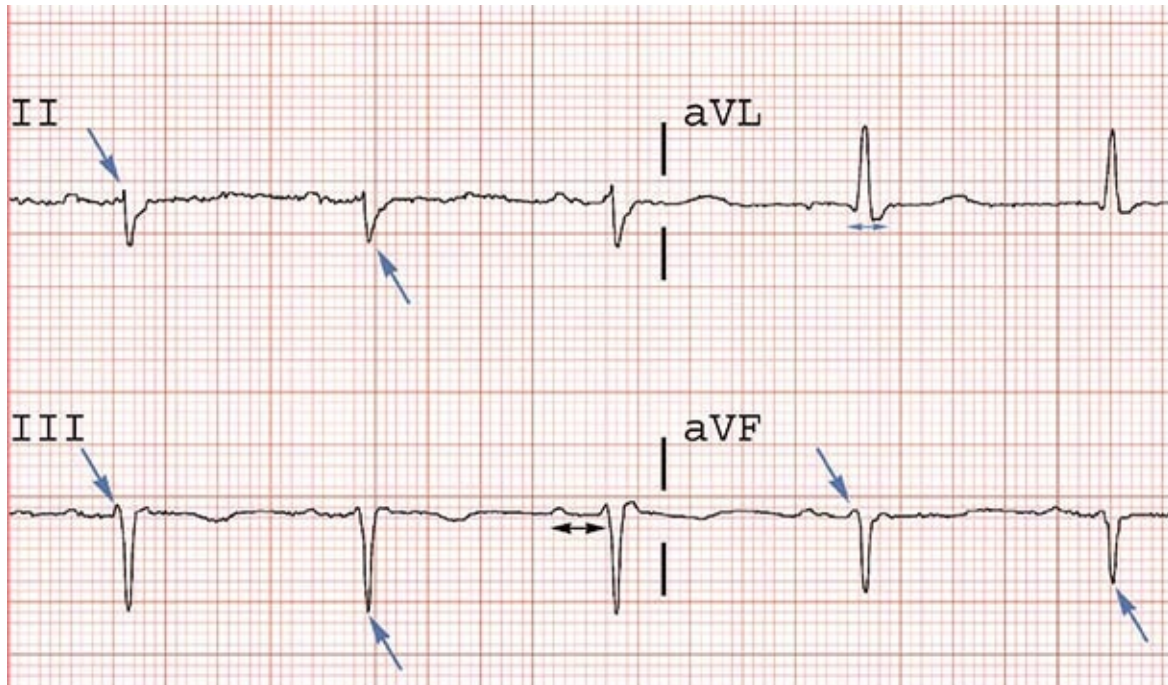


FIGURE 23.23B ■ The QRS is wide with RBBB pattern (blue double arrow). Left anterior fascicular block is present (diagonal arrows) along with a prolonged PR interval and first-degree AV block (black double arrow).

ASHMAN PHENOMENON

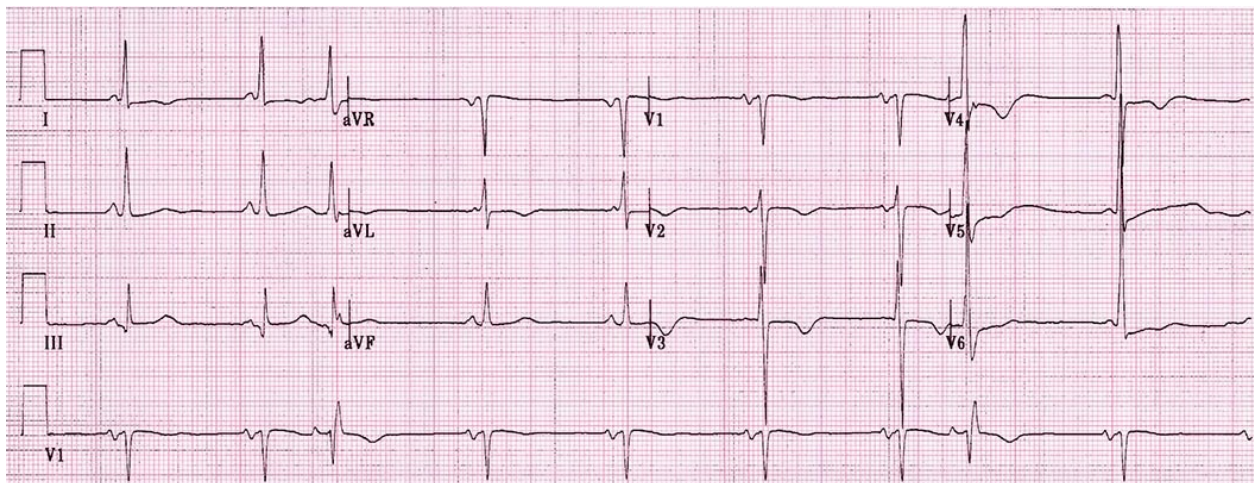


FIGURE 23.24A ■ **Ashman Phenomenon.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Aberrant ventricular conduction, usually with RBBB pattern.

- Altered durations of the refractory period of the bundle branch or ventricular tissue are present, commonly due to atrial fibrillation, atrial ectopy, and atrial tachycardia.

Pearls

1. After depolarization, tissue repolarizes during its refractory period. Refractory period changes with the preceding cardiac cycle, with longer R-R intervals producing longer refractory periods and shorter R-R intervals producing shorter refractory periods.
2. A longer R-R interval lengthens the following refractory period. When an early or premature (ectopic) depolarization reaches the ventricular conduction system before it has completely repolarized, aberrant conduction may occur and be manifest on the ECG with a bundle branch block pattern.
3. Ashman phenomenon most commonly appears with an RBBB pattern, since the right bundle has a longer refractory period than the left bundle.
4. Ashman phenomenon is often seen in atrial fibrillation, when a long R-R interval is followed by a much shorter R-R interval.
5. In the setting of a premature atrial beat (as seen in this example), the earlier in the cycle the premature atrial contraction occurs and the longer the preceding R-R interval is, the more likely aberrant conduction of the beat will occur.



FIGURE 23.24B ■ After a relatively long R-R interval (double arrow), a premature atrial contraction (diagonal arrow) is followed by an aberrantly conducted QRS with RBBB morphology (arrowhead). After a short pause (single arrow), the next beat is conducted normally as it has occurred outside of the refractory period set by the previous beat.

Part 3: Rhythm Disturbances

JUNCTIONAL ESCAPE RHYTHM

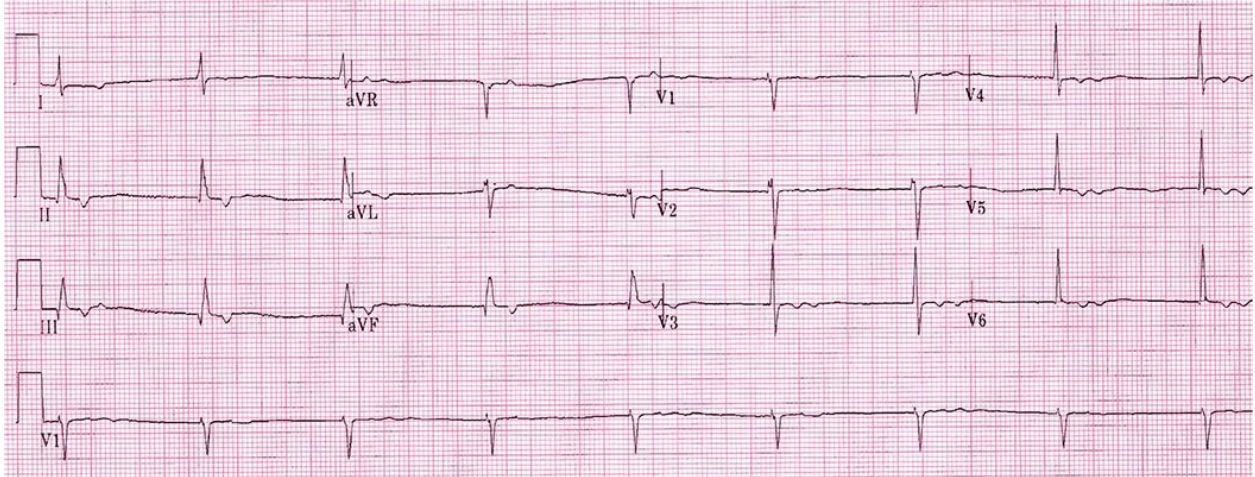


FIGURE 23.25A ■ Junctional Rhythm with Retrograde P Waves. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Bradycardia, typically with a rate of 40 to 60 bpm.
- The QRS complex is narrow, < 120 ms.
- P waves are absent, retrograde, very slow, or unrelated to the QRS complex.

Pearls

1. When the atria fail to initiate a cardiac rhythm, or when no pacing signal reaches the lower AV node, the AV node or His bundle usually picks up the pacemaking responsibility.
2. P waves may be conducted retrograde and buried in the T wave, as seen in this example.
3. If a bundle branch block is also present, the QRS may be wide and may be difficult to discern from a primary ventricular rhythm.



FIGURE 23.25B ■ The QRS is narrow. P waves are not present before the QRS. In this example, the signal that originated in the His bundle is conducted retrograde through the AV node into the atria, and retrograde P waves are apparent in the ST segment (arrows).

VENTRICULAR ESCAPE RHYTHM

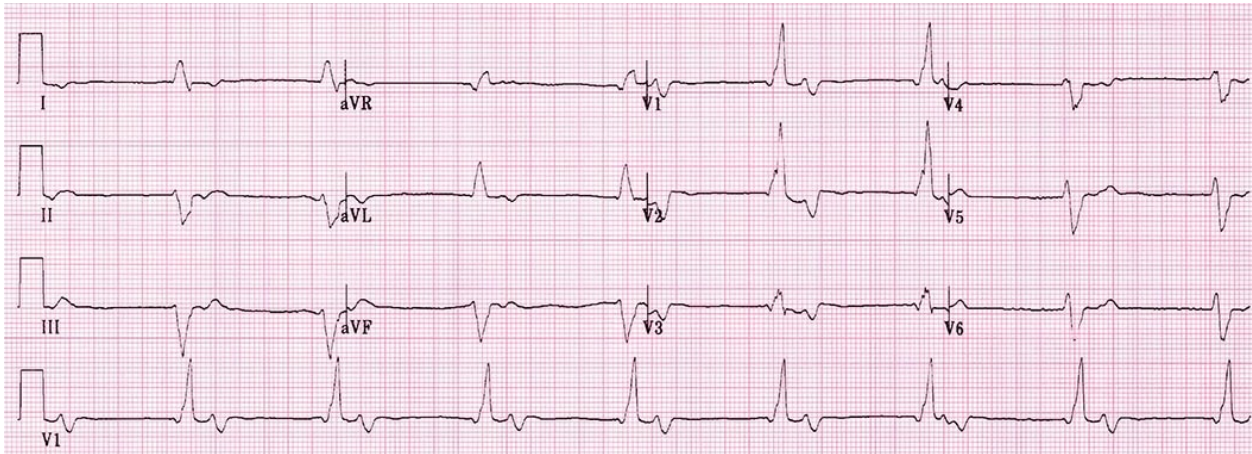


FIGURE 23.26A ■ Ventricular Rhythm with Retrograde P Waves. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Bradycardia, typically with a rate of 20 to 40 bpm.
- The QRS complex is wider than 120 ms.
- Can have LBBB or RBBB morphology.

Pearls

1. When the atria and the AV node fail to initiate a cardiac rhythm, or when no pacing signal reaches the ventricle, the ventricular tissue usually picks up the pacemaking responsibility.
2. P waves may also be conducted retrograde and buried in the T wave.



FIGURE 23.26B ■ Wide-complex (double arrow) regular QRS at a rate of approximately 50 bpm. Retrograde P waves are seen in this example (arrow).

PACED RHYTHM

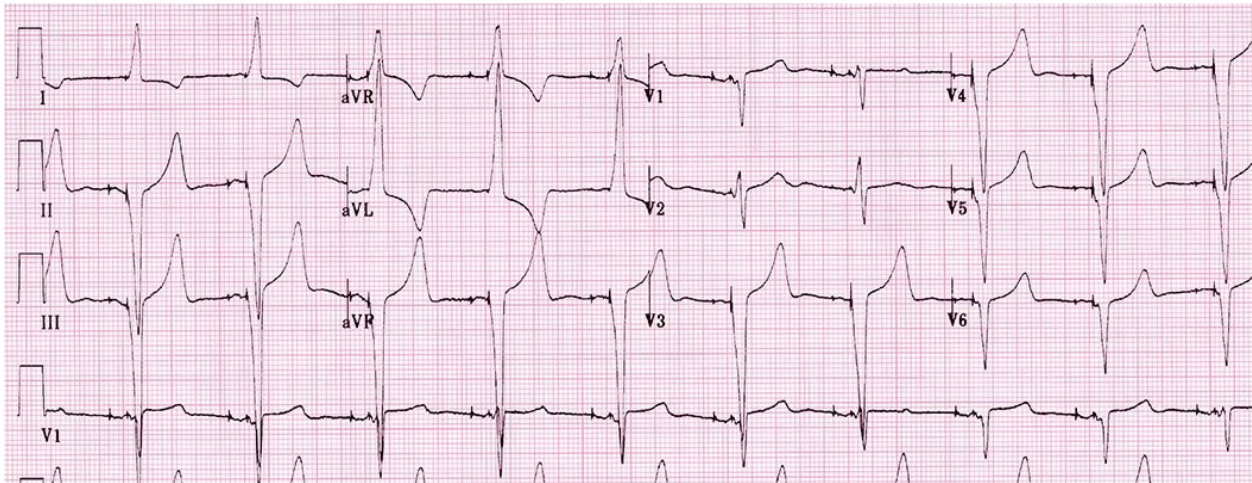


FIGURE 23.27A ■ Dual-Chamber Pacemaker, Paced Rhythm. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Pacing spikes.
- If a pacer spike is seen preceding a P wave, the patient is atrial paced. This can be intermittent.
- If a pacer spike is seen preceding a QRS complex, the patient is ventricular paced. This can be intermittent. The QRS complex will typically be wide with a QRS duration > 120 ms. Right ventricle pacing leads will typically give an LBBB morphology, while left ventricle pacing leads will give an RBBB morphology.

Pearls

1. Pacemakers are designated by the “five letter” system. In this system, the letter “A” denotes atrium, “V” denotes ventricle, “D” denotes dual (both chambers), and “O” denotes neither. The first three letters are the most commonly used:
 - A. First letter—designates chamber(s) paced
 - B. Second letter—designates chamber(s) sensed
 - C. Third letter—designates pacemaker response to sensed electrical activity:
 - T: triggered—fires even when beat sensed, I: inhibitory—holds when beat

sensed, D: dual— atrial triggered and ventricle inhibited

D. Fourth letter—extra options: P: programmable, M: multiprogrammable, C: communicating, R: rate adaptation, O: none

E. Fifth letter—cardioverting options: P: pacing, S: shocking, D: dual (P+S), O: none

2. The two most common pacemaker malfunctions are failure to pace and failure to sense.
3. The absence of paced complexes does not mean pacemaker malfunction if the pacemaker is set to inhibitory mode. When a normal native complex is detected within a preset time frame, the pacemaker will remain silent when it detects a native beat.
4. Some ECG machines perceive small pacer spikes as artifact and do not reproduce them on the printed tracing.

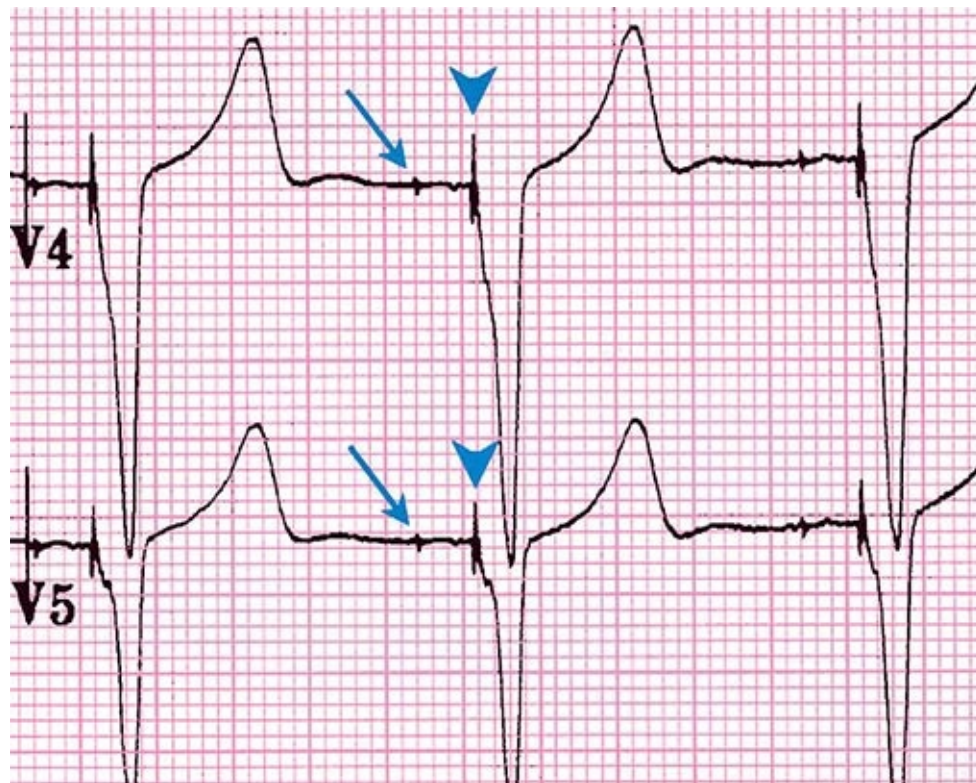


FIGURE 23.27B ■ Tiny pacer spikes (arrows) precede the P waves, and somewhat larger pacer spikes precede the QRS complexes (arrowheads). The QRS complexes are wide, with discordant T waves.

ATRIAL FIBRILLATION

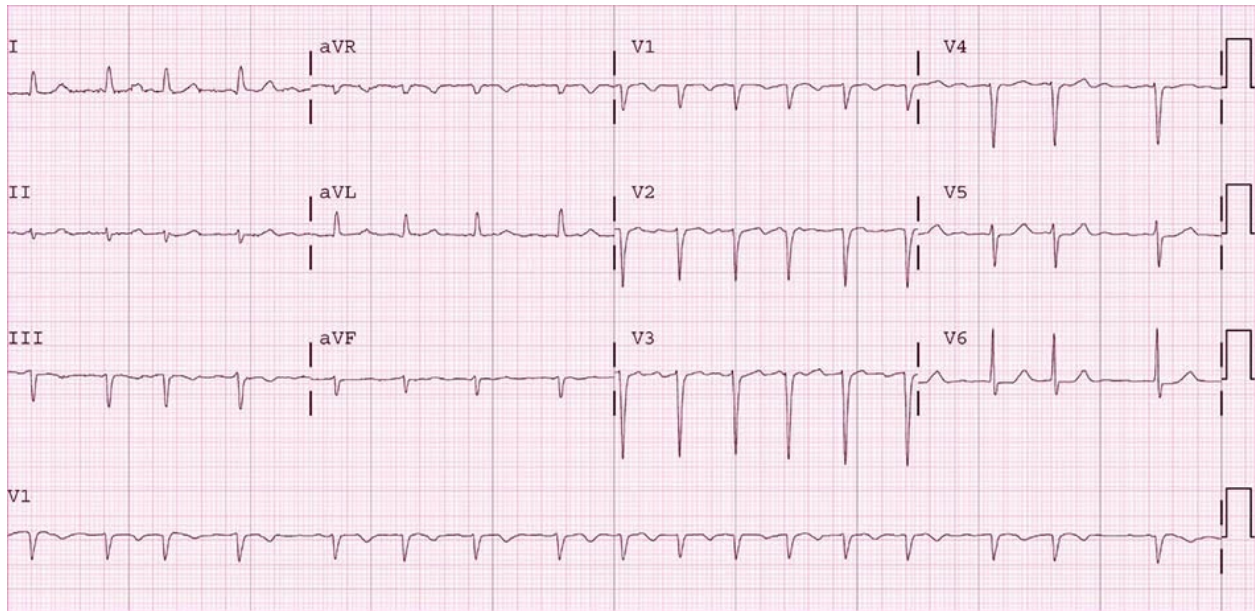


FIGURE 23.28A ■ **Atrial Fibrillation.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- Irregularly irregular narrow-complex tachycardia
- Absence of P-waves, but fibrillatory atrial activity may be seen

Pearls

1. Atrial fibrillation is typically a narrow-complex rhythm, but in presence of underlying bundle branch block, aberrant conduction, or accessory pathway, it may be wide complex.
2. Most “irregularly irregular” rhythms are due to atrial fibrillation, but other rhythms may produce similar findings. These include multifocal atrial tachycardia, atrial flutter with variable AV block, and frequent premature ventricular contractions.
3. Hemodynamically stable patients are treated with chemical rate or rhythm control with a goal heart rate of < 120 bpm. In hemo-dynamically unstable patients, synchronized cardioversion is the treatment of choice. Risks of cardioversion include subsequent stroke.
4. When a very fast (>200 bpm) irregular tachycardia is seen with changing QRS shape and morphology, suspect atrial fibrillation with Wolff-Parkinson-White syndrome (WPW) and avoid AV nodal blocking agents as this can promote conduction over the accessory pathway and lead to ventricular tachycardias

and death.



FIGURE 23.28B ■ R-to-R interval varies in an “irregularly irregular” pattern (double arrows). The baseline “rumble,” representing “F waves,” may be very fine or even indiscernible.



FIGURE 23.28C ■ The baseline “rumble” may be very coarse, resembling atrial flutter waves.

ATRIAL FLUTTER

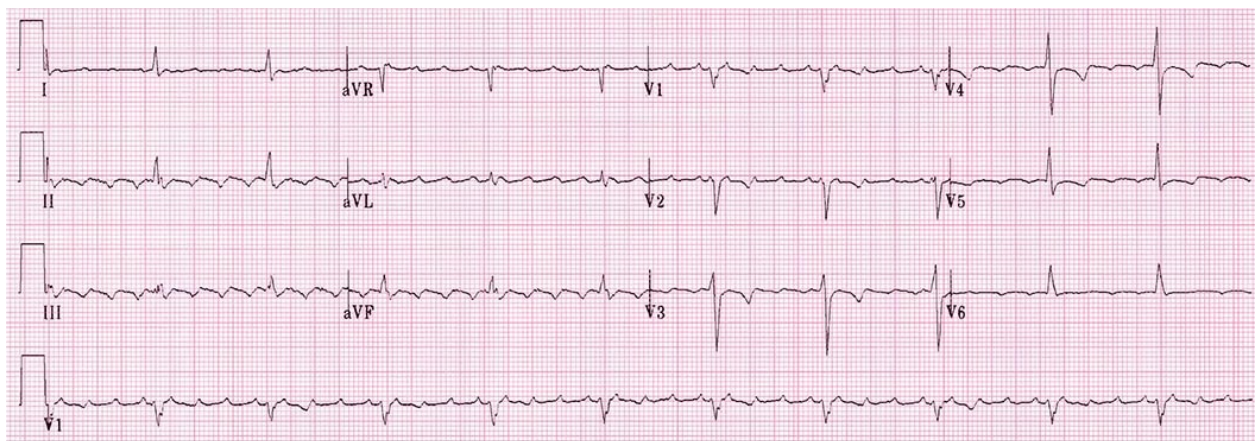


FIGURE 23.29A ■ **Atrial Flutter.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Regular narrow-complex tachycardia.

- Flutter waves appear in a rapid sine wave or “sawtooth” pattern, usually in the inferior leads (II, III, aVF).
- Atrial activity in lead V₁ often appears as rapid P waves at a rate approximating 300 bpm.

Pearls

1. The AV node’s refractory period usually prevents 1:1 conduction to the ventricles. Usually, conduction is blocked at a ratio of 1:2 to 1:4. The QRS complexes should appear with regular periodicity. However, AV conduction may be variable from beat to beat, creating irregular R-to-R intervals.
2. A conduction ratio of 2:1 is usually difficult to discern, because the two flutter peaks between QRS complexes may look like normal P and T waves. A ventricular rate of 140 to 160 bpm should prompt consideration of the possibility of atrial flutter with 2:1 block.
3. Atrial flutter tends to be more refractory to chemical cardioversion and more susceptible to electrical cardioversion when compared to atrial fibrillation.



FIGURE 23.29B ■ Atrial flutter with 4:1 block. The flutter waves (arrows marking every other flutter wave) may be mistaken for P and T waves.

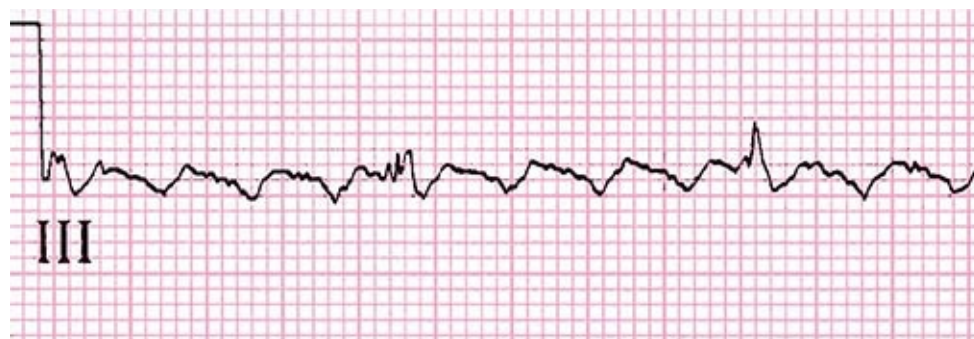


FIGURE 23.29C ■ The “sawtooth” pattern is most apparent in the inferior leads.

MULTIFOCAL ATRIAL TACHYCARDIA

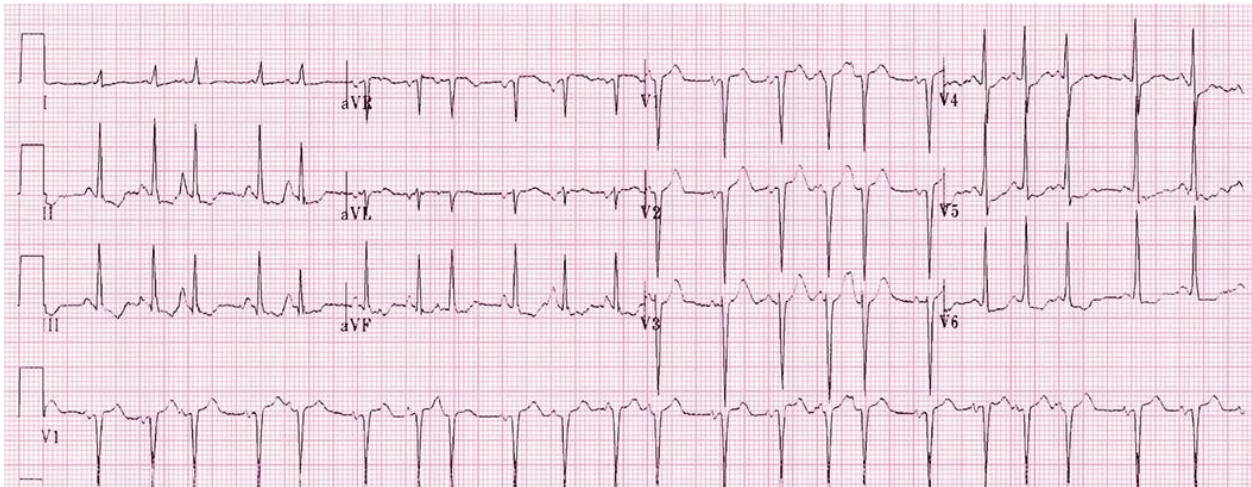


FIGURE 23.30A ■ **Multifocal Atrial Tachycardia.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Irregularly irregular narrow-complex tachycardia at a rate > 100 bpm
- Multiple P-wave morphologies, with at least three distinct P-wave morphologies in the same lead
- Varying PP, PR, and RR intervals

Pearls

1. Multiple atrial foci are capable of acting as pacemakers. When irritated by stretching, medications, or certain acute medical conditions, these foci compete in pacing the atria.
2. The different atrial foci produce P waves of different morphologies.
3. Since the atrial foci vary in distance to the AV node, PR intervals vary.
4. Multifocal atrial tachycardia (MAT) usually results from exacerbation of another condition that produces distention or irritation of the atria. The most common cause of MAT is chronic obstructive pulmonary disease (COPD) exacerbation. Treatment of the underlying condition should correct the arrhythmia.
5. Even though MAT itself is not usually fatal, it is a poor prognostic sign.



FIGURE 23.30B ■ Multiple P morphologies (arrowheads), varying PR intervals (lower double arrows), and varying R-R intervals (upper double arrows) with heart rate greater than 100 bpm.

SUPRAVENTRICULAR TACHYCARDIA (SVT)

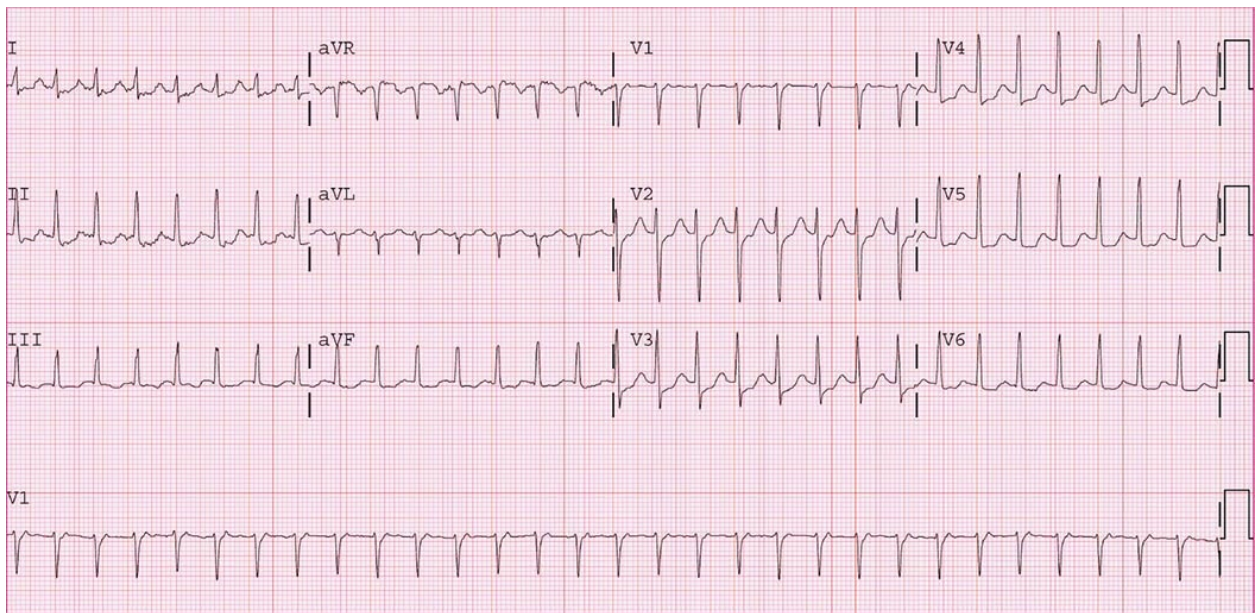


FIGURE 23.31A ■ **Supraventricular Tachycardia (AVNRT).** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- Narrow complex, regular tachycardia, rate usually > 140 bpm.
- Absent, retrograde, or unusual P waves.
- P waves may be buried in the QRS complex.

Pearls

1. SVT occurs when the SA node rhythm is superseded by a faster rhythm, usually originating in the AV node.
2. Three common types are:
 - A. Atrial tachycardia—originates from an ectopic focus in the atrium. P waves may have an unusual morphology or may be hidden by the preceding T wave.
 - B. AV nodal reentrant tachycardia (AVNRT) occurs when an electrical impulse reenters the AV node in a circular pattern causing rapid depolarizations of the ventricles. Since the AV node is the origin of the atrial depolarization, the P-wave deflection should be inverted if seen (eg, downgoing in II, III, aVF).
 - C. AV reentrant tachycardia (AVRT) as seen with bypass tracts outside of the AV node (WPW). Instead of intra-nodal reentrant activity as seen with AVNRT, an accessory tract provides the reentrant pathway to propagate the tachycardia.



FIGURE 23.31B ■ A narrow-complex tachycardia, with no clear P waves preceding the QRS. R-R intervals are regular (double arrows), differentiating this from fine atrial fibrillation. This rhythm converted to a normal sinus rhythm after the administration of intravenous adenosine.

SVT WITH ABERRANCY

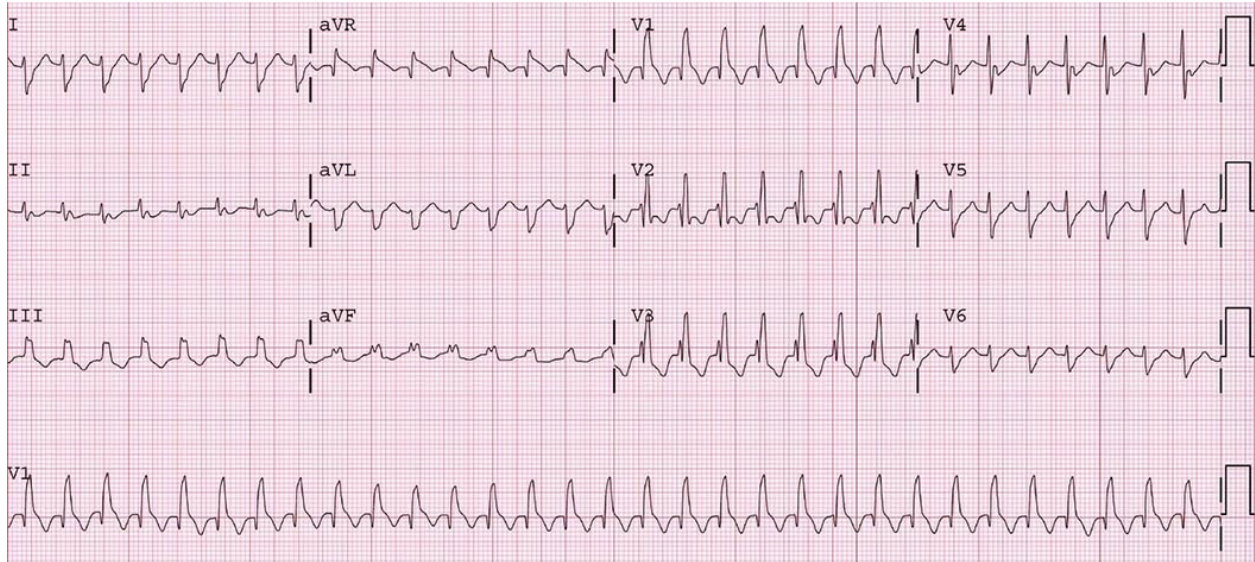


FIGURE 23.32A ■ Supraventricular Tachycardia with Aberrant Conduction, Underlying RBBB.
(ECG contributor: Walter Clair, MD.)

ECG Findings

- Wide complex, regular tachycardia (usually > 140 bpm)
- QRS morphology consistent with one of the bundle branch block patterns

Pearls

1. The rapid rate of an SVT may “outrun” the ventricular conducting system’s ability to repolarize quickly, producing a rate-related bundle branch block. The signal then must propagate cell to cell, producing a wide-complex tachycardia. A typical bundle branch pattern usually results.
2. When a person with a chronic wide-complex (aberrant) bundle branch block enters an SVT, the ECG will display a wide-complex, regular tachycardia. However, when the patient does not have prior ECGs, it can be difficult to distinguish SVT with aberrancy from a ventricular tachycardia (VT). Many different criteria have been proposed to distinguish the two, but unfortunately, most are neither extremely sensitive nor specific. When in doubt, treat as a VT, as many of the typical SVT pharmacologic agents are detrimental in patients with VT and can lead to ventricular fibrillation and death.

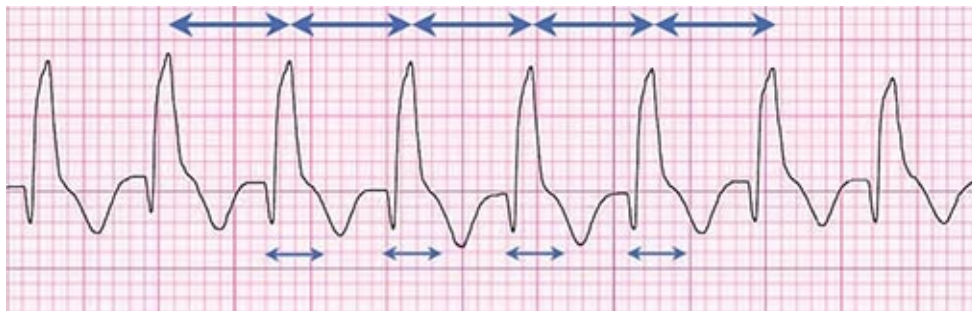


FIGURE 23.32B ■ Wide-complex tachycardia with a rate of 188 bpm. This patient has sudden onset of SVT with a known underlying RBBB. QRS complexes are wide (lower double arrows) and R-R intervals are regular (upper double arrows).



FIGURE 23.32C ■ Wide-complex tachycardia at approximately 150 bpm. The R-R interval is regular, except for one pause, when characteristic atrial flutter waves are apparent (arrowhead).

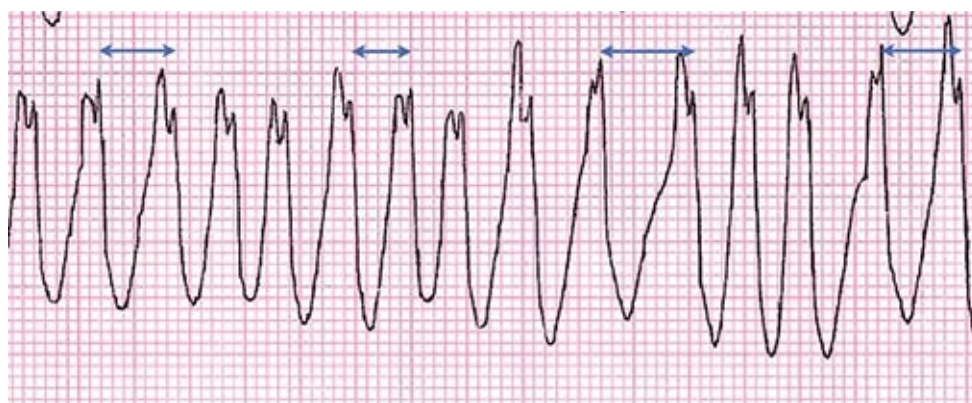


FIGURE 23.32D ■ Irregularity in the R-R interval, as seen most easily in the baseline (double arrows), strongly suggests the presence of rapidly conducted atrial fibrillation with aberrancy.

VENTRICULAR TACHYCARDIA

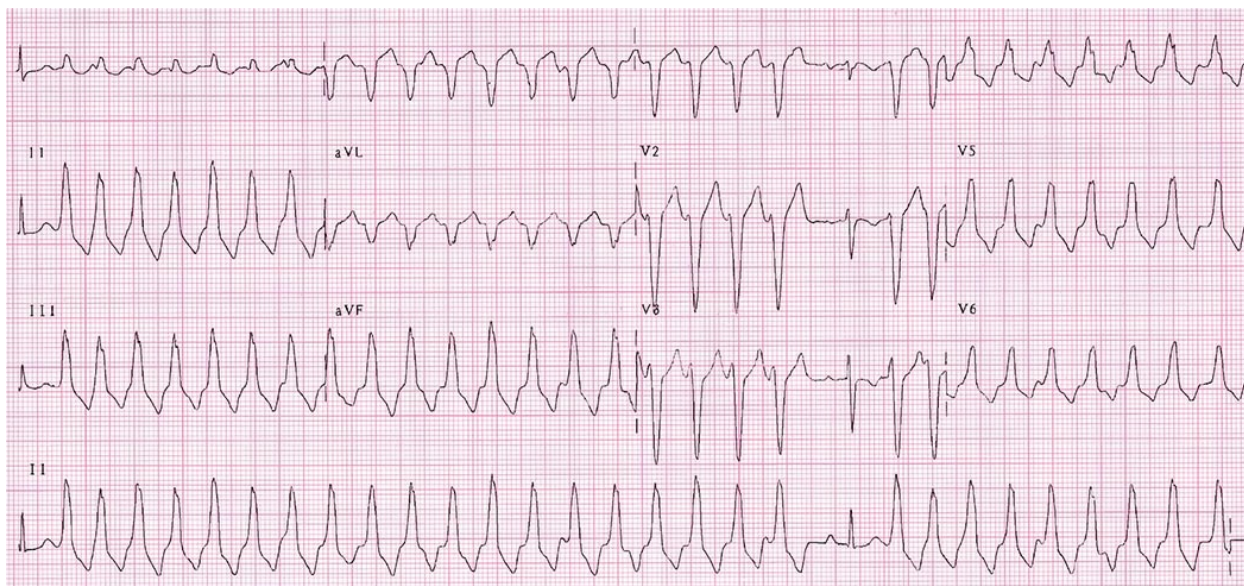


FIGURE 23.33A ■ Ventricular Tachycardia with Capture Beat. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Wide, regular tachycardia (usually > 120 bpm).
- Evidence of AV dissociation may be found: P waves may appear periodically in the T wave or baseline.
- “Capture” beats may occur if atrial depolarization occurs prior to the intrinsic firing of the ventricle.
- “Fusion” beats may occur if atrial depolarization passes through the AV node at the same time as the intrinsic ventricle depolarization, producing a QRS that appears to be different or narrower than the other QRS complexes.

Pearls

1. It can be difficult to distinguish VT from SVT with aberrancy. When in doubt, treat as VT.
2. Patient factors that make VT more likely include history of coronary artery disease, congestive heart failure, and advanced age.
3. ECG features that increase likelihood of VT include absence of typical RBBB or LBBB morphology, extreme axis deviation, AV dissociation, presence of capture beats, presence of fusion beats, Brugada sign (distance of onset of the QRS complex to the nadir of the S wave is > 100 ms), positive or negative

concordance in the chest leads, Josephson's sign (notching near the nadir of the S wave), or R-wave peak time > 50 ms in lead II.

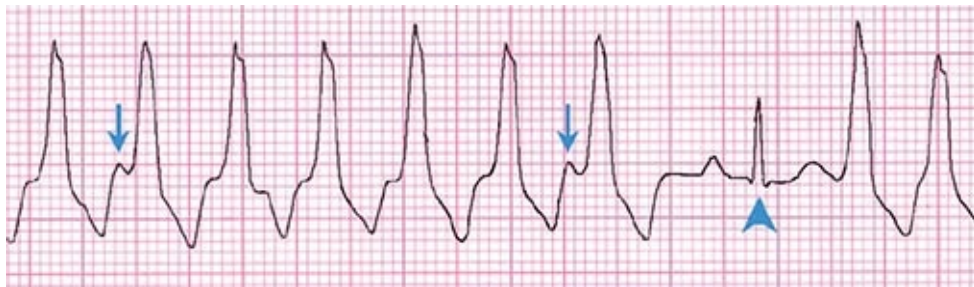


FIGURE 23.33B ■ A wide-complex tachycardia. AV dissociation is apparent, as P waves occasionally appear superimposed in the ST segment or just prior to the QRS (arrows). A capture beat occurs following a lapse in the VT (arrowhead).

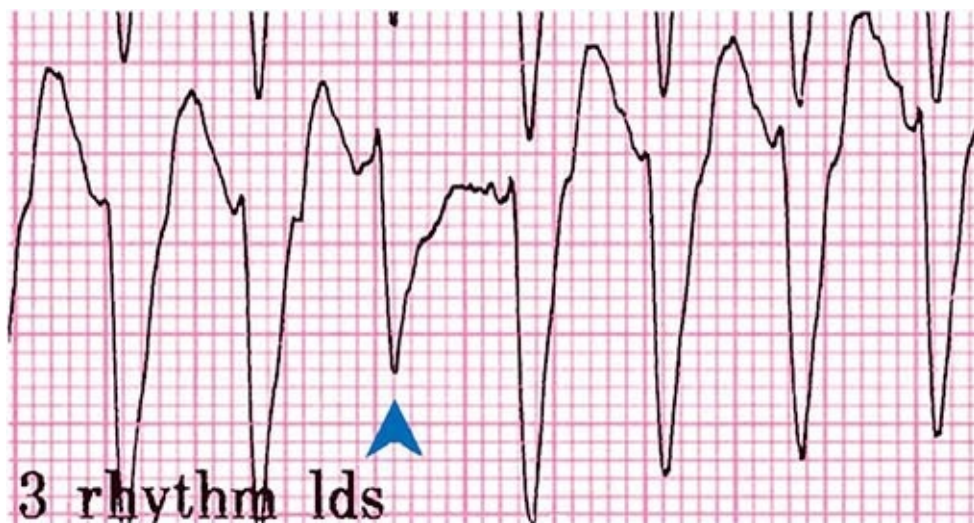


FIGURE 23.33C ■ Another example of ventricular tachycardia, featuring a fusion beat (arrowhead). (ECG contributor: Marc Mickiewicz, MD.)

VENTRICULAR FLUTTER

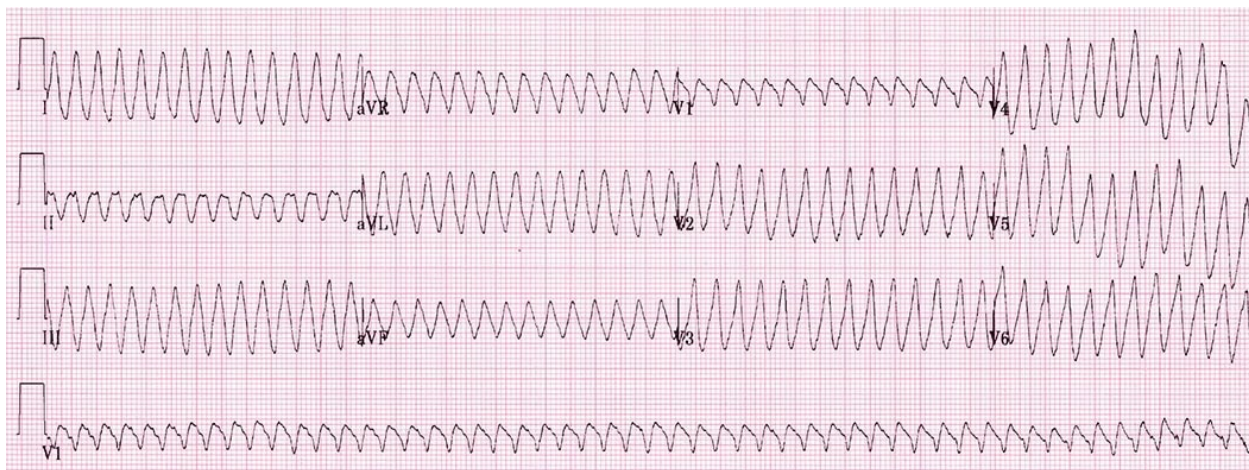


FIGURE 23.34A ■ **Ventricular Flutter.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Tachycardia with a wide monomorphic QRS complex.
- Ventricular rate may be very rapid (300 bpm).
- Sine wave appearance with regular large oscillations.

Pearls

1. Imagine an atrial flutter sawtooth with much larger amplitude.
2. When you see a very rapid wide-complex tachycardia (> 240 bpm), consider ventricular flutter or WPW with atrial fibrillation or flutter.
3. WPW with atrial flutter may be indistinguishable from ventricular flutter.
4. Ventricular flutter is treated as VT and usually leads to ventricular fibrillation if not promptly corrected with antiarrhythmic medications or electrical cardioversion.
5. Patients with such a rapid rate are almost always unstable. Emergent cardioversion is indicated. If the patient appears to be stable enough for chemical cardioversion, choose a medication that is safe to use with WPW, such as procainamide or amiodarone.

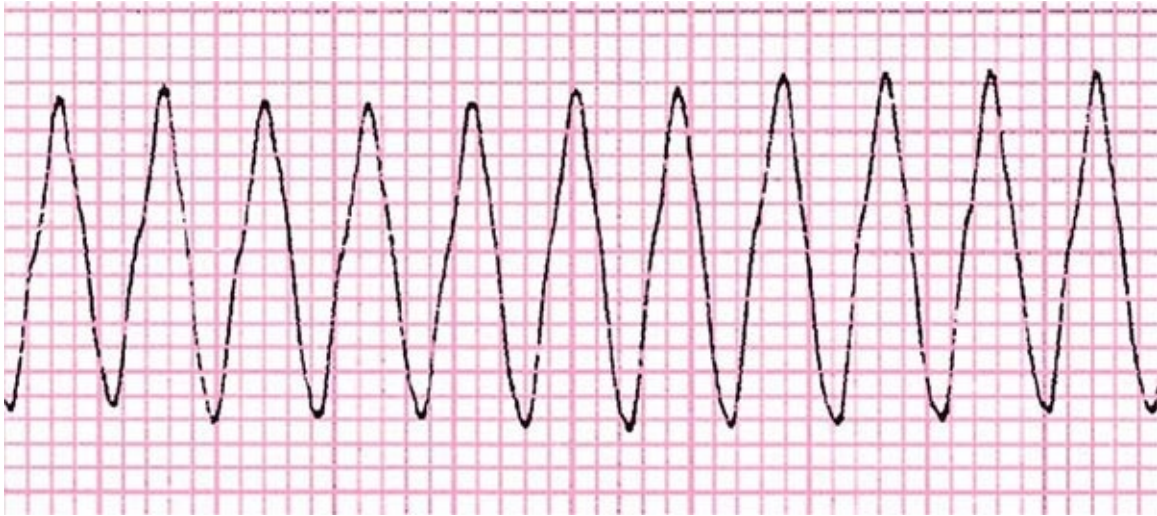


FIGURE 23.34B ■ Very rapid, regular, wide-complex tachycardia with sine wave appearance. The rate in this example is 330 bpm. Differential diagnosis includes WPW with atrial flutter.

POLYMORPHIC VENTRICULAR TACHYCARDIA



FIGURE 23.35A ■ **Torsades de Pointes.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Wide-complex irregular, extreme tachycardia
- QRS complexes with changing morphology

Pearls

1. Polymorphic ventricular tachycardia (PVT) may occur in association with a normal QT interval or a prolonged QT interval. When associated with a prolonged QT interval, torsades de pointes is diagnosed.
2. Normal-QT PVT is typically associated with cardiac ischemia. Immediate treatment consists of cardioversion; ventricular antiarrhythmics such as amiodarone, procainamide, or lidocaine may be used followed by assessment for and treatment of ischemia.
3. Torsades de pointes (literally translated “twisting of the points” in French) has a characteristic “twisting” appearance of the QRS complexes around the ECG baseline, as shown in [Figs. 23.24A-B](#).
4. Immediate treatment for torsades de pointes consists of cardioversion; if cardioversion is unsuccessful, a bolus of intravenous magnesium should be given. If the arrhythmia persists, overdrive electrical pacing or overdrive chemical pacing (with isoproterenol) will help shorten the relative QT and convert the patient back to sinus rhythm. Following conversion, a magnesium infusion should be initiated, and the cause of the prolonged QT should be aggressively sought out and treated.



FIGURE 23.35B ■ Very rapid wide-complex tachycardia with sine wave appearance and fluctuations in the amplitude of the QRS complexes consistent with torsades de pointes.

VENTRICULAR FIBRILLATION



FIGURE 23.36A ■ Ventricular Fibrillation. (ECG contributor: Frank G. Yanowitz, MD. Used with permission from Frank G. Yanowitz, MD at the ECG Learning Center [<https://ecg.utah.edu>].)

ECG Findings

- Chaotic, irregular deflections without clear P waves, QRS complexes, or T waves
- Rate up to 600 bpm

Pearls

1. This rhythm is not compatible with effective cardiac output; hence, the patient will be in pulseless cardiac arrest. Immediate defibrillation is indicated.
2. Given the above, ventricular fibrillation should generally not be identified on ECG, but rather by rhythm strip and clinical picture (similar to the principle of tension pneumothorax on chest x-ray). The examples seen here were obtained when patients receiving continuous ECG monitoring fibrillated during stress testing.
3. Amplitude of deflections often decreases over time (coarse or fine ventricular fibrillation).

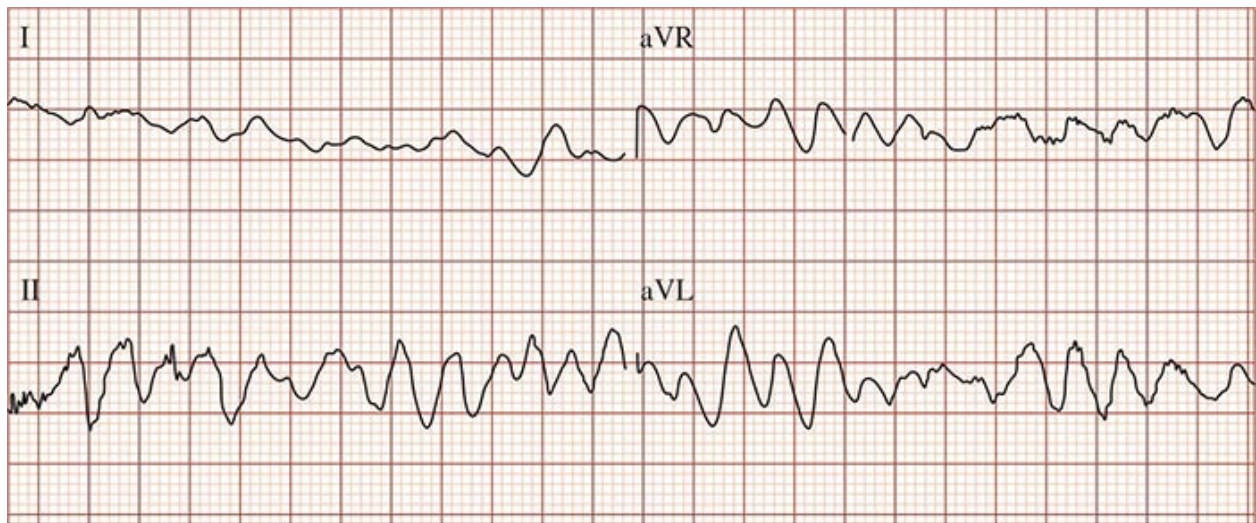


FIGURE 23.36B ■ Note the chaotic baseline with no discernable complexes. Immediate defibrillation is indicated. (ECG contributor: Jason Winter. Used with permission from <https://ecg-educator.blogspot.com/?m=0>.)

Part 4: Structural Abnormalities

DEXTROCARDIA

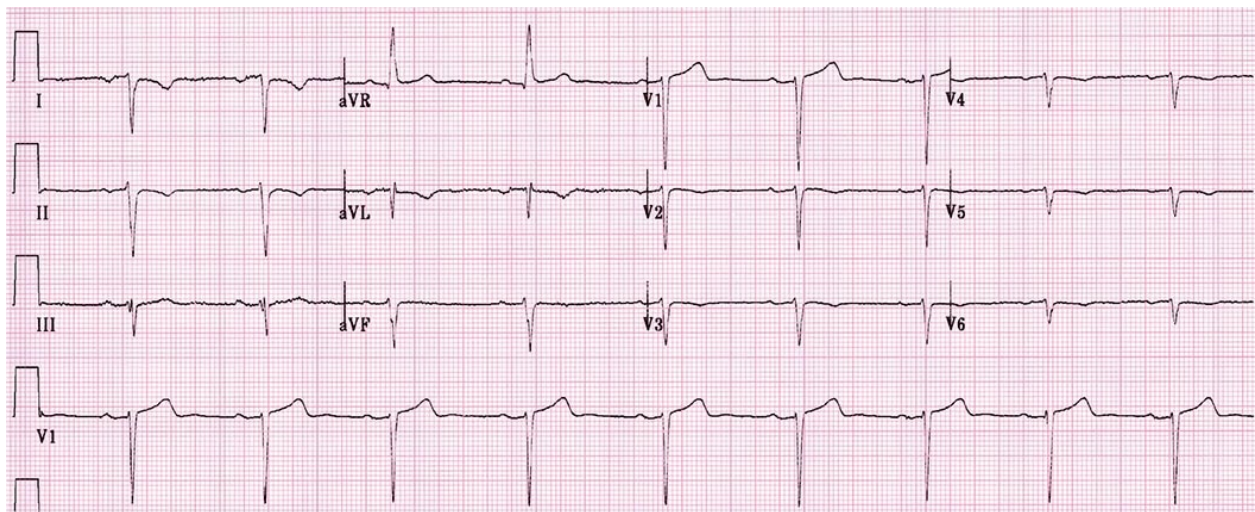


FIGURE 23.37A ■ **Dextrocardia.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Rightward axis.
- Positive QRS complex in aVR.

- Inversion of the entire complex in lead I.
- Loss of R wave progression.
- QRS deflections in V_4 to V_6 are small and downgoing.

Pearls

1. The orientation of the heart in the chest cavity is reversed with the predominant electrical activity moving left to right (as opposed to right to left).
2. Normally placed precordial leads in a patient with dextrocardia are actually placed over the thinner right ventricle instead of the left ventricle.
3. Reversing all ECG leads should produce an essentially normal ECG.
4. A “reversed” lead I (“downward” QRS) and “reversed” lead aVR (“upward” QRS) with normal-appearing V leads strongly suggests limb lead reversal.



FIGURE 23.37B ■ The P wave, QRS, and T wave are downgoing in lead I. Differential diagnosis includes limb lead reversal and dextrocardia. The 12-lead ECG above represents dextrocardia as evidenced in the abnormal precordial leads.

LEFT VENTRICULAR HYPERTROPHY

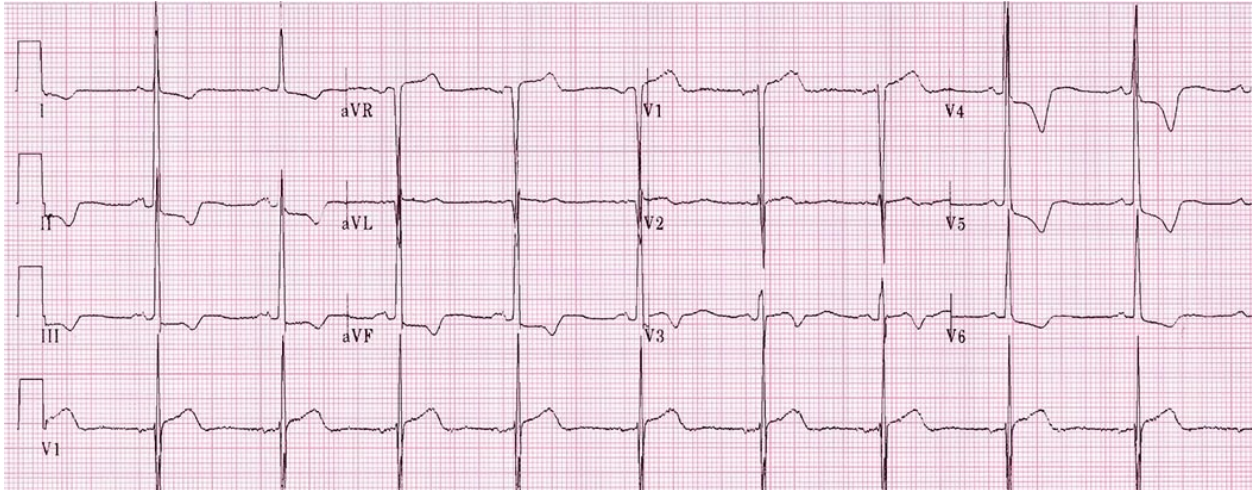


FIGURE 23.38A ■ Left Ventricular Hypertrophy with Strain Pattern. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Largest R wave plus largest S wave in precordial leads > 45 mm
- R wave in V_4 , V_5 , $V_6 > 26$ mm
- S wave in V_1 + R wave in V_5 or $V_6 > 35$ mm
- R wave in $aVL > 11$ mm
- R wave in $aVF > 20$ mm
- S wave in $aVR > 14$ mm
- R wave in I + S wave in III > 25 mm
- ST depression and T-wave inversion in left-sided leads
- Left axis deviation

Pearls

1. LVH is often a sign of disease states such as systemic hyper-tension or aortic stenosis.
2. ST elevations seen in LVH can be confused as myocardial ischemia. There are no clear criteria to distinguish the two; the clinical presentation is of essence, as well as comparison to old ECGs and serial ECGs.
3. LVH may manifest on the ECG in many different ways. Several different systems for diagnosing LVH by ECG have been promoted (listed earlier), but none have been shown to be perfectly sensitive or specific.

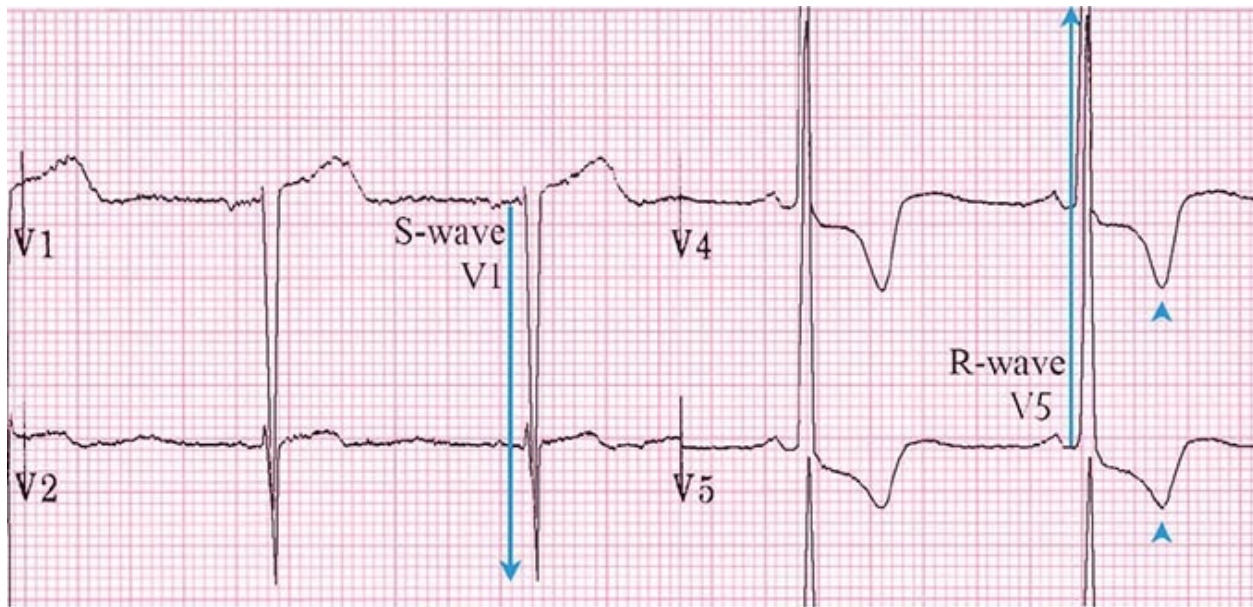


FIGURE 23.38B ■ The QRS deflections are very large. The R wave in V₅ plus the S wave in V₁ total approximately 75 mm (arrows). ST downsloping to inverted T waves in V₄ and V₅ (arrowheads) may also be seen, a finding often referred to as “LVH with strain.”

RIGHT VENTRICULAR HYPERTROPHY

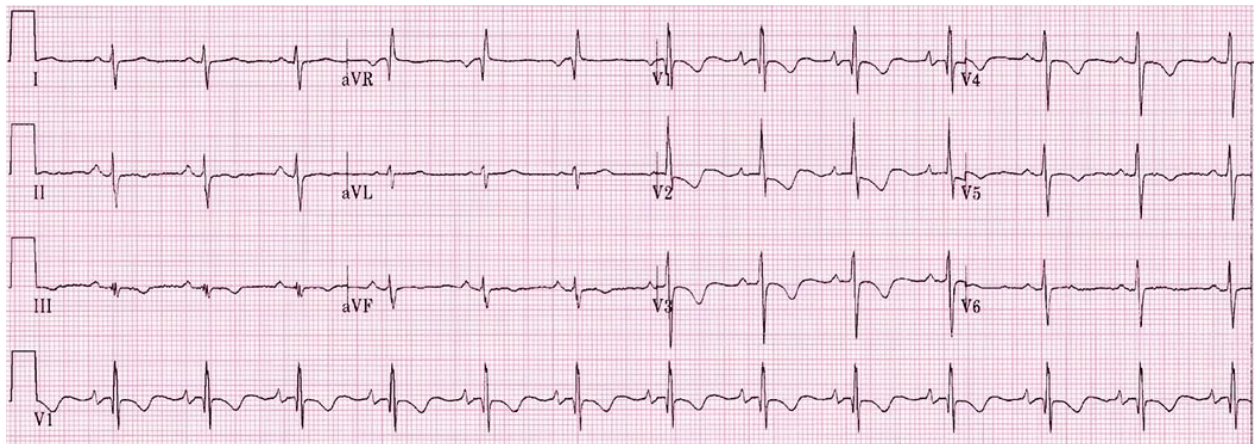


FIGURE 23.39A ■ **Right Ventricular Hypertrophy.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Rightward axis
- Dominant S wave in lateral leads (V₅, V₆, I, aVL)

- Dominant R wave in aVR, V₁, V₂, which may exceed the S-wave amplitude (especially in lead V₁)
- T-wave inversions in relation to QRS complex

Pearls

1. Right ventricular hypertrophy (RVH) causes characteristic ECG changes as the predominant electrical signal of the left ventricle is overcome.
2. Congenital heart disease, pulmonic or mitral stenosis, and pulmonary hypertension are common causes of RVH.
3. As RVH persists, right atrial enlargement may occur as seen in the example (P-wave amplitude in V₁ > 1.5 mm).

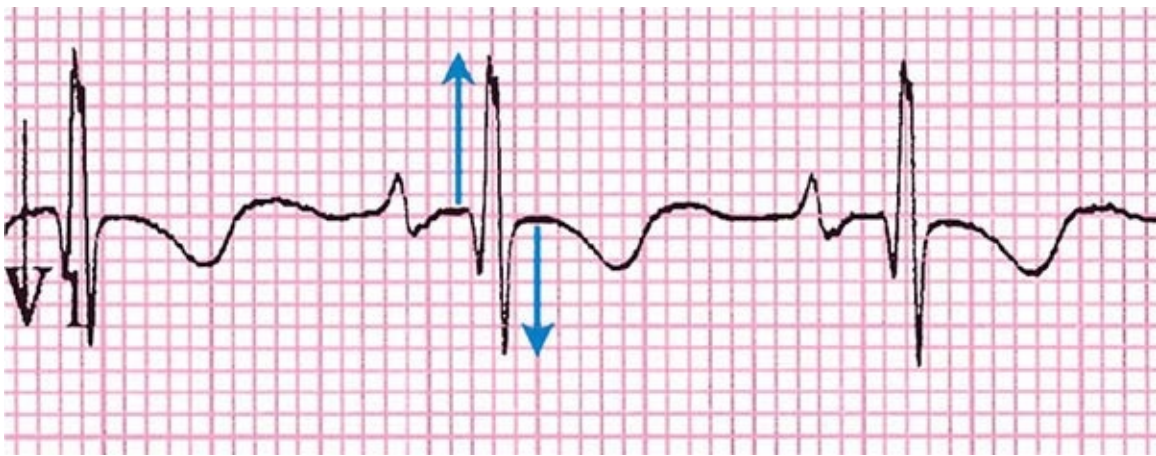


FIGURE 23.39B ■ The R-wave amplitude exceeds the S-wave amplitude (arrows) in lead V₁. In addition, the P-wave upward deflection exceeds 1.5 mm, indicating concomitant right atrial enlargement.

LEFT ATRIAL HYPERTROPHY

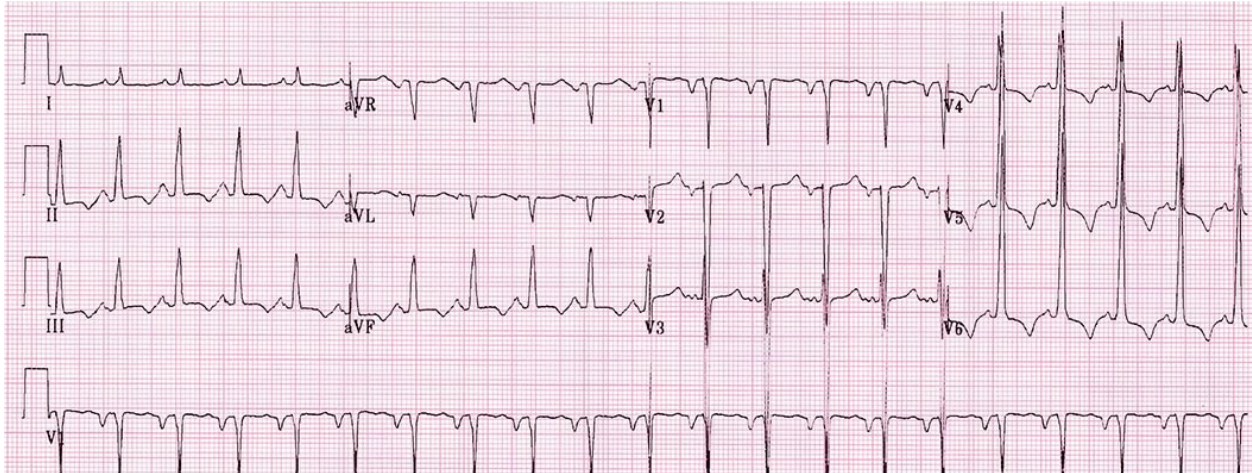


FIGURE 23.40A ■ Left Atrial Hypertrophy with LVH Present. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Increased duration (width) of the P wave without affecting its upward amplitude (as commonly seen with right atrial abnormalities)
- Negative P-wave deflection in lead V_1 , with width and depth > 40 ms
- Bifid P wave in lead II with total P duration > 110 ms and > 40 ms between two peaks
- Notched P wave in II, III, or aVF with duration ≥ 120 ms

Pearls

1. Normal P-wave morphology has an amplitude of < 2.5 mV (2.5 vertical boxes) and a duration (width) of < 120 ms (three small boxes).
2. The left atrium depolarizes after the right atrium and therefore has the most effect on the 2nd portion of the P wave.
3. Causes of left atrial abnormality or P-mitrale include valvular heart disease (mitral and aortic), coronary artery disease, cardiomyopathy, hypertension, and LVH.



FIGURE 23.40B ■ The P wave in V_1 is downgoing. The downgoing segment is wider and deeper than one small block (double arrows).

RIGHT ATRIAL HYPERTROPHY

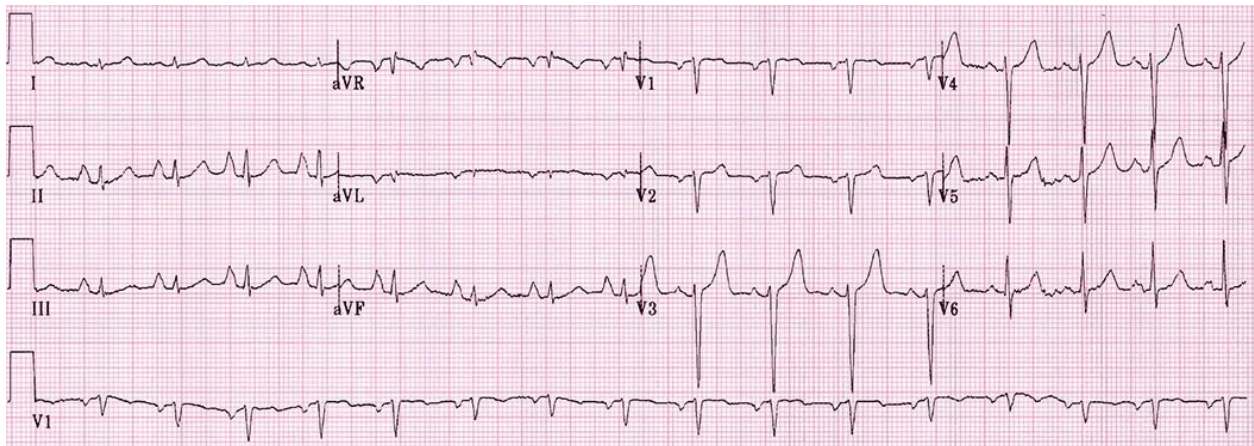


FIGURE 23.41A ■ **Right Atrial Hypertrophy.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Increased amplitude of P wave without affecting duration (as commonly seen with left atrial abnormalities)
- Peaked P waves (> 2.5 mm) in leads II, III, aVF
- P-wave upward deflection > 1.5 mm in lead V_1 or V_2

Pearls

1. Normal P-wave morphology has amplitude of < 2.5 mV (2.5 small vertical boxes) and duration (width) of < 120 ms (three small boxes).
2. The right atrium depolarizes before the left atrium and therefore has the most effect on the 1st portion of the P wave.
3. Right atrial enlargement is often associated with RVH, COPD, some congenital heart diseases, and pulmonary hypertension, and may be seen transiently with pulmonary embolus.



FIGURE 23.41B ■ The P wave in lead II (an inferior lead) is greater than 2.5 mm in amplitude (double arrow).

HYPERTROPHIC CARDIOMYOPATHY

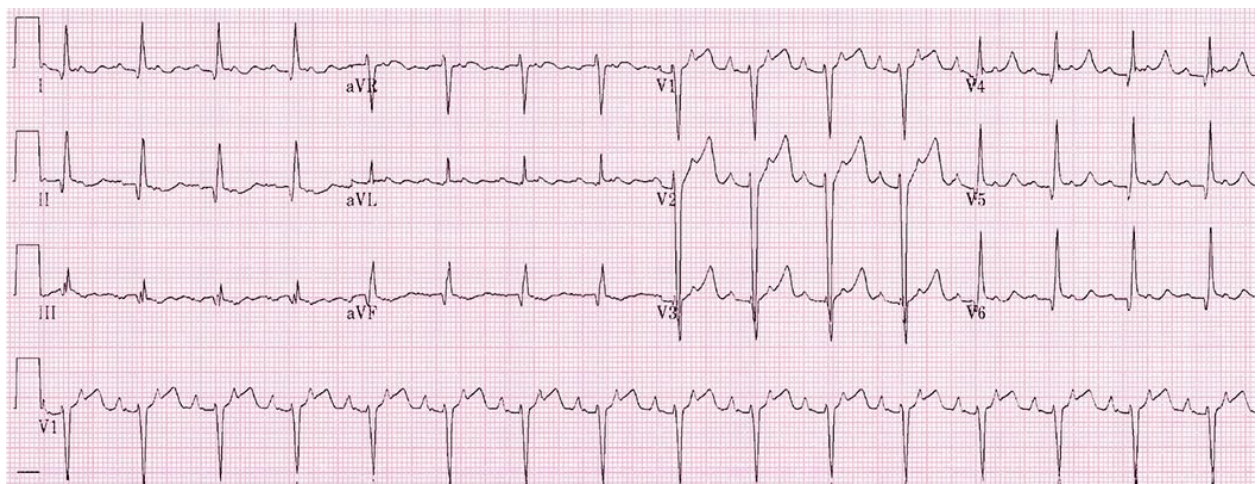


FIGURE 23.42A ■ Hypertrophic Cardiomyopathy with Underlying Atrial Flutter with 2:1 Block.
(ECG contributor: James V. Ritchie, MD.)

ECG Findings

- High-voltage QRS suggesting LVH
- Deep, narrow, “dagger-like” Q waves, especially in the lateral (V_5 , V_6 , I, aVL) and inferior (I, III, aVF) leads
- Deep S waves in anterior precordial leads
- Lateral T-wave inversions

Pearls

1. Hypertrophic obstructive cardiomyopathy (HOCM) causes hypertrophy of the interventricular septum, causing outflow obstruction, and predisposes the patient to ventricular arrhythmias.
2. Always consider this condition in young athletes with syncope or severe dyspnea on exertion.
3. The Q waves seen in HOCM can be mistaken for Q waves seen in myocardial ischemia; however, infarction Q waves are typically > 40 ms, whereas HOCM Q waves are < 40 ms.

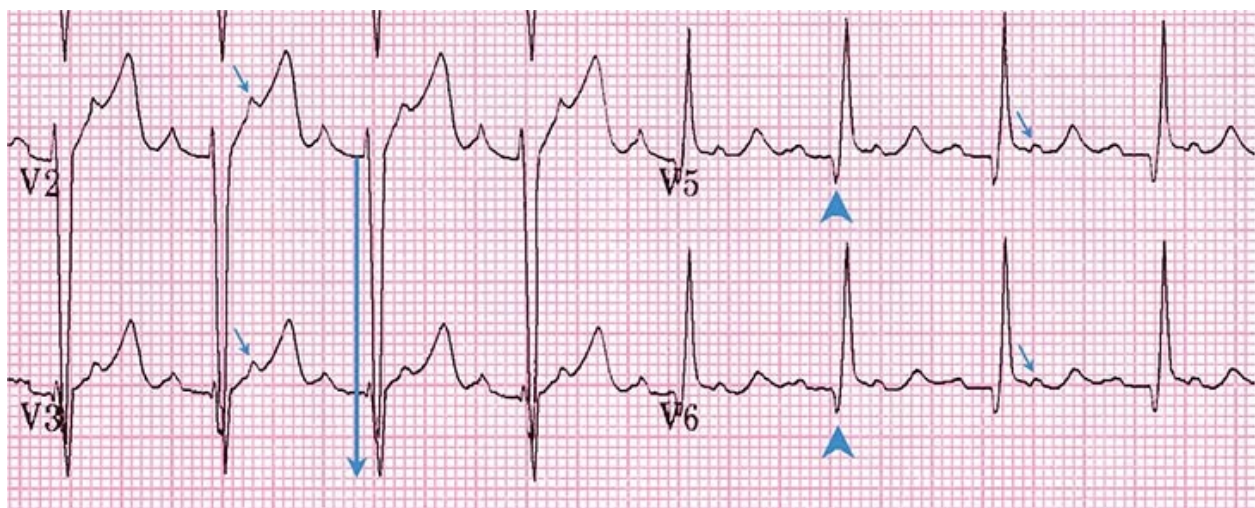


FIGURE 23.42B ■ Deep S-wave voltage (28 mm S in V_2 , large arrow) and narrow Q waves in V_5 and V_6 (arrowheads). This patient also has atrial flutter with 2:1 block. The additional P waves appear in the ST segments (small arrows).

BRUGADA SYNDROME

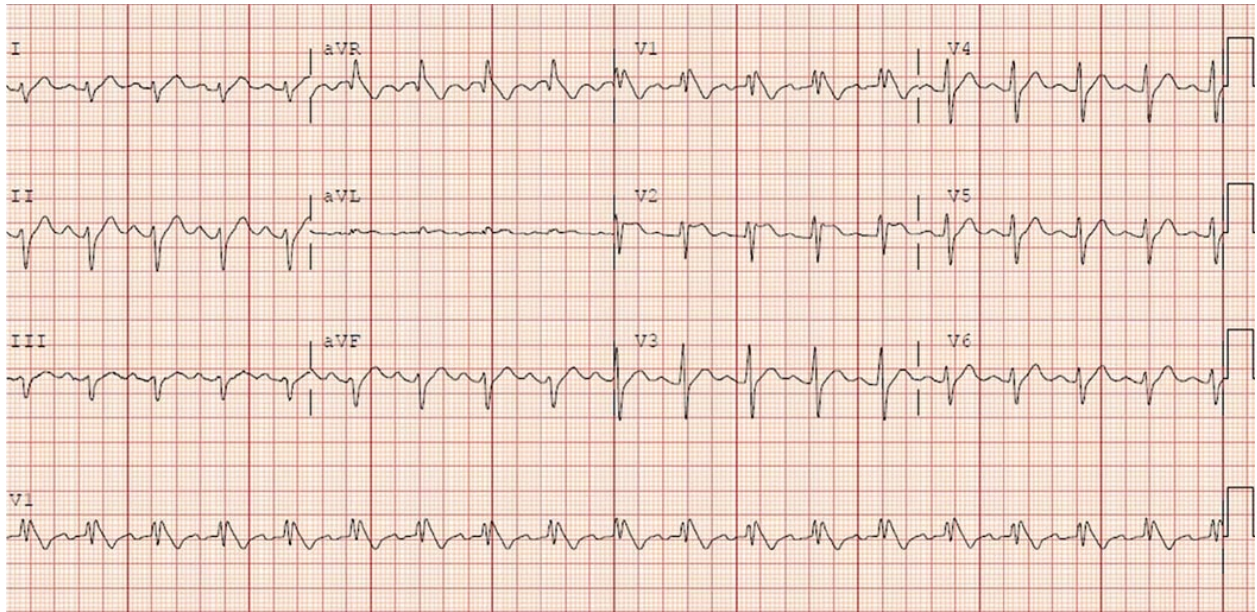


FIGURE 23.43A ■ Brugada Syndrome. (ECG contributors: Clifford L. Freeman, MD, and Nicole S. McCoin, MD.)

ECG Findings

- Incomplete or complete RBBB pattern in leads V_1 and V_2 with ST-segment elevation.
- There are two types of ST elevation: (1) the type I pattern has ST elevation that is convex upward, terminating in a negative T wave; this is the most sensitive and specific pattern; (2) the type II pattern has ST elevation that is concave upward (saddleback pattern) and is less sensitive and less specific.

Pearls

1. Brugada syndrome is thought to be a sodium channelopathy.
2. Patients with Brugada syndrome are at risk for spontaneous arrhythmias and sudden cardiac death.
3. ECG changes in Brugada syndrome patients are transient and often unmasked by fever, ischemia, and sodium channel– blocking drugs.
4. Consultation with a cardiologist is recommended for electrophysiologic testing and automated implantable defibrillator placement.



FIGURE 23.43B ■ RBBB pattern with ST elevation (type I Brugada syndrome).

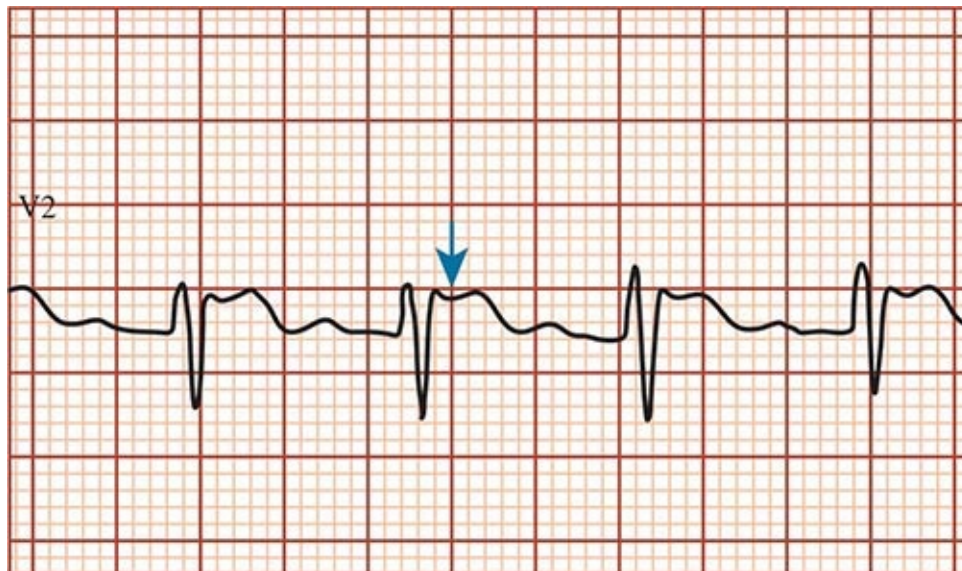


FIGURE 23.43C ■ ST elevation with “saddleback” morphology (arrow) (type II Brugada syndrome).

WOLFF-PARKINSON-WHITE SYNDROME

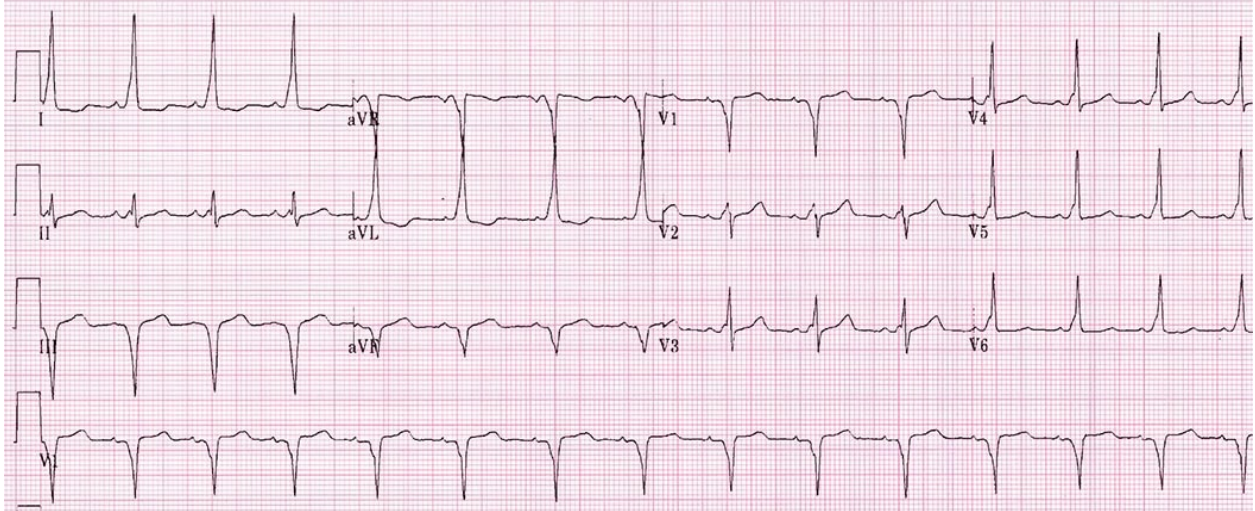


FIGURE 23.44A ■ **Wolff-Parkinson-White Syndrome.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Normal P waves
- Shortened PR interval
- Prolonged QRS interval
- Delta waves (slurring of the initial upstroke of R wave)

Pearls

1. Accessory tracts from the atria to the ventricles lead to depolarization of ventricles without using the AV node as the primary connecting route.
2. Tachycardia associated with WPW may be mistaken for VT. Suspect WPW if the QRS complex is wide and tachycardia is extreme (ventricular rate > 240).
3. Do not treat irregular atrial tachycardias (atrial fibrillation) in the setting of WPW with AV nodal blocking agents (calcium channel blockers, β -blockers, digoxin). This may lead to unopposed ventricular stimulation through the accessory tract and may worsen the tachycardia.
4. Procainamide and cardioversion are accepted methods for conversion of a tachycardia associated with WPW.
5. Depolarization via the accessory pathway may produce “pseudo-Q waves.”



FIGURE 23.44B ■ The PR interval is shortened (double arrow) and a delta wave (upsloping initial QRS segment) is seen (arrow, shaded area).

Part 5: ECG Abnormalities of Noncardiac Origin

HYPOTHERMIA

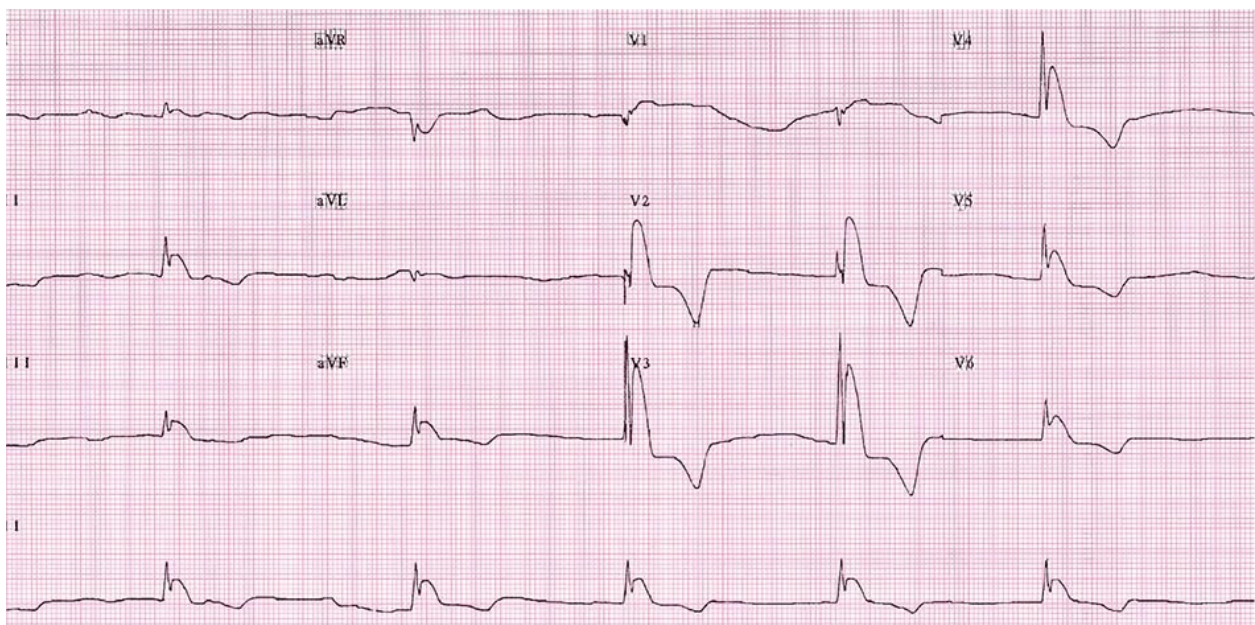


FIGURE 23.45A ■ **Hypothermia with Osborne Waves (“J” Waves) Present.** (ECG contributor: Michael L. Juliano, MD.)

ECG Findings

- Sinus bradycardia or atrial fibrillation with slow ventricular response.
- PR, QRS, and QT intervals are typically prolonged.
- Osborne or “J” wave (a positive deflection of the terminal portion of the QRS complex). The J wave may be subtle or large and “humped.”

Pearls

1. Myocardial damage and ECG changes associated with hypothermia are not necessarily due to low temperature. They may be indirectly caused by systemic circulatory issues such as hypoperfusion.
2. The hypothermic patient’s rhythm slows, proceeding from sinus bradycardia to atrial fibrillation with slow response and may proceed to other arrhythmias including ventricular fibrillation and asystole.
3. The amplitude of the “J” wave corresponds to the degree of hypothermia.
4. Defibrillation and medications may be ineffective in the hypothermic patient. Rapid rewarming is indicated as an initial and critical resuscitative measure.
5. Osborn waves can also be seen in other conditions, including hypercalcemia, brain injury, and subarachnoid hemorrhage.

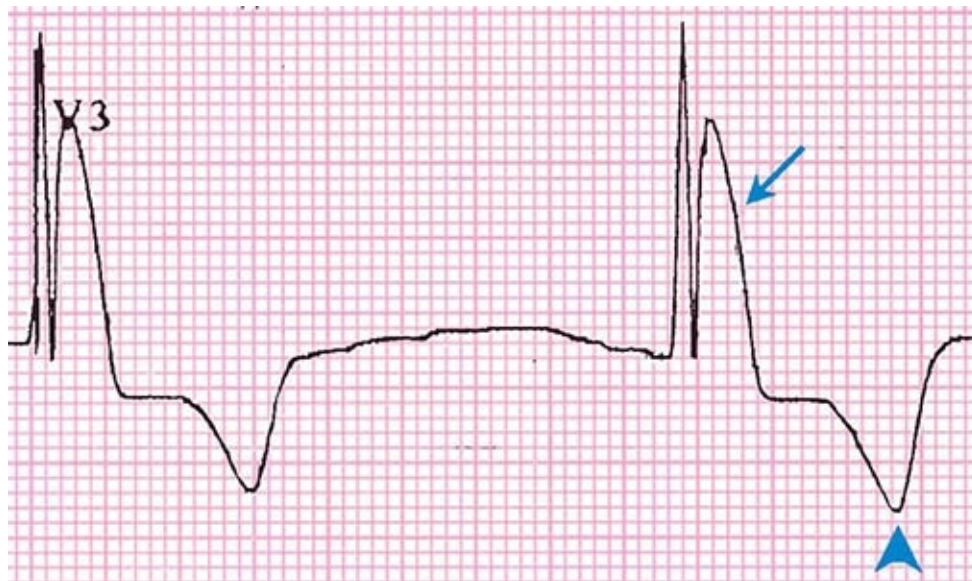


FIGURE 23.45B ■ A large Osborn wave (J wave) (arrow) follows the QRS, and is distinct from the T wave (arrowhead).



FIGURE 23.45C ■ This is a more typical appearance of a J wave (arrow).

“CEREBRAL” T WAVES

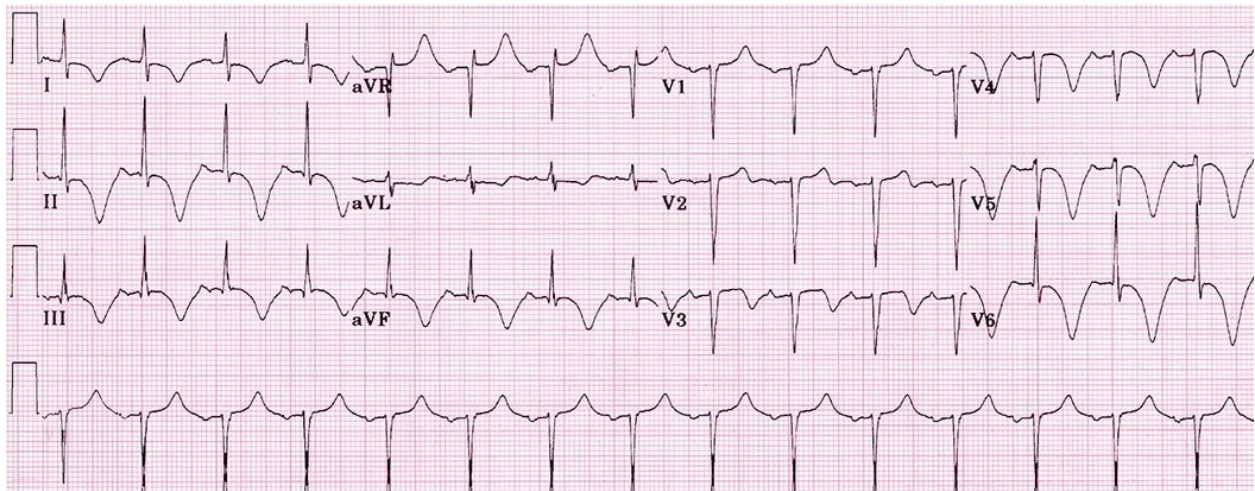


FIGURE 23.46A ■ **Cerebral T Waves.** This ECG was obtained on a patient with a severe acute hemorrhagic cerebrovascular accident. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Inverted, deep, and wide T waves are most notable in pre-cordial leads (can be seen in any lead).
- QT interval prolongation.
- ST segment changes are variable.
- High-amplitude R waves.

- Arrhythmias may also occur in the setting of acute cerebral emergencies.

Pearls

1. These ECG changes are associated with increased intracranial pressure.
2. Cerebral T waves resolve spontaneously, but can persist up to 6 weeks.
3. Suspect increased intracranial pressure in patient presenting with altered mental status and the previously mentioned ECG changes.

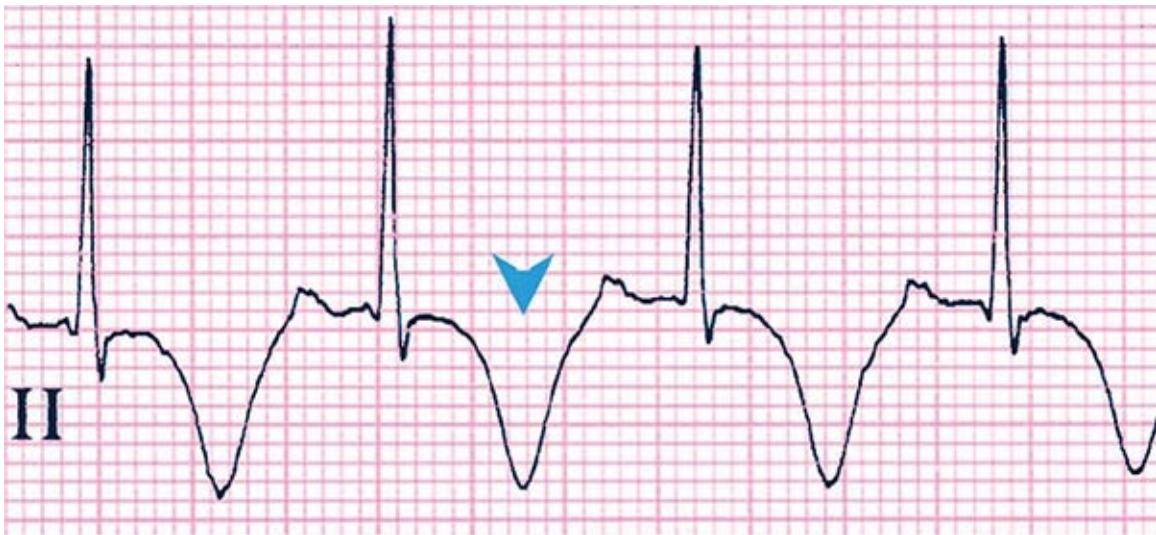


FIGURE 23.46B ■ Deep, symmetrical, inverted T waves (arrowhead) with a prolonged QT interval.

HYPERKALEMIA

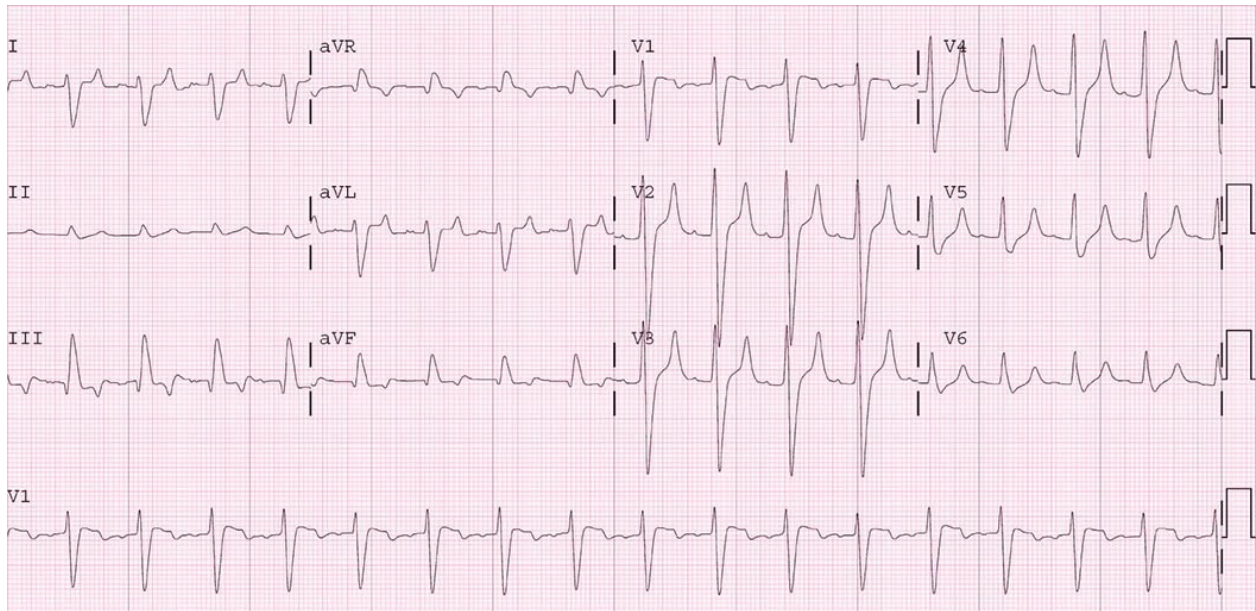


FIGURE 23.47A ■ **Hyperkalemia (potassium of 7.1).** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

Findings are variable but tend to correlate with increasing serum potassium levels following the order below:

- Peaked T waves, tented with a narrow base (may be > 10 mm high in precordial leads and/or > 6 mm in limb leads)
- QRS complex widening
- PR interval prolongation
- Decreased P-wave amplitude
- As potassium levels approach and exceed 8.0 mEq/L:
 - Indiscernible P waves
 - New bundle branch blocks or fascicular blocks
 - Sine wave appearance of QRS-T complex
 - VT, fibrillation, or asystole

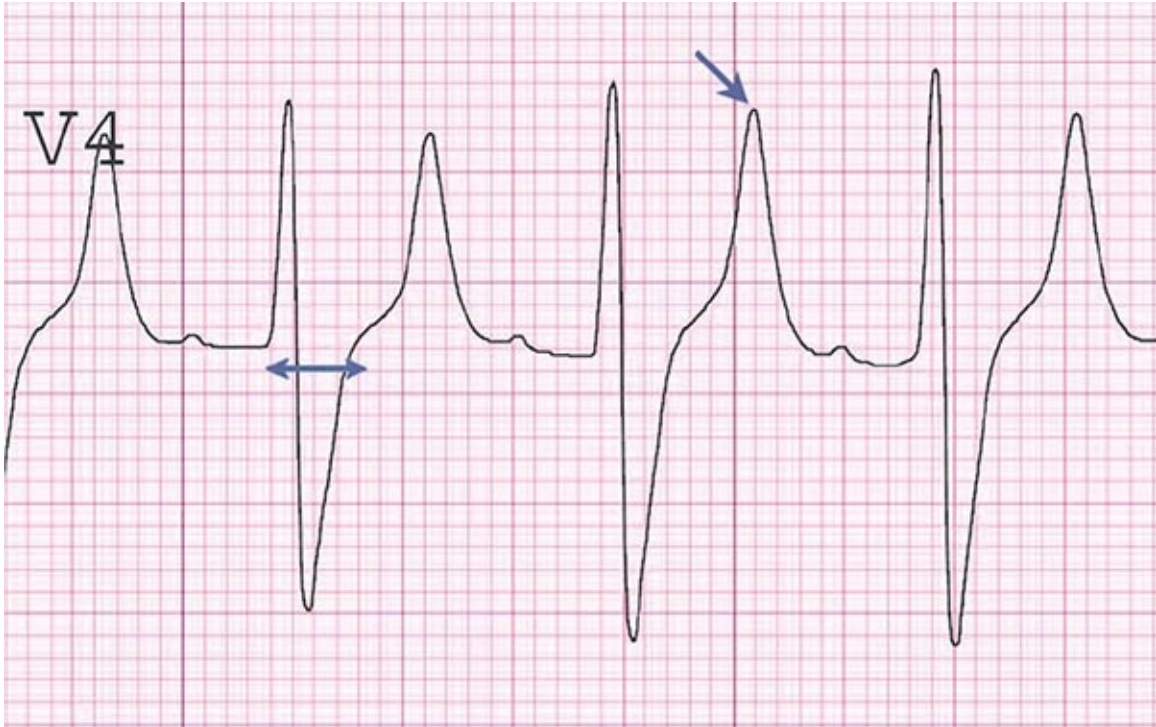


FIGURE 23.47B ■ Peaked T waves (arrow), widened QRS (double arrow), and subtle flattening of the P waves are seen in this patient with a serum potassium of 7.1.

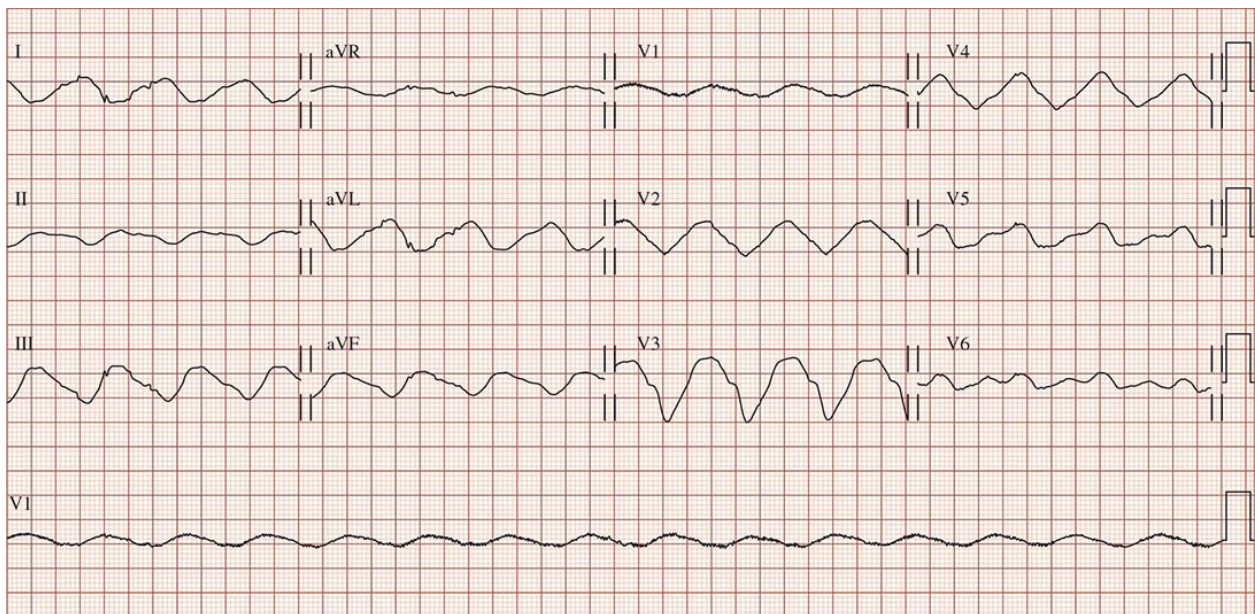


FIGURE 23.48A ■ **Severe Hyperkalemia—Sine Wave.** (ECG contributors: Sam Parnell, MD, and Tracy Fennessy, MD.)

Pearls

1. Hyperkalemia is often missed on the initial ECG as it can produce nonspecific changes.
2. Acute treatment for hyperkalemia includes insulin and glucose, sodium bicarbonate, and β -agonists in an attempt to drive potassium into the cell. Intravenous calcium may be used to stabilize the myocardium but has no effect on serum potassium levels. These are temporizing measures that must be followed by definitive treatment of the underlying problem, which may include the need for dialysis.

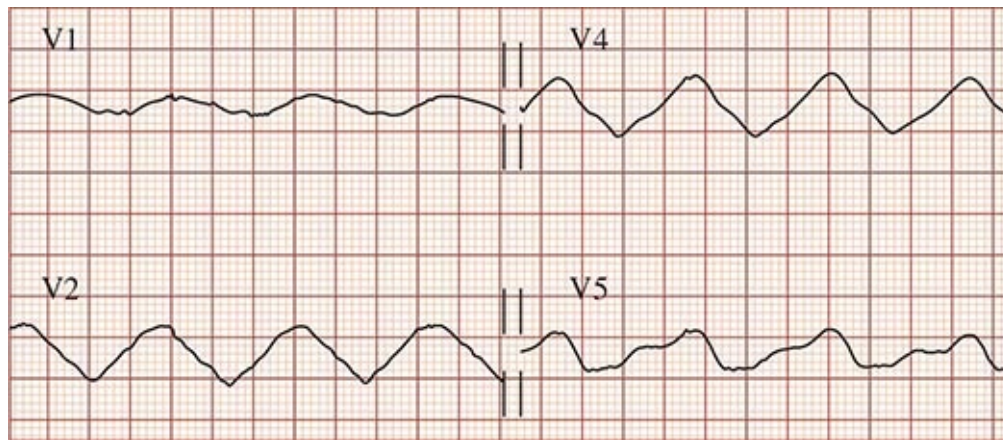


FIGURE 23.48B ■ Wide blunted QRS with sine wave appearance. No P waves are visible. Serum potassium was > 8 in this patient.

HYPOKALEMIA

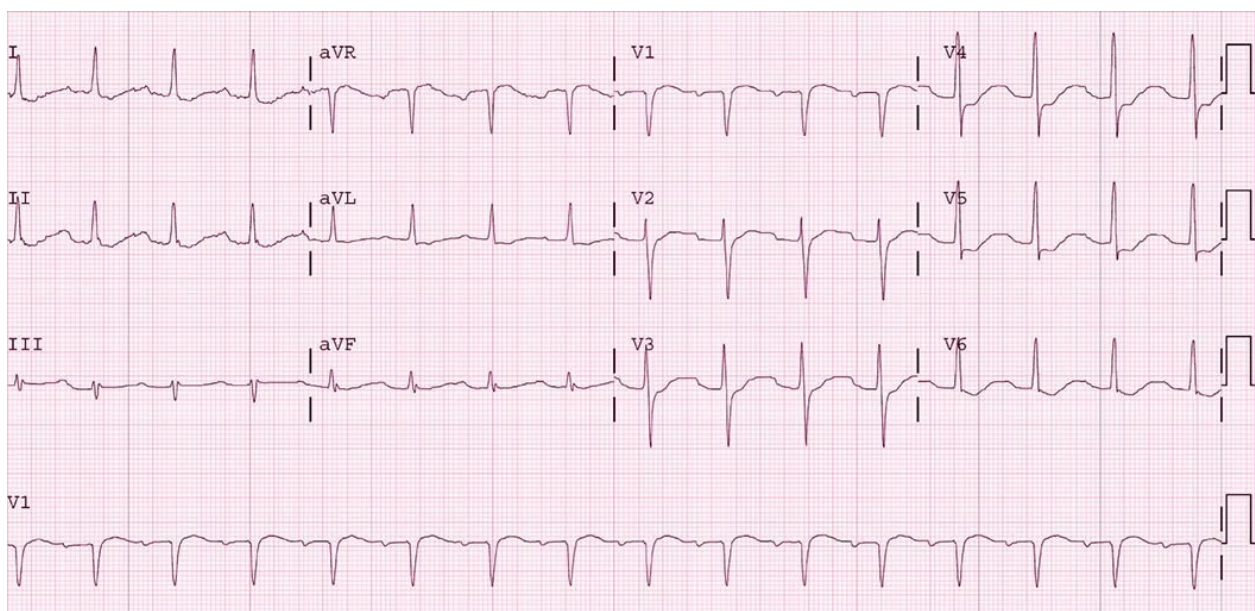


FIGURE 23.49A ■ **Hypokalemia.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- Flattened or inverted T waves
- Prominent U waves that can merge with the T waves producing the appearance of a prolonged QT
- ST-segment depression
- Conduction disturbances

Pearls

1. Hypokalemia can produce varied ECG changes associated with the repolarization phase of the cardiac cycle.
2. Unlike hyperkalemia, in hypokalemia, there is no direct correlation with the potassium level and the severity of ECG changes. However, more ECG changes may become apparent as the potassium level falls.
3. Suspect hypomagnesemia if the ECG does not normalize after potassium replacement.

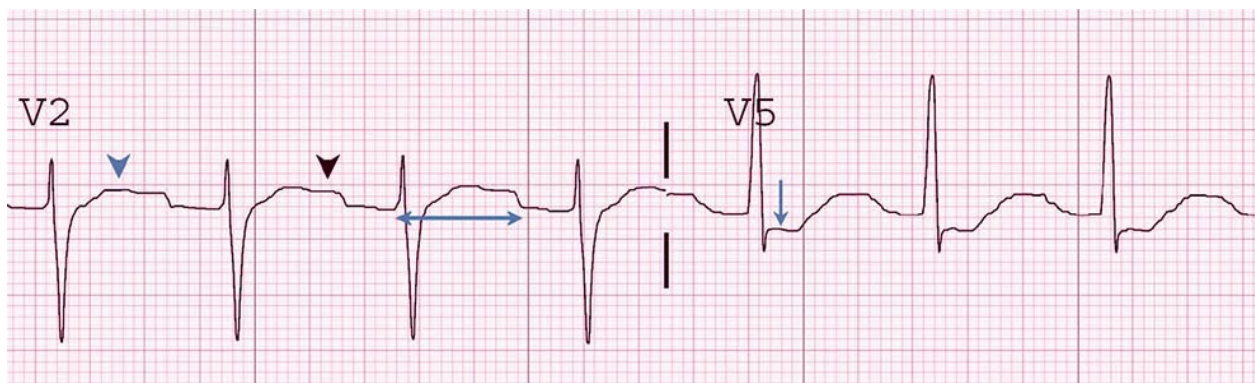


FIGURE 23.49B ■ This ECG demonstrates multiple findings consistent with hypokalemia: flattened T waves (blue arrowhead), U waves (black arrowhead), prolonged QT (QU) intervals (double arrow), and ST-segment depression (arrow). This patient's potassium level was 1.9.

PULMONARY EMBOLISM

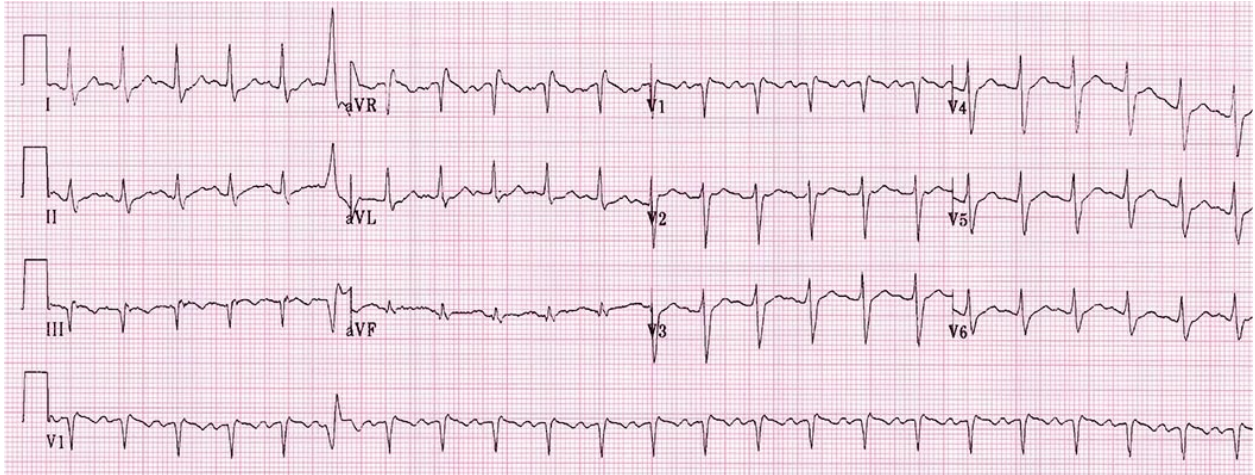


FIGURE 23.50A ■ Sinus Tachycardia and S1Q3T3 Pattern in a Patient with Acute Pulmonary Embolism. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Sinus tachycardia, nonspecific ST-T changes.
- Precordial T-wave inversions.
- Prominent S wave in lead I, Q wave in lead III, and inverted wave in III ($S_1/Q_3/T_3$).
- Incomplete or complete RBBB, P pulmonale (lead II).
- Rightward axis.
- ST-segment elevation (especially in leads V_1 , aVR) and ST-segment depression may occur in cases of large pulmonary emboli.

Pearls

1. No ECG pattern is diagnostic for pulmonary embolism. Small-to-moderate emboli may not affect the ECG.
2. With large emboli, increased resistance to pulmonary arterial flow produces right ventricle overload and dilation.
3. Increased right atrial pressures may produce P pulmonale (tall P waves > 2.5 mm in lead II) or atrial dysrhythmias.

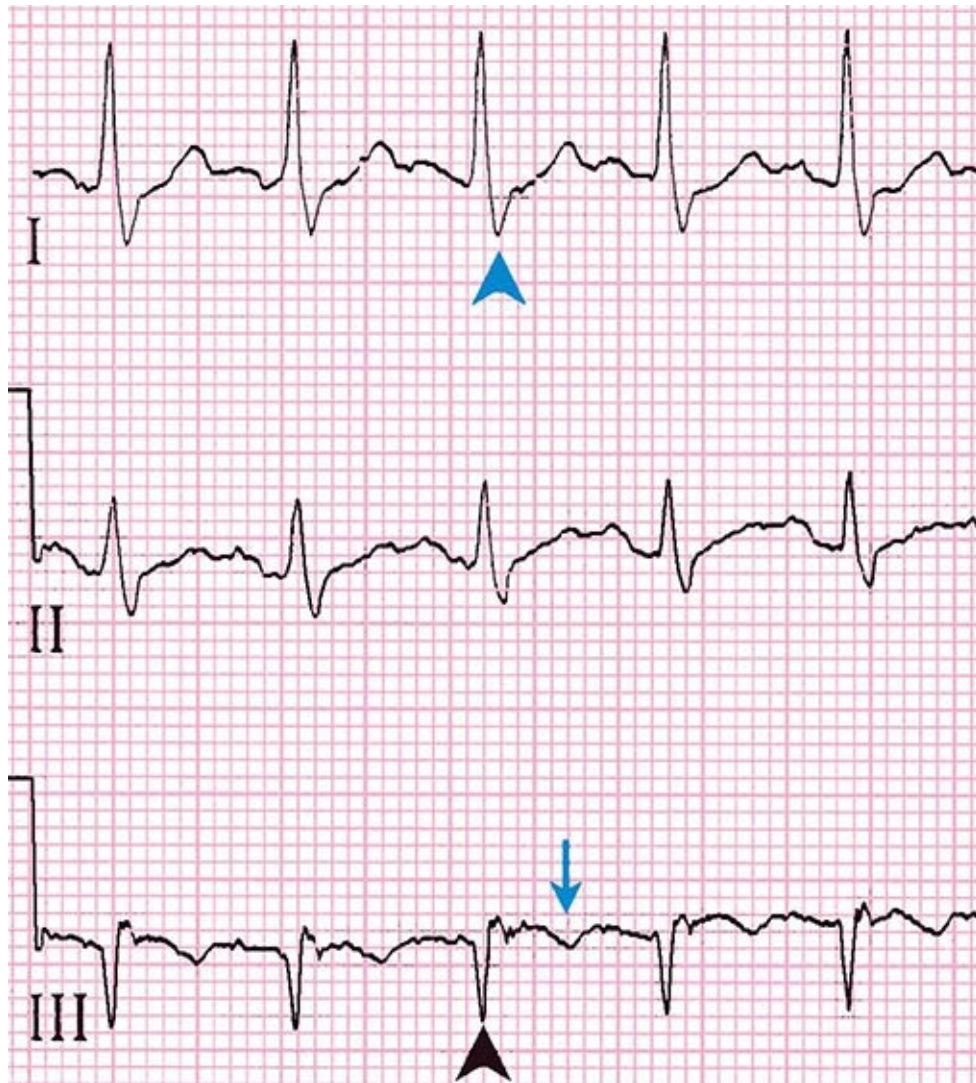


FIGURE 23.50B ■ S wave is apparent in lead I (blue arrowhead), Q wave in lead III (black arrowhead), and inverted T wave in lead III (blue arrow).

PERICARDIAL EFFUSION

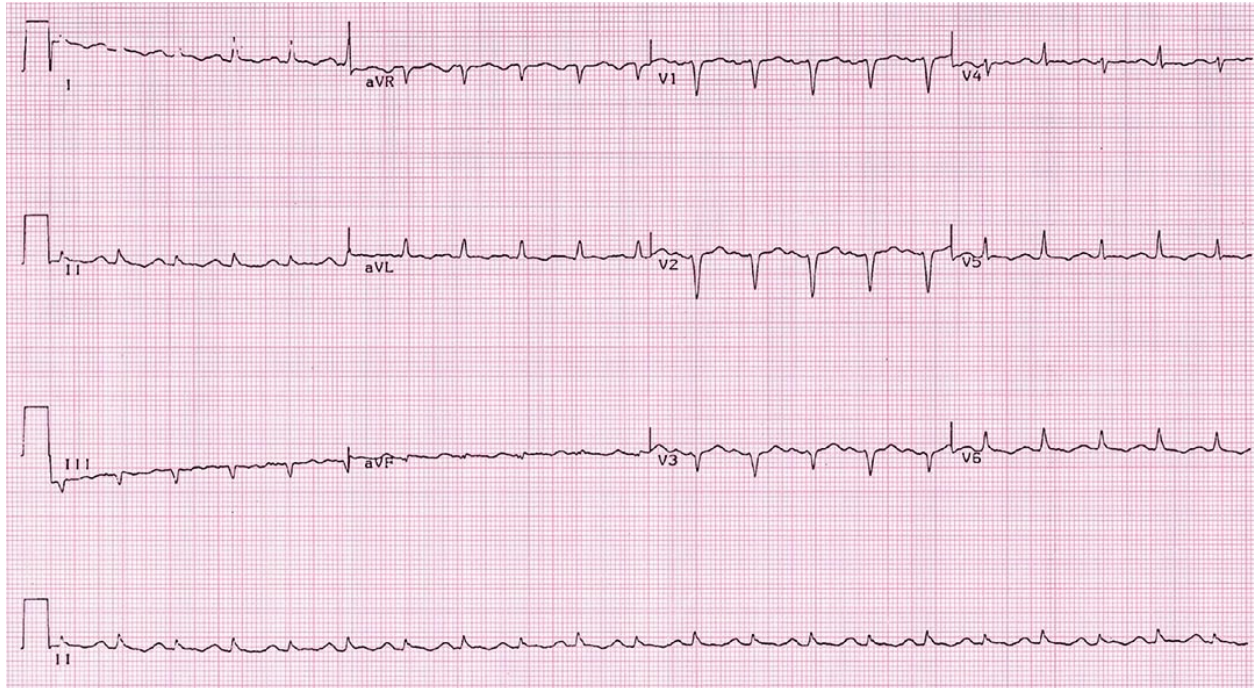


FIGURE 23.51A ■ Pericardial Effusion with Electrical Alternans. (ECG contributor: Kevin E. Zawacki, MD.)

ECG Findings

- Sinus tachycardia
- Low voltage of QRS complex (QRS averaging < 5 mm height in limb leads or < 10 mm height in precordial leads)
- Electrical alternans (beat-to-beat change in electrical axis and/or amplitude of the QRS complex)

Pearls

1. A physiologically significant pericardial effusion compresses the heart and affects the ability of the heart to fill properly. This causes pressure on the heart, which decreases its pre-load and thereby cardiac output.
2. Pericardial effusion may be caused by pericarditis, malignancy, uremia, trauma, iatrogenic injury, aortic dissection with retrograde involvement of the pericardium, and free wall rupture after an MI.
3. Initial treatment of physiologically significant pericardial effusion is with intravenous fluid bolus to increase preload. Emergent pericardiocentesis should be reserved for hemo-dynamically unstable patients. Surgical

pericardial window may be considered.

4. Electrical alternans, although “classic,” is present in less than one-third of cases. Pericardial effusion should be suspected in the setting of a sinus tachycardia and low voltage with or without electrical alternans.

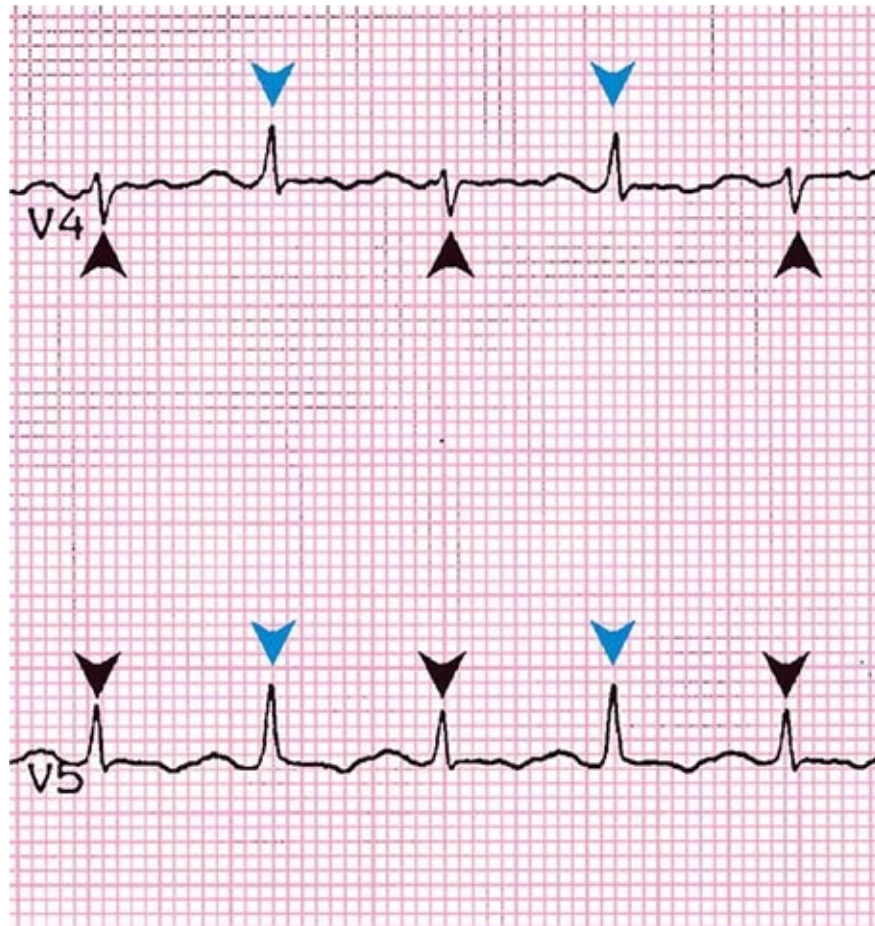


FIGURE 23.51B ■ Low voltage, sinus tachycardia, and electrical alternans (arrowheads) demonstrate beat-to-beat alternating QRS electrical axis and/or amplitude. Electrical alternans is often best seen in the anterior precordial leads V₃ and V₄.

DIGOXIN EFFECT, TOXICITY

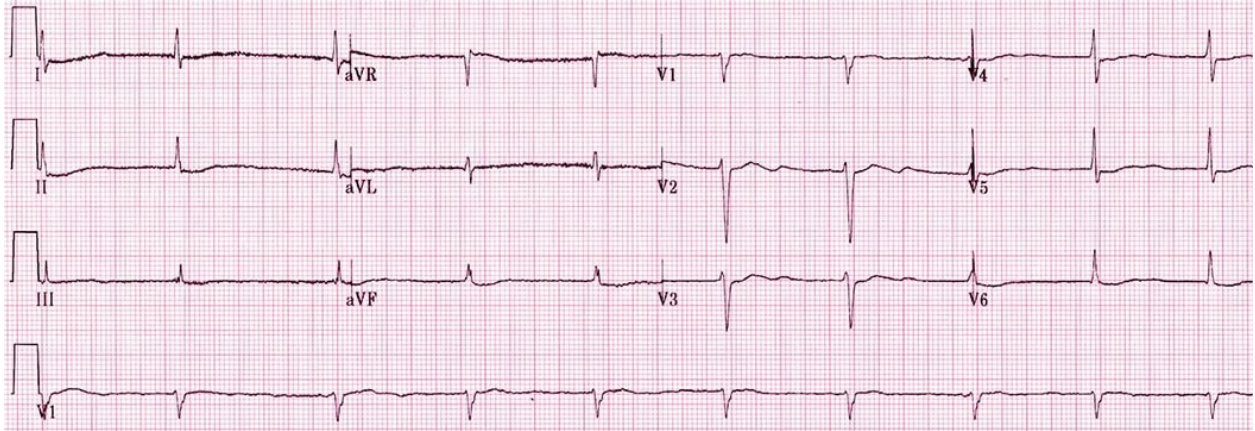


FIGURE 23.52A ■ **Digoxin Effect with Evidence of Toxicity.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment shortening and depression leading to a “scooped” appearance.
- QT interval shortening.
- PR interval prolongation.
- Decreased T-wave amplitude.
- Premature ventricular complexes are the most common dysrhythmia.
- Bradydysrhythmias, heart block, especially with findings consistent with increased automaticity (atrial tachycardia with block, atrial fibrillation with slow ventricular response, accelerated junctional rhythms).
- Bidirectional VT may rarely be seen (see [Fig. 17.99](#)).

Pearls

1. ECG changes associated with digoxin can be seen from therapeutic or toxic levels.
2. ST-segment changes may be exaggerated by myocardial disease or tachycardia.
3. An acute overdose of a digoxin is usually associated with hyperkalemia, which may increase the height of the T wave.
4. Avoid calcium for treatment of hyperkalemia in the setting of digoxin toxicity as this may potentiate adverse effects of digoxin.



FIGURE 23.52B ■ The “sagging” appearance of the ST segment (arrow) is characteristic of digoxin therapy and is not a sign of toxicity. However, this patient also has a sign of chronic digoxin toxicity. Atrial fibrillation is present, but the R-to-R interval has become regular. Digoxin toxicity has produced a total AV block but has also excited the AV node, producing a relatively accelerated junctional escape rate.

TRICYCLIC ANTIDEPRESSANT EFFECT

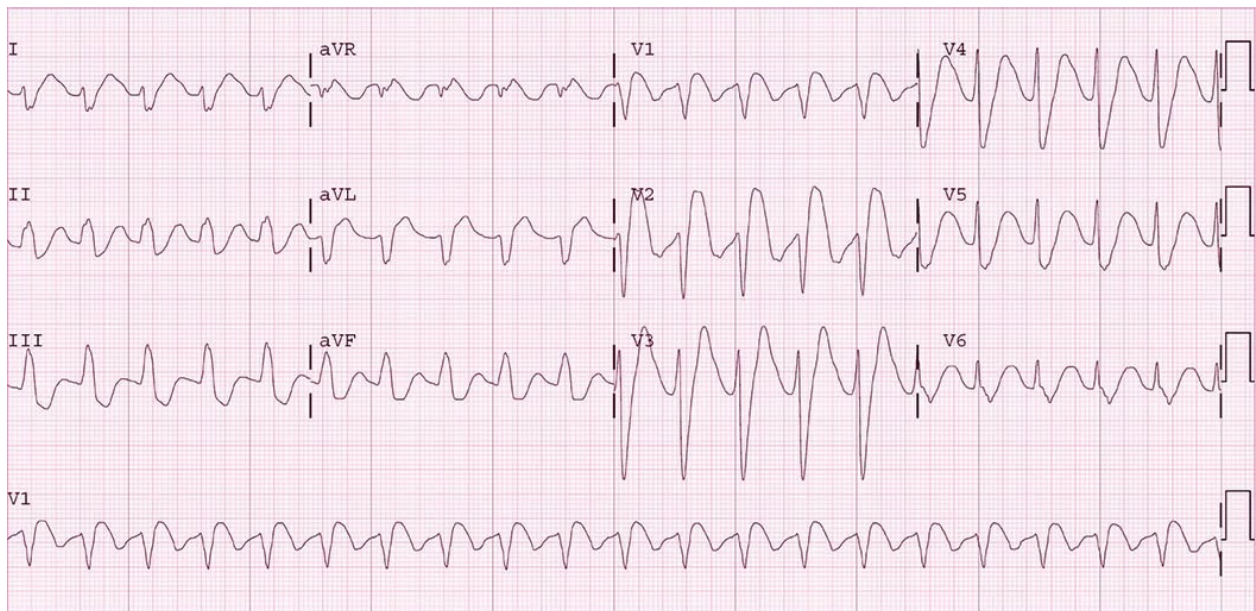


FIGURE 23.53A ■ **Tricyclic Antidepressant Toxicity.** (ECG contributor: Saralyn R. Williams, MD.)

ECG Findings

- Tachycardia
- QRS complex widening
- QT prolongation
- Prominent terminal R wave in aVR or V₁

- Prominent S in lead I

Pearls

1. Tricyclic antidepressants (TCAs) produce their effects by several mechanisms. Anticholinergic effects may induce tachycardia, and sodium channel blockage may lead to QRS widening.
2. The QRS widening seen in a TCA overdose has a nonspecific pattern and is typically unlike any bundle branch block morphology.
3. ECG effects are rate dependent and become more pronounced with tachycardia and acidosis.

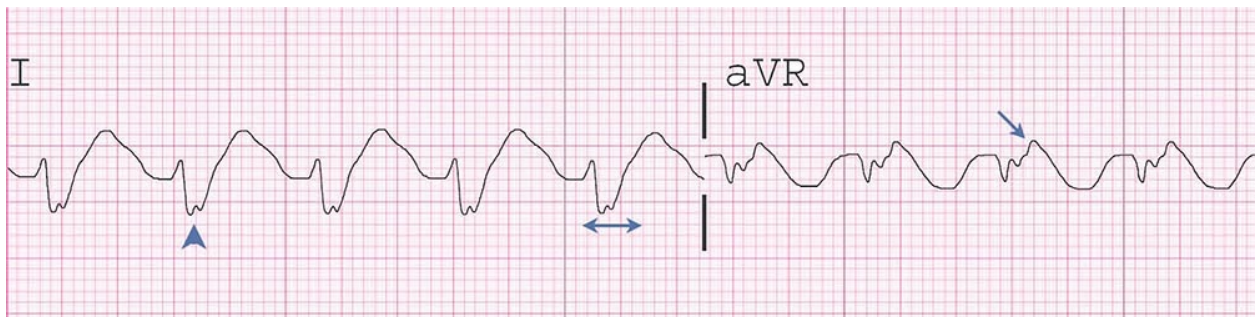


FIGURE 23.53B ■ Prominent S wave in lead I (arrowhead) with prominent terminal R wave in aVR (arrow). The QRS complex is wide (double arrow), the QT interval is prolonged, and the patient is tachycardic.

LIMB LEAD REVERSAL

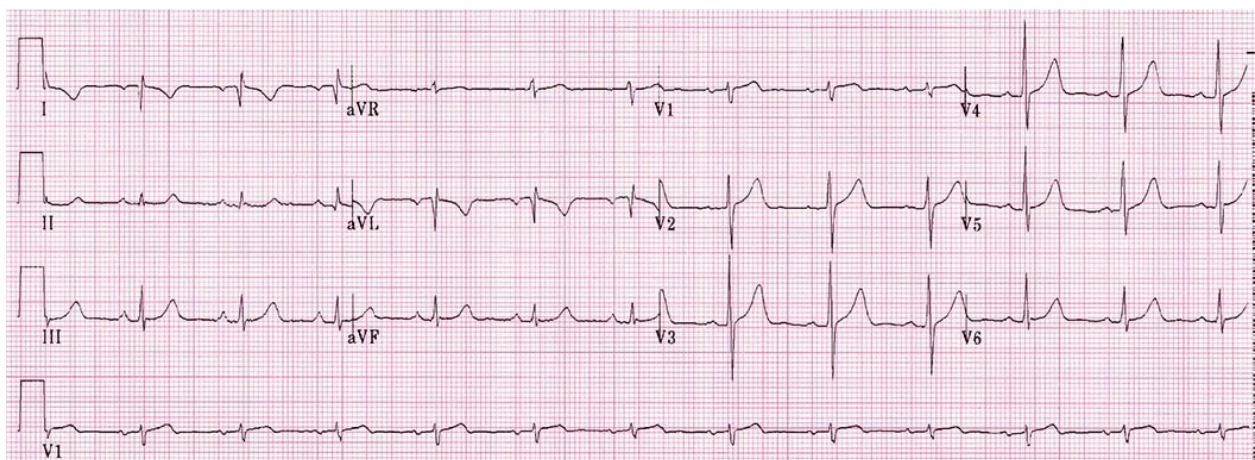


FIGURE 23.54A ■ Limb Lead Reversal (LA to RA). (ECG contributor: Michael L. Juliano, MD.)

ECG Findings

(dependent on which leads are reversed)

- Reversal of the left arm (LA) and right arm (RA) most common
P, QRS, and T predominantly downgoing in lead I
P, QRS, T upgoing in lead aVR
Precordial leads unaffected
- Reversal of the leg leads (left leg [LL] and right leg [RL]) Does not commonly produce ECG changes because RL is used as a grounding electrode
- Reversal of LA-LL
Transposition of leads I and II and leads aVF and aVL with reversal of lead III
- Reversal of RA-RL
Transposition of aVR and aVL and inversion of lead II
- Incorrect precordial lead placement
Isolated reversal of the usual R-wave progression from V_1 to V_6

Pearls

1. If the ECG seems to have an unusual axis or appearance, especially when compared with a prior ECG on the same patient, consider a lead misplacement and repeat the tracing, confirming correct lead positions.
2. A “reversed” lead I with normal-appearing V leads strongly suggests accidental limb lead reversal as opposed to dextrocardia. Dextrocardia features a “reversed” lead I, while QRS deflections in V_4 to V_6 appear small and downgoing.



FIGURE 23.54B ■ The P wave, QRS, and T wave are inverted in lead I in this ECG. Normal-appearing V leads in the 12-lead ECG above suggest limb lead reversal rather than dextrocardia. The arm leads were indeed reversed, and correction produced a normal-appearing tracing.

LOW VOLTAGE

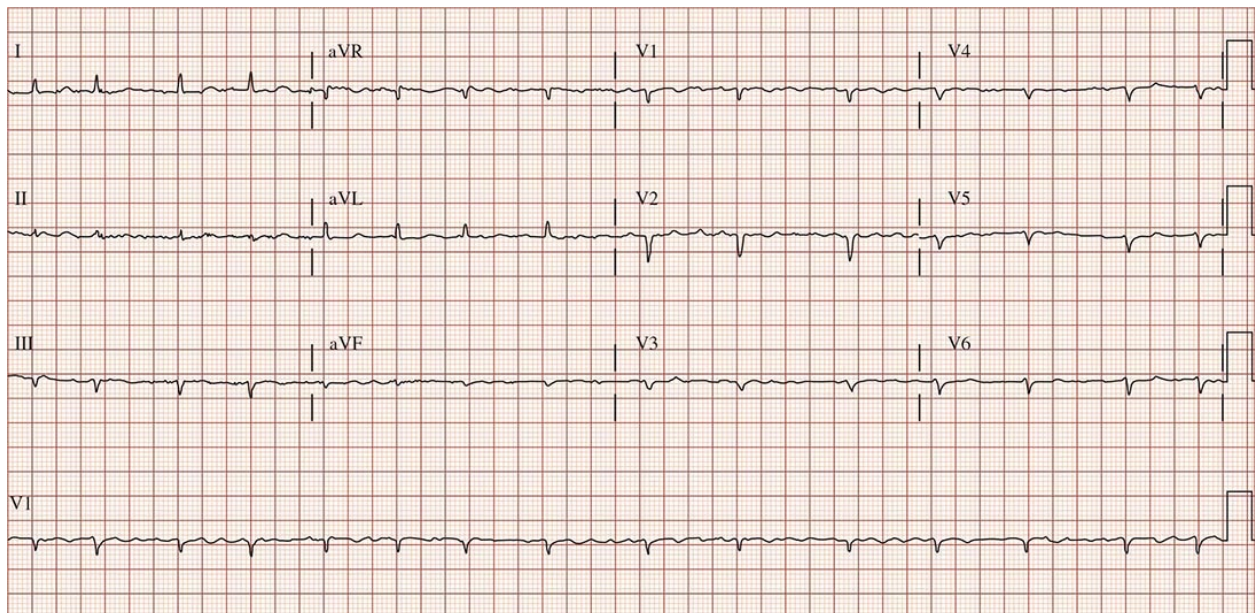


FIGURE 23.55A ■ **Low-Voltage ECG with Underlying Atrial Flutter.** (ECG contributors: Clifford L. Freeman, MD, and Nicole S. McCain, MD.)

ECG Findings

- QRS amplitude of ≤ 5 mm in all limb leads or QRS amplitude ≤ 10 mm in all precordial leads

Pearls

1. Differential diagnosis includes normal variant, low standardization of the ECG machine, pericardial or pleural effusion, obesity, anasarca, COPD/emphysema, cardiac infiltrate (tumor, amyloid), MI, myocarditis, cardiomyopathy, adrenal insufficiency, or hypothyroidism.
2. Always check the calibration markings on the ECG to check for low standardization of the ECG machine as an etiology for the observed tracing.

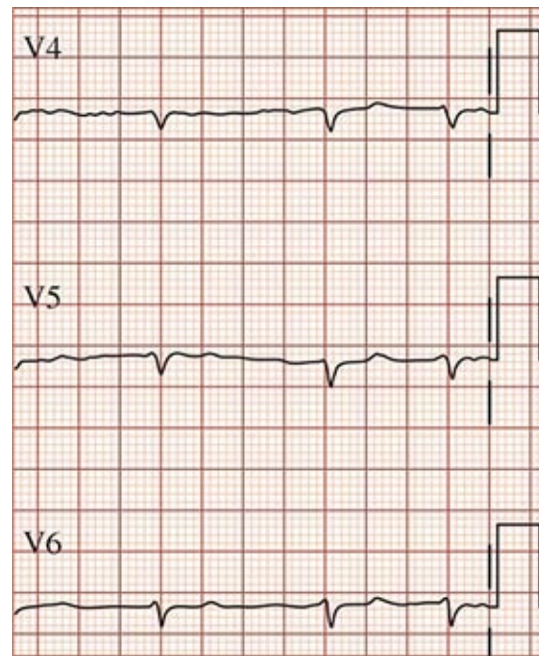


FIGURE 23.55B ■ QRS height is less than 5 mm in the precordial leads in this normally calibrated tracing.

The authors acknowledge the excellent contributions of James V. Ritchie, MD, and Michael L. Juliano, MD, to prior editions of this chapter.

Chapter 24

EMERGENCY ULTRASOUND

Jeremy S. Boyd
Myles Melton
Jordan D. Rupp
Robinson M. Ferre



Abdominal Aortic Aneurysm (AAA). Transverse image of a large AAA (11 cm). Small pockets of hypoechoic fluid (blood) are seen in the thickened atherosclerotic walls. Turbulent hyperechoic blood flow

is seen in the vascular lumen. (Ultrasound contributor: Jeremy S. Boyd, MD.)

EMERGENCY ULTRASOUND: INTRODUCTION

Emergency ultrasound (EUS) is the use of point-of-care ultra-sound by clinicians in the emergency department (ED) to answer focused clinical questions and/or assist in performing invasive procedures. It is not an extension of the physical exam by means of advanced technology, but rather uses sophisticated ultrasound technology to sonographically assess patients for the presence or absence of pathologic conditions that commonly present to the ED. The use and scope of EUS have evolved rapidly since the Focused Assessment with Sonography in Trauma (FAST) exam was first introduced to modern trauma care. Since May 2001, EUS has been a required component of Emergency Medicine residency training with additional training offered through dedicated EUS fellowship programs. There are currently over 100 such dedicated EUS fellowship programs in operation.

EUS can be used by clinicians across the spectrum of diseases that present to the ED. Properly trained emergency clinicians use ultrasound to answer focused questions regarding the patient's presenting complaint or condition. Rather than a comprehensive radiologic approach, the EUS examination is goal directed and focused, often seeking to answer dichotomous "yes/no" questions such as "Is there fluid within the peritoneum?"

This text is not intended to provide an in-depth description of the limitations, efficacy, sensitivity, or appropriateness of EUS. Every application described herein is intended as a rapid visual review for those trained in basic EUS applications. Applicable protocols are based on the imaging guidelines of the American College of Emergency Physicians and American Institute of Ultrasound in Medicine, as well as the authors' collective experience. Basic information—transducer recommendations, scanning protocols, anatomic schematics—is presented within each application to represent both image acquisition as well as normal and pathologic findings.

Key Terms

Sonographic windows—Anatomical locations on the body where an ultrasound probe is placed in order to view internal organs.

Transducer indicator/probe marker/marker dot—Usually a bump or ridge on

the ultrasound transducer that corresponds to a symbol on the ultrasound screen. Used to indicate which side of the image corresponds with the transducer’s edge (Fig. 24.1).

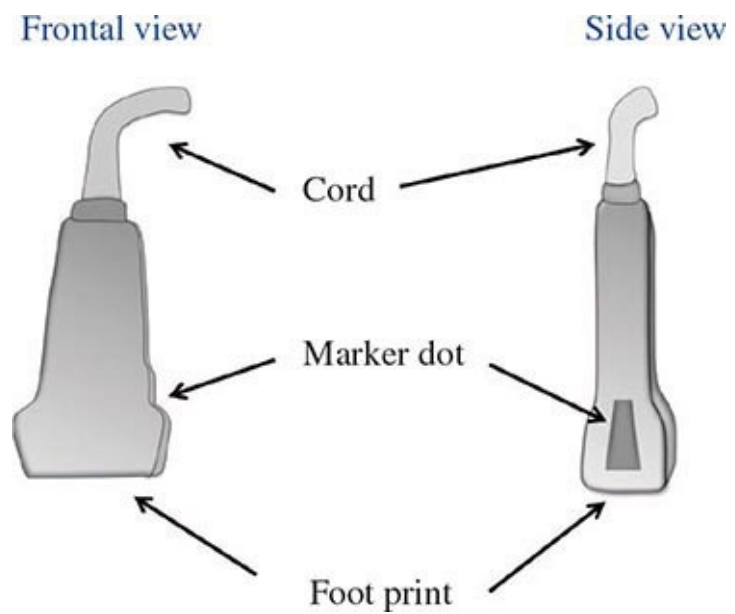


FIGURE 24.1 ■ Probe anatomy demonstrating location of transducer indicator/probe marker/marker dot and foot print. (Illustration contributor: Robinson M. Ferre, MD.)

Foot print—The part of the ultrasound probe that contacts the patient’s skin (Fig. 24.1).

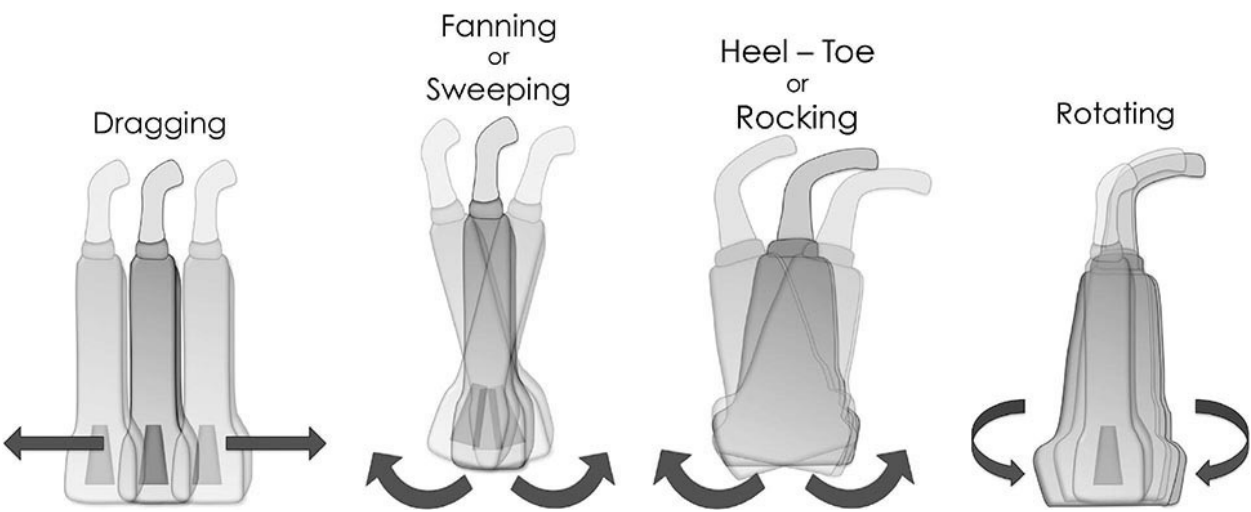











FIGURE 24.2 ■ **Transducer Movements.** (Illustration contributor: Robinson M. Ferre, MD.)

TABLE 24.1 ■ TRANSDUCER, APPEARANCE, AND APPLICATIONS
--

Transducer	Image Appearance	Applications*
Phased array  FIGURE 24.3A <ul style="list-style-type: none"> • Small footprint for imaging between ribs • Lower frequency (1-5 MHz) = better penetration, less resolution • Wide far-field view (wedge-shaped window) 	Cardiac  FIGURE 24.3B Abdominal  FIGURE 24.3C	(+) Cardiac, abdominal, thoracic (–) Soft tissue, pregnancy, vascular
Linear array  FIGURE 24.4A <ul style="list-style-type: none"> • Flat footprints in various sizes • High frequency (5-13 MHz) = better resolution of superficial structures • Rectangular window (at right) 	 FIGURE 24.4B	(+) Procedural, vascular, soft tissue/musculoskeletal, superficial, thoracic (pleural), ocular (–) Abdominal, cardiac, pregnancy
Endocavitary probe  FIGURE 24.5A <ul style="list-style-type: none"> • Curved footprint probe for examining internal structures • High frequency (5-8 MHz) = better resolution of nearby structures • Designed for intracavitary use, primarily transvaginal ultrasound • Wide wedge-shaped window (at right) 	 FIGURE 24.5B	(+) Transvaginal pregnancy, intraoral (–) Any external exam
Curvilinear/convex array transducer  FIGURE 24.6A <ul style="list-style-type: none"> • Wide, curved footprint with large surface area decreases discomfort with applied pressure to the abdomen • Lower frequency 2-5 MHz (but with better detail than phased array probe) • Wide wedge-shaped window (at right) 	 FIGURE 24.6B	(+) Aorta, abdominal, transabdominal pregnancy (–) Cardiac

* (+) = best use; (–) = not ideal

(Photo contributor: Lawrence B. Stack, MD. Illustration contributor: Robinson M. Ferre, MD.)

Hyperechoic/echogenic—Used to describe objects on the ultrasound screen that are bright and therefore reflect sound waves.

Hypoechoic/anechoic—Used to describe objects on the ultrasound screen that

are dark and therefore transmit sound waves.

Echotexture—The characteristic appearance of specific organs when viewed using ultrasound. The liver has a characteristic echotexture.

Transducer movements—When performing sonography, specific terms are used to describe characteristic movements of the transducer used to obtain the image desired. Some examples of this include dragging, rocking, fanning, and rotating ([Fig. 24.2](#)).

Transducers

Emergency sonography is performed using a wide variety of transducers. Lower frequency transducers are used to visualize structures deep within the body, while higher frequency transducers allow better image resolution of superficial anatomic structures. The size and shape of transducers are also configured for imaging specific anatomic locations. [Table 24.1](#) lists the basic transducer types frequently used in EUS.

TRAUMA ULTRASOUND

Clinical Summary

The Extended Focused Assessment with Sonography in Trauma (E-FAST) is a protocolized series of sonographic views that attempt to identify the presence or absence of fluid or air in anatomic potential spaces such as the pericardium, the peritoneum, and the thorax. The goal of this thoracoabdominal survey is to identify or exclude immediate life threats in the trauma or critically ill patient. Although initially intended for the evaluation of the trauma patient, the E-FAST examination and its component views are also extremely valuable in the evaluation of several emergent complaints and clinical conditions including the patient with undifferentiated hypotension.

Indications

- Trauma (blunt and penetrating)
- Unexplained hypotension (traumatic and nontraumatic)
- Ectopic pregnancy (to evaluate for rupture)

The FAST examination uses four primary sonographic views to evaluate the patient. It is recommended that all four views are evaluated for a complete exam, but isolated views may be obtained when indicated. The extended component of the FAST exam (termed E-FAST) incorporates imaging of both the anterior and lateral hemithoraces to identify the presence or absence of a pneumothorax or hemothorax (Fig. 24.7). It is important to note that these are not static “single” views, but a series of images obtained in each plane as the transducer is moved or “fanned” through the area of interest.

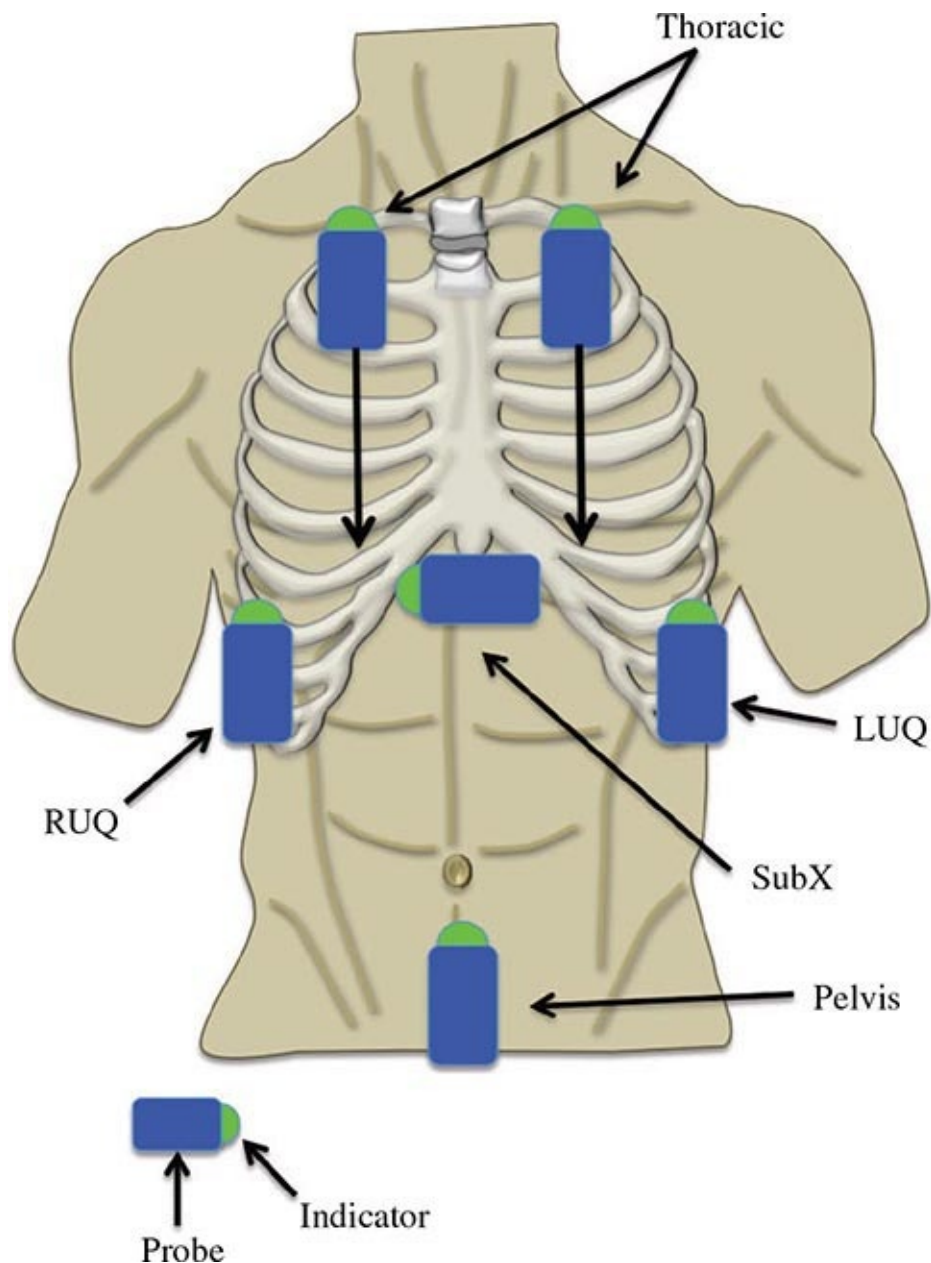


FIGURE 24.7 ■ E-FAST Trauma Series. Ultrasound transducer and probe marker positions for evaluation of pericardial fluid, hemoperitoneum, or pneumothorax. (Illustration contributor: Robinson M. Ferre, MD.)

Views for the E-FAST Examination

1. Pericardial (usually a subcostal/subxiphoid view— alternatively, one may obtain a parasternal long view of the heart if a subxiphoid view is unobtainable)
2. Right upper quadrant (RUQ; pouch of Morison)
3. Left upper quadrant (LUQ) (perisplenic view)
4. Suprapubic (pelvic view)
5. Right thorax
6. Left thorax

Patient Positioning

The E-FAST exam should be done with the patient in the supine position.

Recommended Transducers for the E-FAST Examination

- Convex array (abdominal, cardiac, and lung imaging)
- Phased array (abdominal, cardiac, and lung imaging)
- Linear array (anterior chest/lung only)

The E-FAST examination is a thoracoabdominal examination. Ideally, this is done using a single transducer that can image all three of these areas, but may result in some compromise of image quality and require the use of different probes for different components of the examination.

PERICARDIAL (SUBXIPHOID VIEW)

Technique

- Direct the transducer indicator to the patient's right (in abdominal or general preset). Note that the E-FAST exam is traditionally performed with abdominal preset settings, but the subxiphoid view is often performed with cardiac preset settings as part of a full focused cardiac ultrasound exam. When this is the case, the marker dot is typically on the opposite side of the screen and the indicator is oriented to the patient's left.
- With the transducer indicator pointing to the patient's right, the transducer is directed under the xiphoid process toward the left shoulder in a horizontal or

near-coronal plane ([Fig. 24.8](#)).



FIGURE 24.8 ■ Subxiphoid View. The transducer is directed under the xiphoid process toward the left shoulder in a horizontal plane. (Photo contributor: Lawrence B. Stack, MD.)

- Pivot, sweep, and tilt the transducer as necessary to view all four cardiac chambers and pericardium.
- Identify the liver (if in view), heart, four cardiac chambers, and surrounding pericardium ([Fig. 24.9](#)).

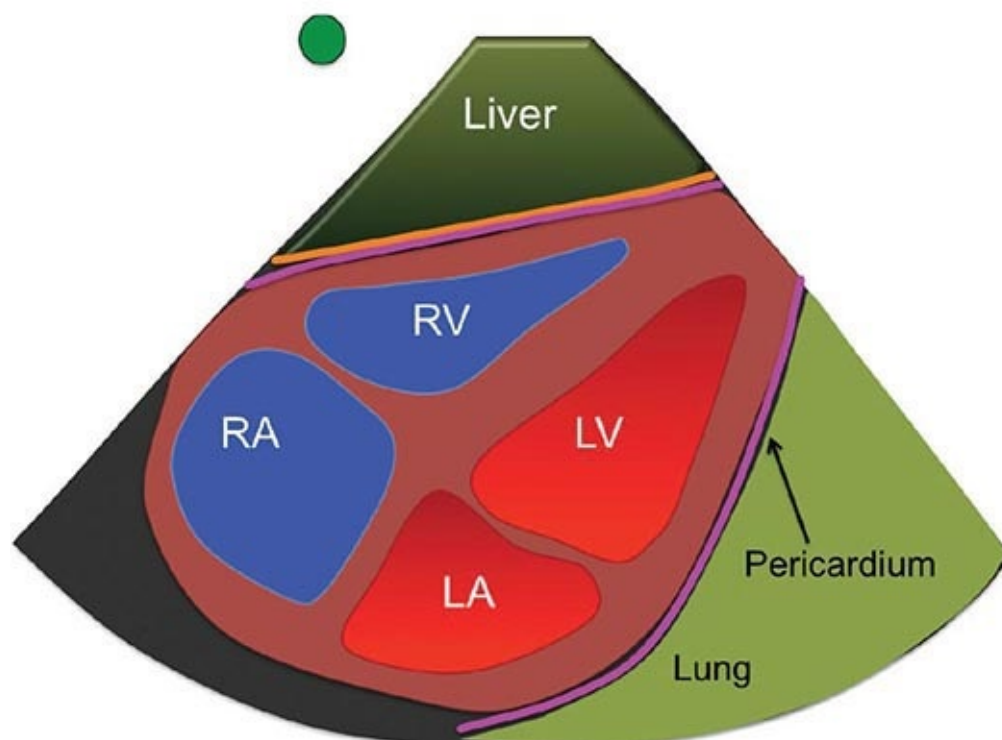


FIGURE 24.9 ■ Subxiphoid View. The heart, four cardiac chambers, and surrounding pericardium are seen in this view. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Abnormal Findings

- Hemopericardium (pericardial effusion): Pericardial fluid will appear as an anechoic (black) region noted between the pericardium and the right ventricle. As more fluid collects, the fluid will be seen completely surrounding all four chambers of the heart. Occasionally, internal echoes representing fibrin, clot, or cardiac tissue may be present within the pericardial space (Fig. 24.10).

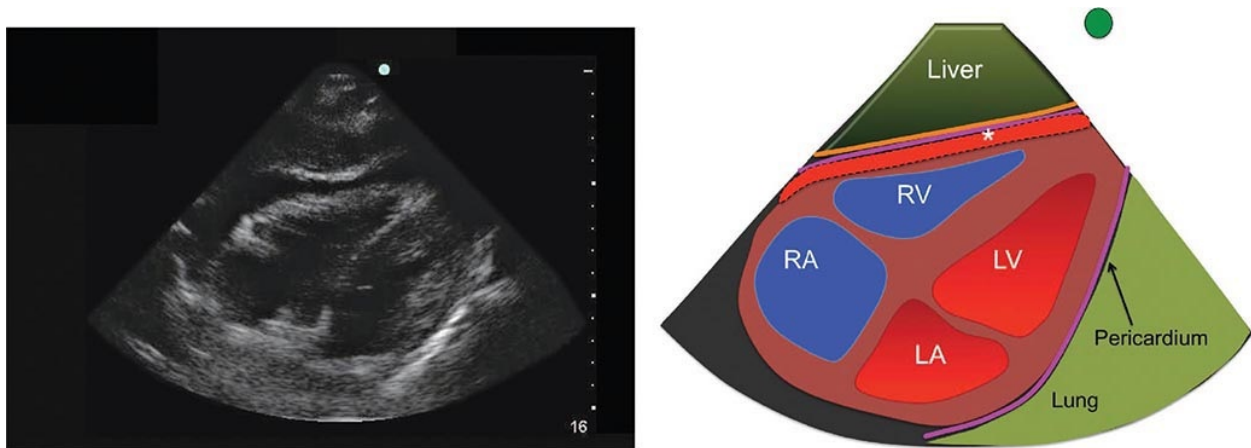


FIGURE 24.10 ■ Hemopericardium. Pericardial fluid appears as an anechoic (black) region noted between the pericardium and the right ventricle. Hemopericardium is denoted by the asterisks in the diagram. (Ultrasound contributor: Jim Fiechtel, MD; illustration contributor: Robinson M. Ferre, MD.)

- Asystole: No cardiac activity present.
- Hyperdynamic cardiac activity: Extensive cardiac contraction with near-total or complete collapse of the cardiac chambers, often associated with tachycardia and hypovolemia.

Pearls

1. When the view is obscured by gas, slide the transducer slightly to the patient's right subcostal region, and use the right lobe of the liver as the sonographic window.
2. If unable to view the heart in the true subxiphoid or subcostal window, move to a parasternal long-axis view (see "Focused Cardiac Ultrasound" later in this chapter).
3. A frequent mistake in imaging is to direct the transducer posterior toward the spine rather than cephalad toward the shoulder. You will often require less than a 30-degree angle between the transducer and the skin to view the heart.

4. Start imaging with the depth/scale setting near its maximum (eg, 24-35 cm). This should allow you to image the anterior and posterior pericardium in your initial view. Gradually decrease the depth/scale (eg, 14-18 cm) to fill the entire sector image with the heart as you continue to optimize your image.
5. Hypotensive trauma patients with a pericardial effusion and findings such as right ventricular (RV) collapse and/or a dis-tended inferior vena cava (IVC) warrant emergent pericardiocentesis (see [Fig. 24.34](#)).

RIGHT UPPER QUADRANT (MORISON'S POUCH)

Technique

- With the indicator directed toward the patient's head, the transducer is oriented in a coronal section through the body in the midaxillary line, extending from the 9th through 12th ribs. Start between the 11th and 12th ribs initially, then move cephalad or caudal, anterior or posterior, to complete the evaluation ([Fig. 24.11](#)).



FIGURE 24.11 ■ Right Upper Quadrant View. The transducer is oriented in a coronal section through the body in the midaxillary line extending from the 9th through 12th ribs. (Photo contributor: Lawrence B. Stack, MD.)

- Identify and evaluate the interface of the liver and right kidney. This region is the potential space known as Morison's pouch. Normally, the liver and

kidney are in direct contact with one another or separated by adipose tissue of heterogeneous echoes (Fig. 24.12).

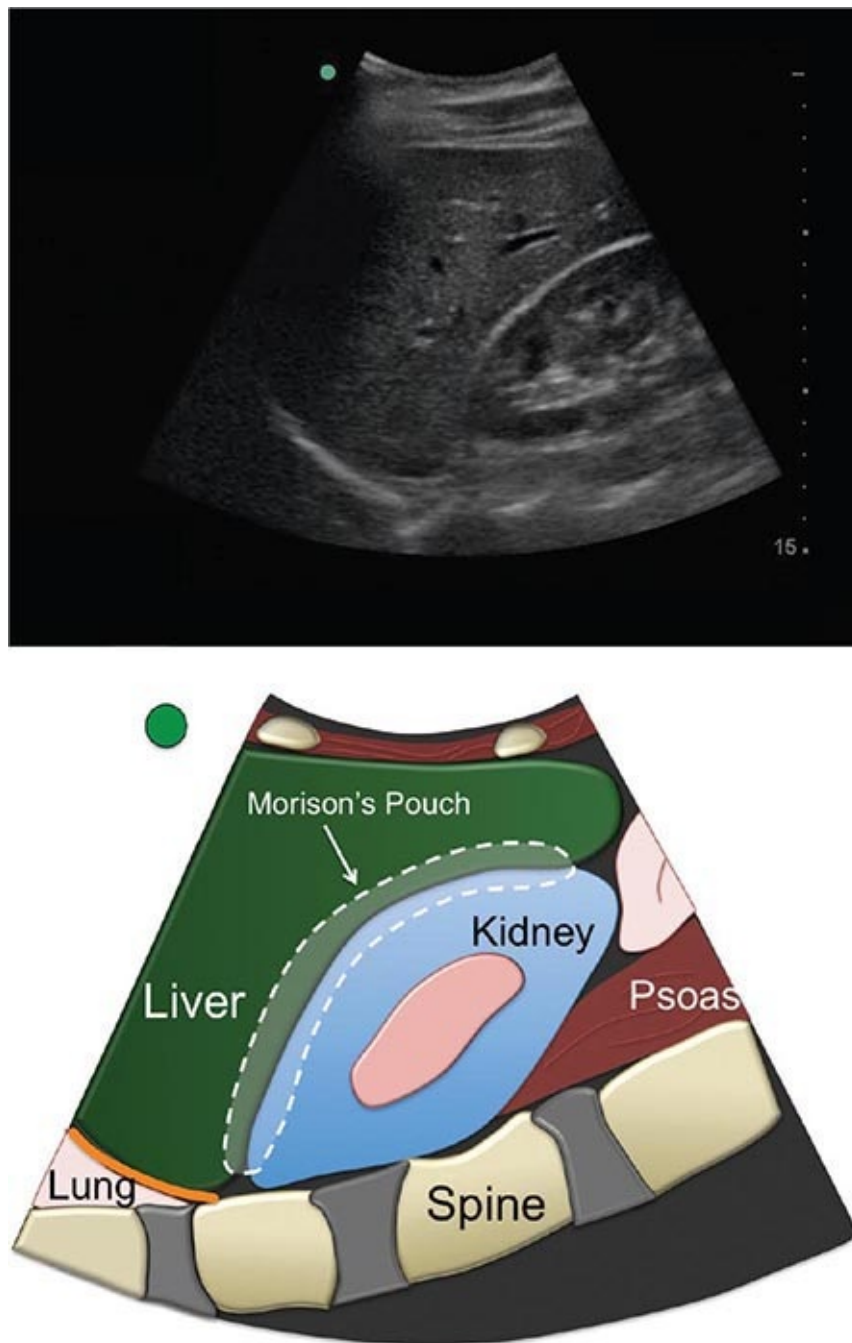


FIGURE 24.12 ■ Normal Right Upper Quadrant View (Morison Pouch). Identify and evaluate the interface of the liver and right kidney. This region is the potential space known as the pouch of Morison. Normally, the liver and kidney are in direct contact with one another or separated by adipose tissue of heterogeneous echoes. The reflection of the anterior border of the lumbar spine is visualized deep to the kidney and terminates at the costophrenic angle in the absence of fluid within the thoracic cavity. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration contributor: Robinson M. Ferre, MD.)

- Evaluate the right paracolic gutter by identifying and evaluating the caudal-most portion of the hepatic parenchyma (inferior tip of the liver).

Abnormal Findings

- Hemoperitoneum: Anechoic (black) region between the liver and right kidney or in the paracolic gutter. Rarely is blood solely seen collecting between the diaphragm and the liver in the subdiaphragmatic space ([Fig. 24.13](#)).

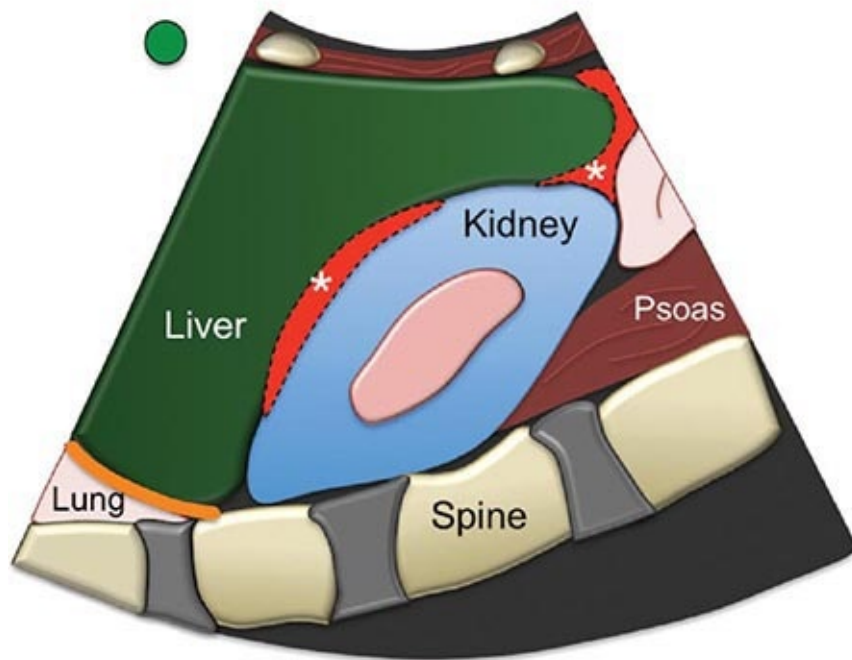


FIGURE 24.13 ■ Hemoperitoneum (Right Upper Quadrant view). An anechoic (black) region between the liver and right kidney is fluid (blood in this case) accumulating within the potential space. The diagram illustrates the typical locations of these peritoneal potential spaces, marked by asterisks. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration contributor: Robinson M. Ferre, MD.)

- Solid organ injury: Ultrasound is an insensitive exam for solid organ injury. Injuries such as hepatic and renal lacerations as well as organ rupture have been described but are not the goal of this examination.

LEFT UPPER QUADRANT (PERISPLENIC VIEW)

Technique

- The transducer indicator is directed toward the axilla.
- With the indicator pointed toward the patient's head, the transducer is oriented in a coronal section through the body in the mid to posterior axillary line extending from the 9th through 12th ribs. Start between the 11th and 12th ribs initially, then move cephalad or caudal, anterior or posterior, to complete the evaluation ([Fig. 24.14](#)). As a general rule of thumb, the perisplenic view is more posterior and cephalad than that of the RUQ view.



FIGURE 24.14 ■ Left Upper Quadrant View. The transducer is oriented in a coronal section through the body in the mid to posterior axillary line extending from the 9th through 12th ribs. (Photo contributor: Lawrence B. Stack, MD.)

- Identify and evaluate the area surrounding the spleen, including its upper and lower poles, the interface with the diaphragm, and the interface with the left kidney. Normally, the surrounding tissues of the spleen and kidney are in direct contact with one another ([Fig. 24.15](#)).

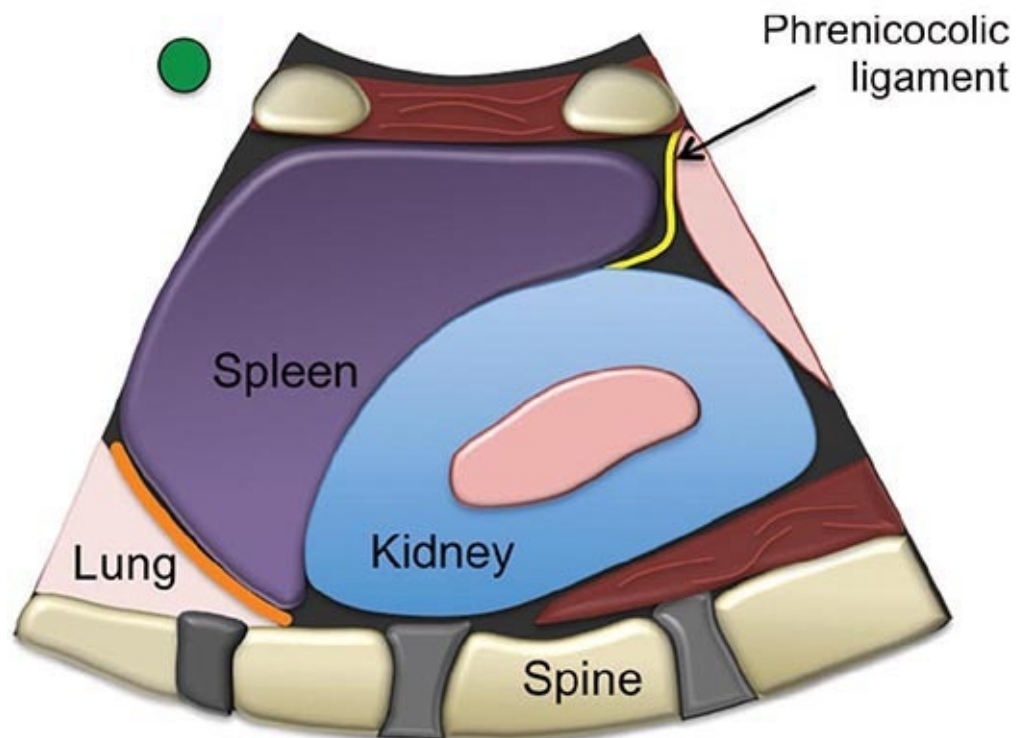
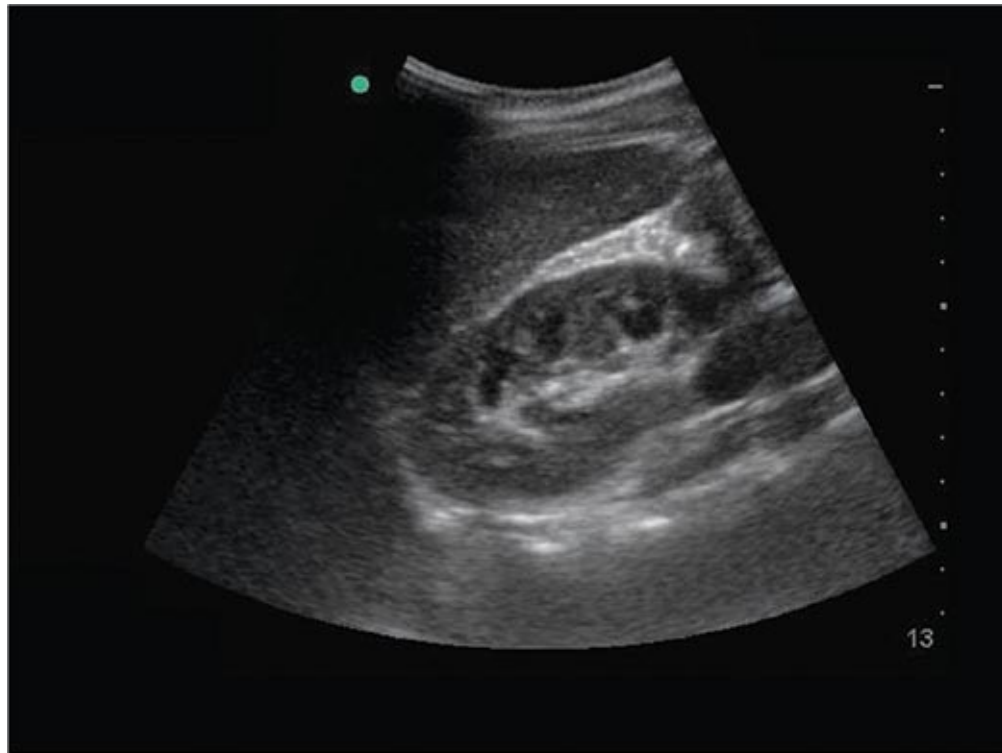


FIGURE 24.15 ■ Normal Left Upper Quadrant View. The region between the spleen and left kidney is a physiologic potential space (splenorenal recess); however, due to the phrenicocolic ligament (rarely seen on ultrasound but shown on illustration), free fluid is often shunted toward the subdiaphragmatic space between the diaphragm (orange line) and spleen. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration contributor: Robinson M. Ferre, MD.)

Abnormal Findings

- Hemoperitoneum: Anechoic (black) region around the spleen. This may be visible at the superior or inferior poles of the spleen, between the spleen and the diaphragm or between the spleen and left kidney. Unlike the Morison's pouch, blood cannot flow beyond the inferior pole of the spleen down the paracolic gutter due to the phrenicocolic ligament. Blood that collects in the perisplenic space must first pass out of the lesser peritoneal sac and into the greater peritoneal sac before it will be seen collecting along the left paracolic gutter (Fig. 24.16).

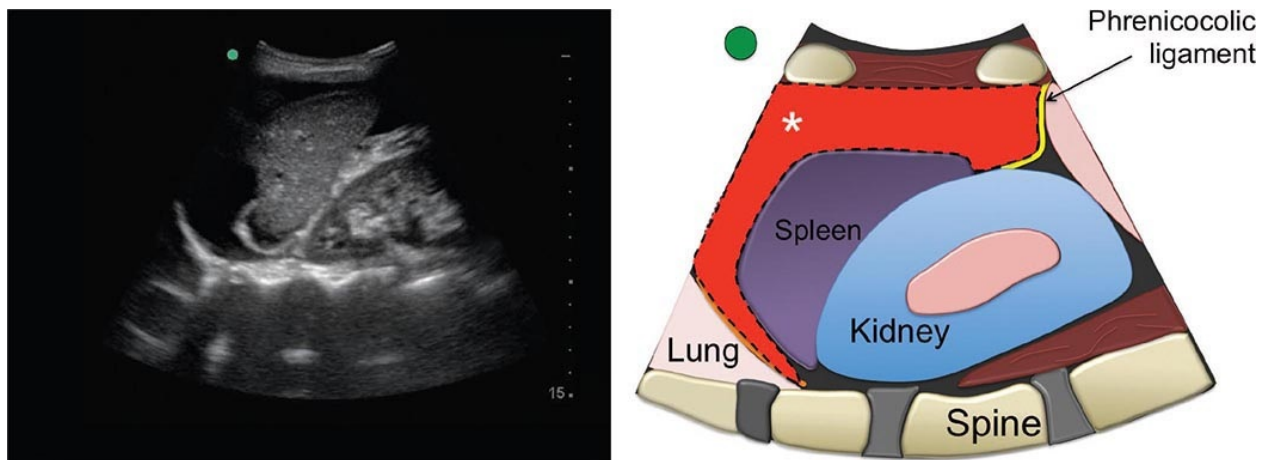


FIGURE 24.16 ■ Hemoperitoneum, Left Upper Quadrant View. In this view, fluid is seen in the subdiaphragmatic space between the diaphragm and the spleen. The diagram illustrates other common potential sites for free fluid, marked by asterisks. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration contributor: Robinson M. Ferre, MD.)

- Solid organ injury: Ultrasound is an insensitive exam for solid organ injury. Injuries such as splenic and renal lacerations as well as organ rupture have been described but are not the goal of this examination.

Pearls for RUQ and LUQ Views

1. The diaphragmatic recess includes a *superior region*, which is the inferior border of the right thorax (often referred to as the costophrenic angle), and an *inferior region (subdiaphragmatic recess)*, which is the superior border of the abdomen. Fluid in the diaphragmatic recess can represent a hemothorax when located superior/cephalad to the diaphragm or hemoperitoneum (inferior to the diaphragm) in the setting of trauma.
2. If you are uncertain whether a finding is real or artifact, evaluate it in a second

plane. Turn the transducer 90 degrees from your initial transducer position to see if the finding is still noted on the image. If the entire image is unchanged, it is less likely to be an artifact.

3. The liver is affixed to the diaphragm via the coronary ligament but the spleen lacks a similar attachment. Dependent fluid collects in the left subdiaphragmatic area more often than the right and this space should be evaluated sono-graphically during the examination.

SUPRAPUBIC

Technique

Sagittal View (Longitudinal)

- With the indicator oriented toward the patient's head, the transducer is placed just above the symphysis pubis and is directed into the pelvis ([Fig. 24.17](#)).



FIGURE 24.17 ■ Suprapubic Sagittal View. The transducer indicator is oriented toward the patient's head and the transducer is placed just above to the symphysis pubis and is directed into the pelvis. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the bladder (triangular in this view when fully dis-tended), uterus

(pear-shaped if present), prostate, seminal vesicles, and rectum (Figs. 24.18 and 24.19).

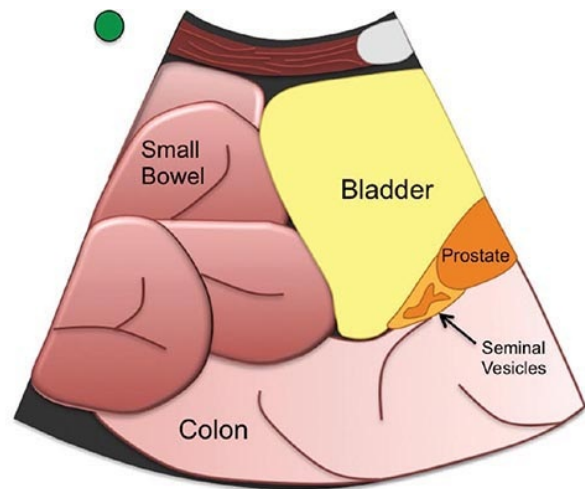


FIGURE 24.18 ■ Normal Male Pelvis, Sagittal View. The bladder appears triangular in this view. The prostate and seminal vesicles can be seen to the right of the screen, just deep to the bladder. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

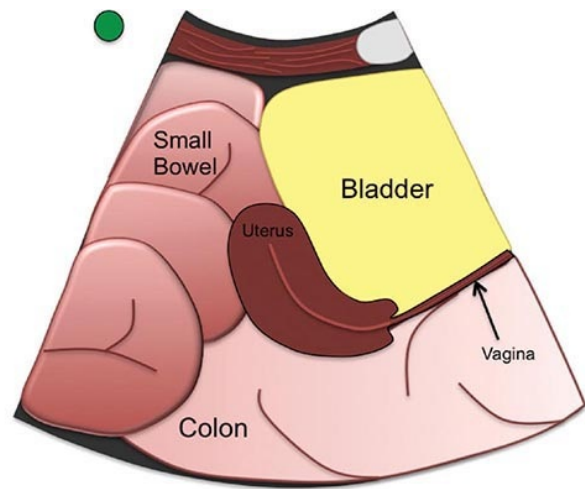


FIGURE 24.19 ■ Normal Female Pelvis, Sagittal View. The uterus appears pear-shaped in this view, and may be anteverted or retroverted. The “pouch of Douglas” is a potential space between the uterus and the rectum, and is deep to the uterus. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Technique: Transverse View

- With the indicator oriented toward the patient’s right, the transducer is placed about 1 to 2 cm above the symphysis pubis and the beam is angled through the bladder into the peritoneum (Fig. 24.20).



FIGURE 24.20 ■ Suprapubic View, Transverse. The indicator is oriented to the patient's right and the beam angled through the bladder into the peritoneum. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the bladder (rectangular in this view when fully dis-tended), uterus (oval hyperechoic structure if present), prostate, seminal vesicles, and rectum ([Fig. 24.21](#)).



FIGURE 24.21 ■ Suprapubic View, Transverse. The bladder appears rectangular in this view. The uterus/cervix or prostate/seminal vesicles can be seen deep to the bladder. (Ultrasound contributor: Jeremy S. Boyd, MD.)

Abnormal Findings

- Hemoperitoneum (male pelvis):** Free fluid in the male pelvis is seen as anechoic (black) areas filling the rectovesicular space, the potential space between the bladder and the rectum. The rectovesicular space is immediately cephalad to the extraperitoneal prostate and seminal vesicles. In the sagittal view, it is easier to see this junction that distinguishes the peritoneal cavity from the extraperitoneal pelvis. A large amount of free fluid may appear as loops of bowel floating lateral to the bladder in the transverse view and superior to the bladder in the sagittal view (Figs. 24.22 and 24.24).

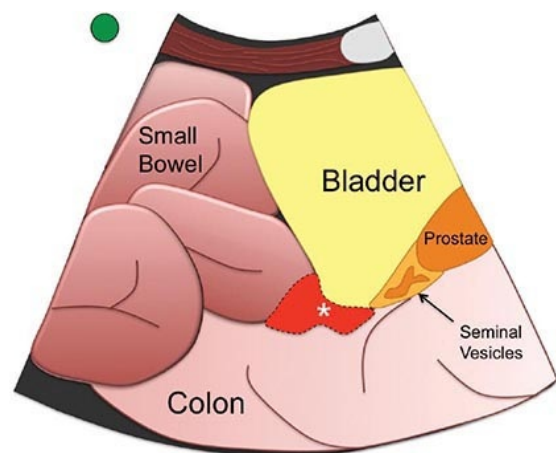
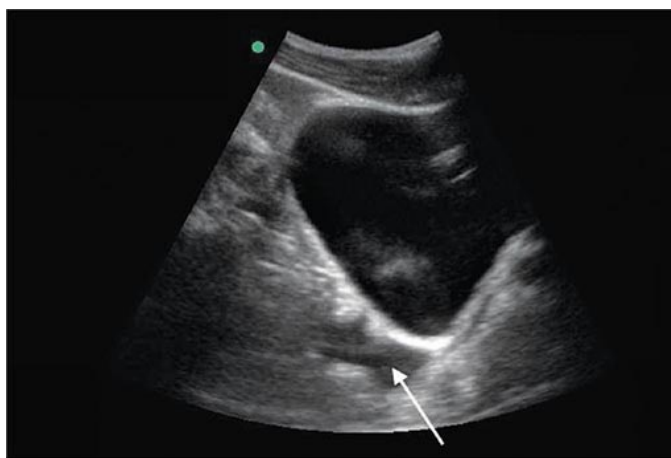


FIGURE 24.22 ■ Hemoperitoneum, Sagittal Male. Hemoperitoneum is seen deep to the bladder in the rectovesicular area on this sagittal view (arrow). Free fluid is seen as an anechoic (black) area in the rectovesicular space as demarcated by the asterisk. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

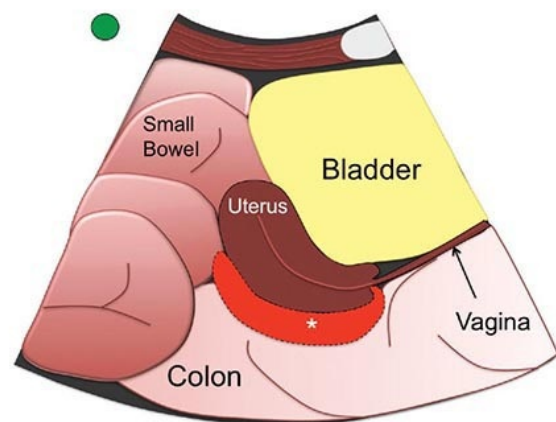


FIGURE 24.23 ■ Hemoperitoneum, Sagittal Female. Hemoperitoneum is seen deep to the uterus, in the pouch of Douglas. Free fluid is seen as an anechoic (black) area in the rectouterine space as demarcated by

the asterisk. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

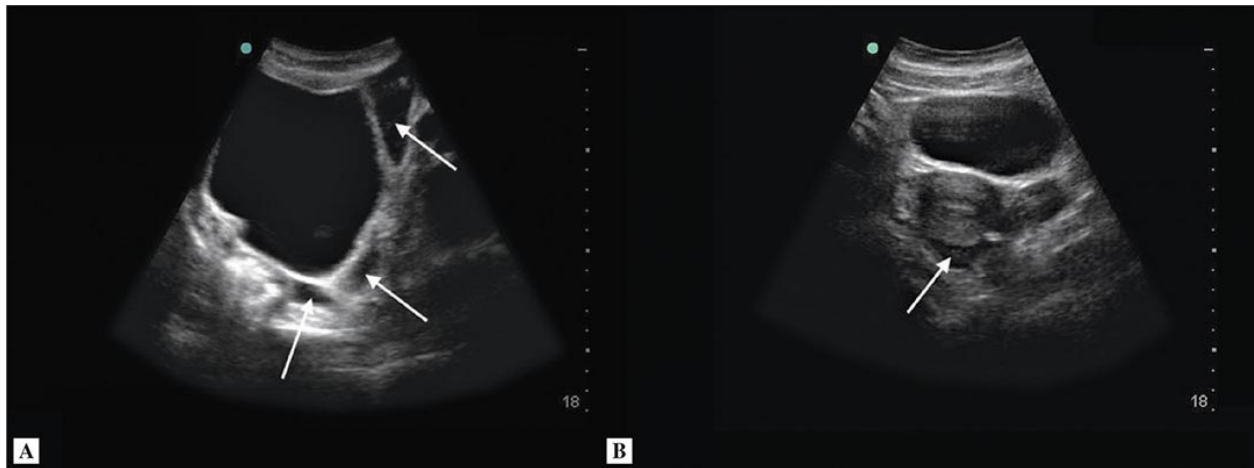


FIGURE 24.24 ■ Hemoperitoneum, Transverse Male (A) and Female (B). Hemoperitoneum (arrows) is seen deep and lateral to the bladder in the male pelvis (A), and deep to the uterus in the female pelvis (B). (Ultrasound contributor: Jeremy S. Boyd, MD; Robinson M. Ferre, MD.)

- **Hemoperitoneum (female pelvis):** Free fluid in the female pelvis is seen as an anechoic (black) area in the rectouterine space, the potential space between the uterus and rectum. The vaginal stripe is an extraperitoneal structure and intraperitoneal fluid will not be seen collecting beneath it. A large amount of free fluid may appear as loops of bowel floating lateral to the bladder in the transverse view and superior to the bladder in the sagittal view (Figs. 24.23 and 24.24).

Pearls

1. It is important to remember that the bladder is within the pelvis; therefore, the transducer must be directed cephalad through the bladder into the peritoneum to evaluate for intraperitoneal fluid or hemorrhage in the rectovesicular or rectouterine space. If the prostate or vaginal stripe can be seen on a transverse suprapubic view, then the beam is directed caudal to the peritoneum and is missing the peritoneal cavity entirely.
2. When in the sagittal plane, rotate the transducer 90 degrees counterclockwise directing the transducer indicator toward the patient's right to obtain a transverse view.
3. An empty bladder will not be able to sufficiently displace the small bowel from the pelvis. This often prevents an adequate view of the dependent portions of the pelvis to assess for free peritoneal fluid. Filling the bladder

with a Foley catheter or waiting for the bladder to distend will allow a better view for free intraperitoneal fluid that has collected in the pelvis.

THORAX (RIGHT AND LEFT)

- Note that evaluation for thoracic fluid is best performed as an extension of the RUQ and LUQ views by sliding the transducer up into the chest in a coronal plane.

Technique

- A linear or convex transducer is preferred for evaluation of pneumothorax.
- A convex or phased array transducer is preferred for evaluation of hemothorax.
- The transducer indicator is oriented toward the patient's head.

Suspected Pleural Fluid (Hemothorax)

- With the indicator directed toward the patient's head, the transducer is placed in the 5th or 6th intercostal space in the midaxillary line.
- Identify the liver (on the right) or the spleen (on the left), the diaphragm, and the vertebral bodies that lie at the bottom of the screen.
- In the fully inflated healthy lung, air prevents direct visualization of structures deep to the interface of the diaphragm and the visceral pleura of the lung. If fluid is present, it is identified as an anechoic (black) area that is caudal to the lung in the costophrenic recess. A hemothorax may have a simple anechoic appearance, but may also contain heterogeneous echoes from clotted blood or portion of lacerated lung tissue.
- The presence of pleural fluid will allow direct visualization of the lung and posterior structures, including the vertebral bodies of the thoracic spine often referred to as the "spine sign" ([Fig. 24.25](#)).

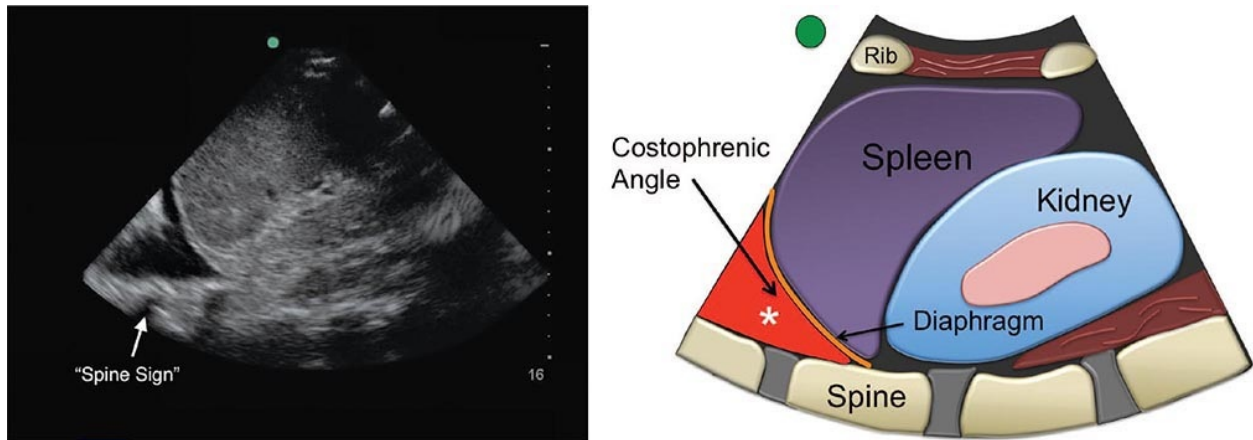


FIGURE 24.25 ■ Hemothorax. Fluid (in this case blood) is identified as an anechoic “wedge” above the diaphragm with loss of the normal mirroring artifact of the diaphragm allowing visualization of the spine extending beyond the costophrenic angle, also known as the “spine sign.” (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Suspected Pneumothorax

- When using ultrasound to evaluate for a pneumothorax, the transducer is placed in the superior most portion of the chest wall in a supine patient to assess for normal apposition of the pleura. Unlike chest radiography, ultrasound must look for normal apposition of the visceral and parietal portions of the pleura in a stepwise fashion. Indeed, it is important to note that ultrasound can only assess for the presence or absence of a pneumothorax in the area of the lung where the transducer is currently imaging. Thus, when using ultrasound, we assume that there is no prior history of pleural scarring or pleural adhesions.
- With the indicator directed toward the patient’s head, the transducer is placed just below the clavicle in the midclavicular line. To provide maximum sensitivity for the presence of a pneumothorax, the probe should be dragged caudal in the midclavicular line until the diaphragm is seen just below the costal margin ([Fig. 24.26](#)).



FIGURE 24.26 ■ Anterior Thorax View. A linear transducer is placed below the clavicle in the midclavicular line dragged inferiorly to assess multiple rib spaces for pneumothorax. Other probe types may be used as well. (Photo contributor: Lawrence B. Stack, MD.)

- Once the probe is placed on the chest wall, identify the pectoralis and intercostal muscles and ribs with associated acoustic shadows that project to the bottom of the screen. The interface of the visceral and parietal pleura, known as the pleural line, will be seen immediately deep to the rib and intercostal muscle.
- The depth should be adjusted so that the pleural line is positioned in the middle or upper half of the screen.
- If there is normal apposition of the visceral and parietal pleura (no pneumothorax), sliding will be seen along the pleural line during normal respiration. If a pneumothorax is present, no sliding will be seen ([Fig. 24.27](#)).

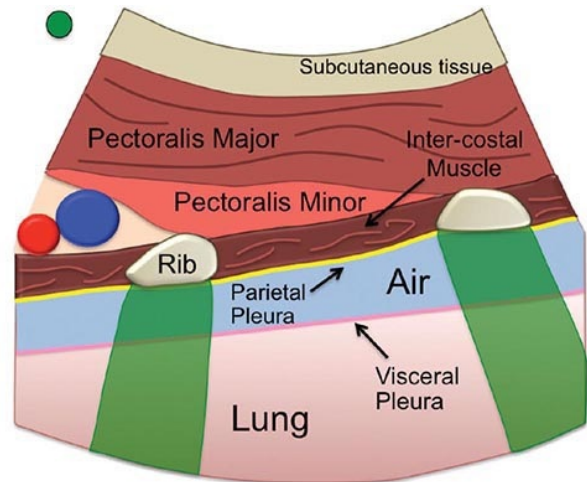
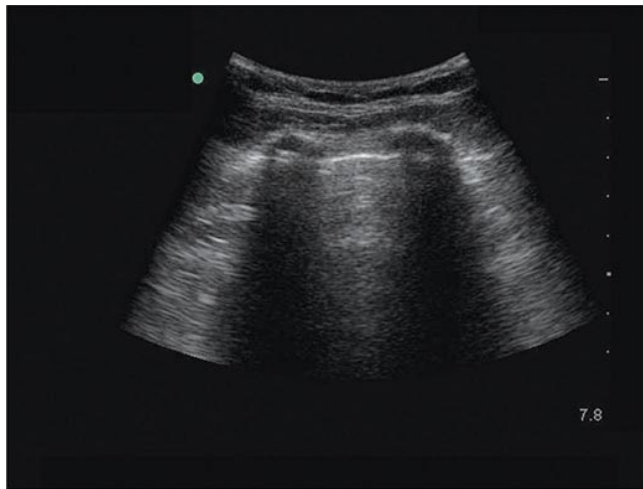


FIGURE 24.27 ■ Pneumothorax. A still image of a pneumothorax as seen on ultrasound using a curvilinear or convex array transducer. The diagram illustrates the layer of air between the pleura causing the absence of “lung sliding” seen in real-time imaging. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

- B-lines, a type of comet tail artifact, are occasionally seen arising from the pleural line and will project to the bottom of the screen (see “Lung Ultrasound”). B-lines are only present if there is normal apposition of the visceral and parietal pleura. In essence, they “rule out” a pneumothorax in the area of lung that is being imaged. If more than two B-lines are present in any one view, it indicates the presence of fluid within the extravascular lung tissue, which may indicate pulmonary contusion in the setting of trauma.
- M-mode can assist with the detection and documentation of normal lung sliding. The “seashore sign,” described as “waves on the beach,” is noted with M-mode when no pneumothorax is present ([Fig. 24.28](#)). Conversely, a “stratosphere sign” is seen when a pneumothorax is present, the result of the M-mode tracing only a reverberation artifact ([Fig. 24.29](#)).

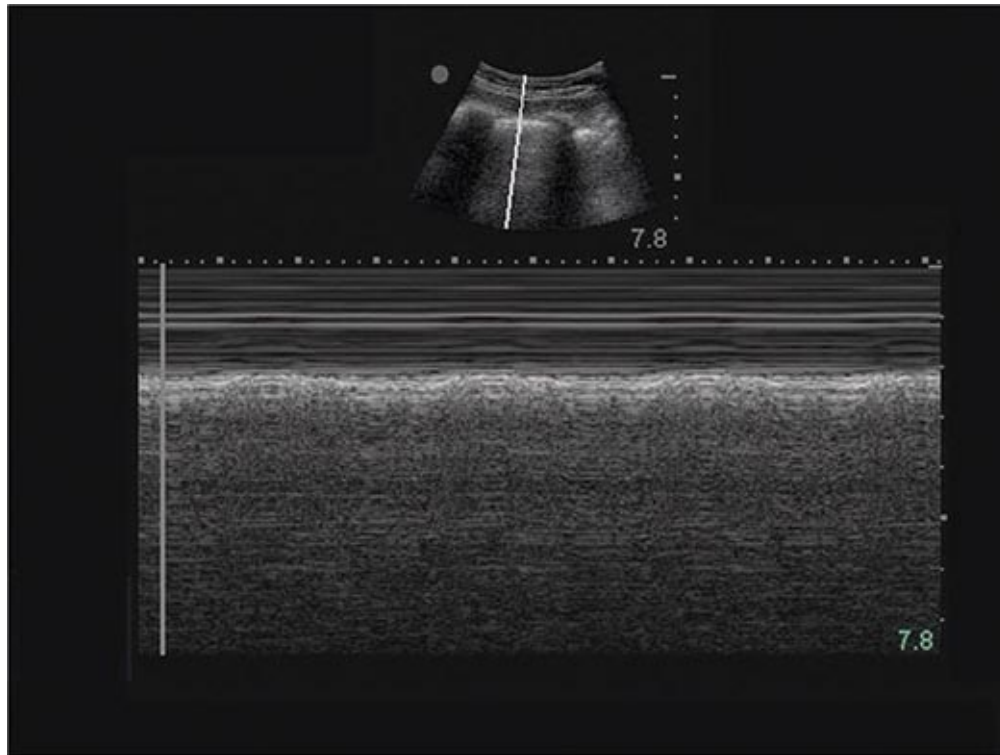


FIGURE 24.28 ■ Seashore Sign. The “seashore sign” describes the M-mode appearance on thoracic ultrasound when there is no pneumothorax. The near-field image resembles waves on a beach, and the far-field image resembles a sandy beach, an artifact caused by lung sliding. (Ultrasound contributor: Jeremy S. Boyd, MD.)

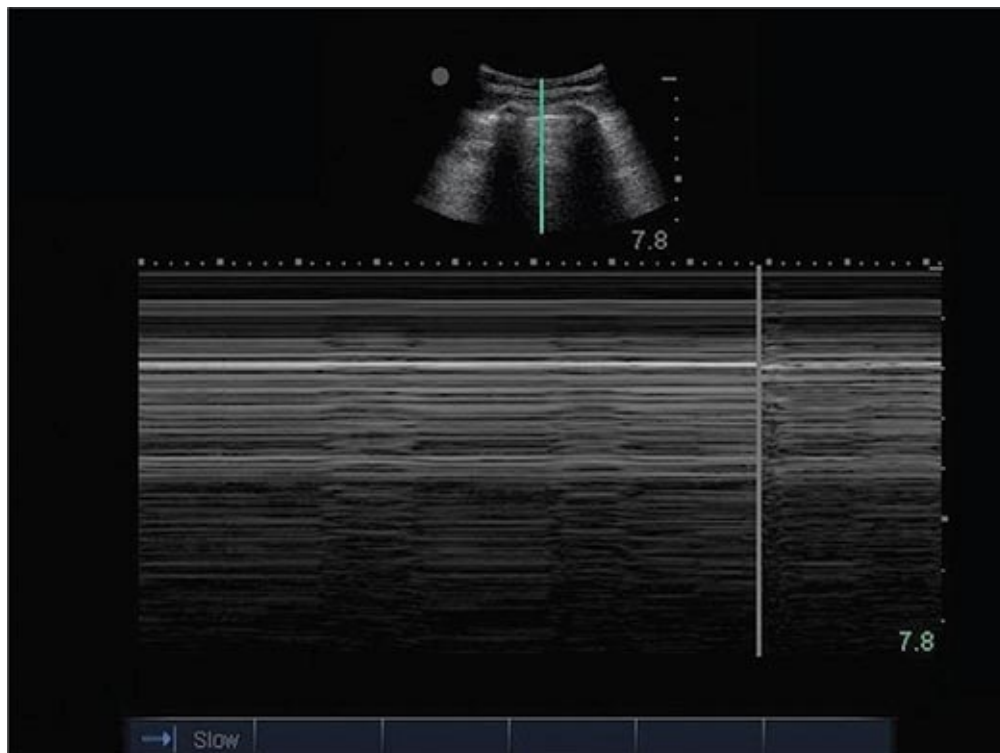


FIGURE 24.29 ■ Barcode or Stratosphere Sign (Pneumothorax). This describes the M-mode appearance on thoracic ultrasound when pneumothorax is present. The “sandy beach” is now gone due to the absence of lung sliding. (Ultrasound contributor: Jeremy S. Boyd, MD.)

Abnormal Findings

- **Hemothorax:** As visualized at the upper quadrants of the lateral chest, a hemothorax will appear as an anechoic (black) area between the diaphragm and the lung within the costophrenic recess. Heterogeneous echoes may be present due to clotted blood, lacerated lung, or other material. The presence of pleural fluid will allow direct visualization of the lung and posterior structures, including the vertebral bodies of the thoracic spine, often referred to as the “spine sign” ([Fig. 24.25](#)).
- **Pneumothorax:** The absence of sliding along the pleural line is a highly sensitive finding for pneumothorax. If lung sliding is absent, one should search for the leading edge of the pneumothorax, also known as lung point. Estimation of the pneumothorax size is possible by evaluating progressively more lateral views of the thorax in the supine patient.

Pearl

1. Lung point is the point of transition from normal lung sliding to the absence of lung sliding that occurs at the leading edge of the pneumothorax. The finding of a “lung point” is 100% diagnostic of a pneumothorax ([Fig. 24.30](#)).

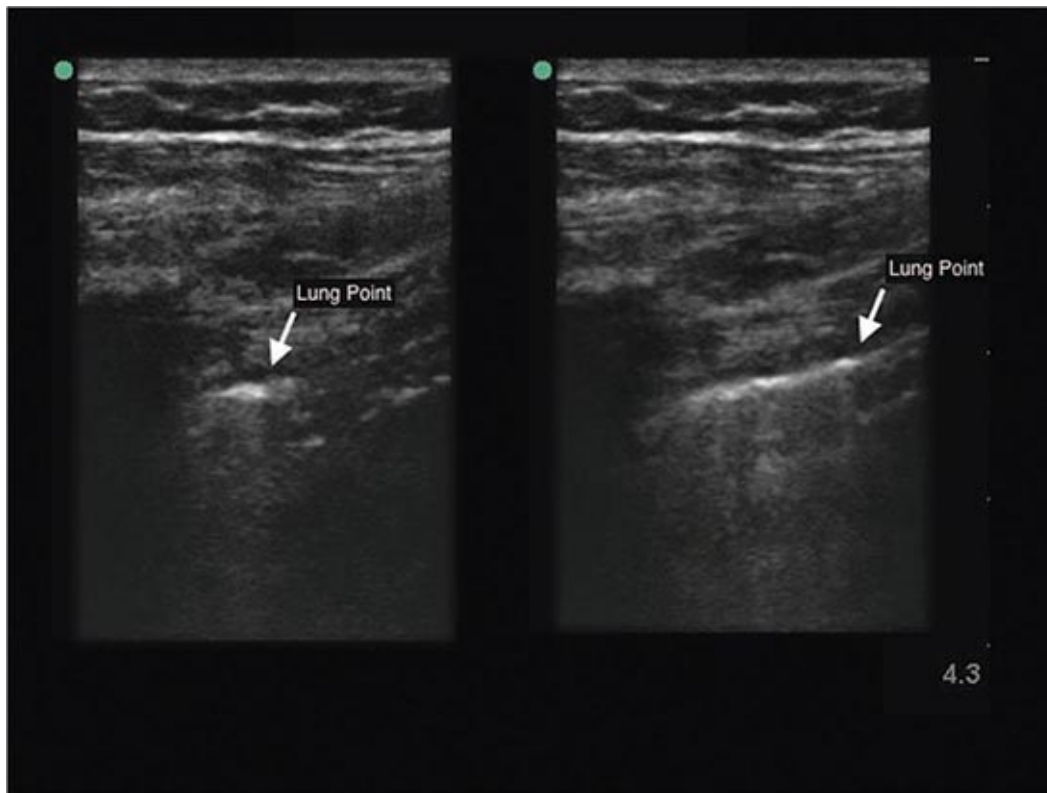


FIGURE 24.30 ■ Lung Point. The point of transition from normal lung sliding to the absence of lung sliding occurs at the leading edge of a pneumothorax. It is seen in this pair of images to move from left to right as the patient inhales. (Ultrasound contributor: Jeremy S. Boyd, MD.)

CARDIAC ULTRASOUND

Clinical Summary

Focused cardiac ultrasound can yield significant diagnostic information for the patient presenting with cardiac arrest, shock, shortness of breath, and a host of other complaints or physical findings. Although the intricacies of comprehensive echocardiography are beyond the scope of practice of most emergency medicine providers, with experience, one can incorporate focused bedside cardiac ultrasound into the diagnostic armamentarium safely to answer specific diagnostic questions such as cardiac activity, volume status, gross cardiac function, right heart strain, and the presence of pericardial effusion.

It is important to note that by convention, unlike abdominal sonography, cardiac ultrasound is viewed with the transducer indicator displayed on the right of the screen. This will require the indicator on the transducer to be directed

toward the patient's left in an anatomically transverse view. This may be disorienting for many who have not performed cardiac ultrasound before. Most ultrasound systems include cardiac presets that automatically reverse the indicator orientation to the right of the display screen. The following section describes a sonographic approach for a conventionally oriented image using standard cardiac windows.

Indications

- Cardiac arrest, pulseless electrical activity (PEA)
- Penetrating thoracic/abdominal trauma
- Unexplained hypotension or shock
- Dyspnea
- Chest pain
- Acute myocardial infarction
- Suspected aortic dissection

Specific pathologic states investigated with bedside cardiac ultrasound include asystole, cardiac activity, pericardial effusion, acute heart failure, aortic root dilatation/dissection, and right heart enlargement.

The sonographic windows for focused cardiac ultrasound include the subxiphoid view presented within the trauma/FAST examination as well as parasternal and apical views. Focused cardiac ultrasound utilizes standard views that are familiar to cardiologists and sonographers alike. These five windows allow the emergency physician to effectively and efficiently evaluate emergent cardiopulmonary pathology.

Views for Focused Cardiac Ultrasound (Fig. 24.31)

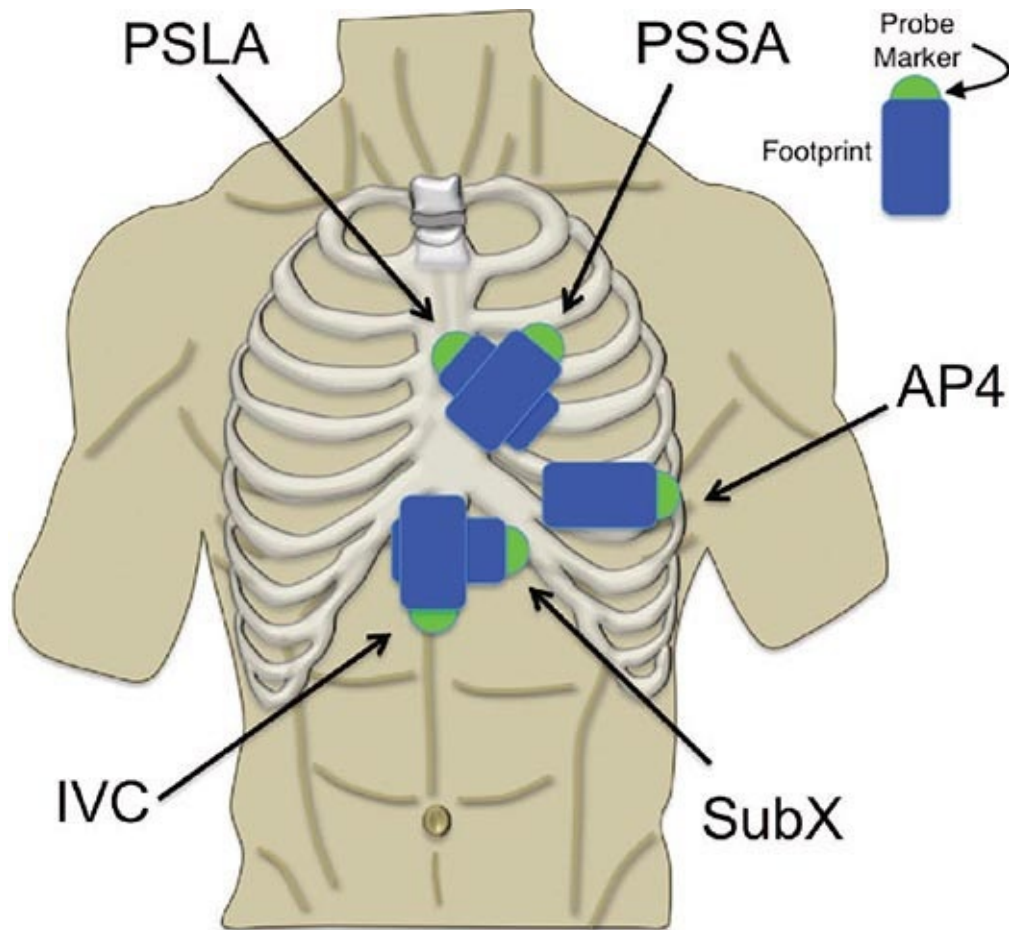


FIGURE 24.31 ■ Focused Cardiac Ultrasound. Transducer and probe marker positions for evaluation of the heart and IVC. (Illustration contributor: Robinson M. Ferre, MD.)

1. Subxiphoid/subcostal (SUBX; see “E-FAST Examination, Subxiphoid View”)
2. Parasternal long-axis view (PSLA)
3. Parasternal short-axis view (PSSA)
4. Apical four-chamber view (AP4)
5. Subxiphoid long-axis view (IVC)

The technique and common findings for each of these views are presented in the next five topics.

Patient Positioning

- The patient should be optimally positioned in the left lateral decubitus position with the left arm above the head (allowing better access to the intercostal spaces) for all but the SUBX and IVC views, which should be performed with the patient supine. If this is not feasible, the patient may be

supine for the entire study.

Recommended Transducer

- Phased array

SUBXIPHOID CARDIAC (SUBCOSTAL VIEW)

Technique

- The transducer indicator should be directed toward the left in a cardiac preset.
- With the transducer indicator pointing to the patient's left, the transducer is placed inferior to the xiphoid process and directed cephalad toward the left shoulder in a horizontal/near coronal plane (see [Fig. 24.32](#)).



FIGURE 24.32 ■ Subxiphoid View. With the transducer indicator pointing to the patient's left, the transducer is placed inferior to the xiphoid process and directed cephalad toward the left shoulder in a horizontal plane. (Photo contributor: Lawrence B. Stack, MD.)

- Pivot, sweep, and tilt the transducer as needed to view all four cardiac chambers.

- Identify the heart, four cardiac chambers, and surrounding pericardium (see [Fig. 24.33](#)).

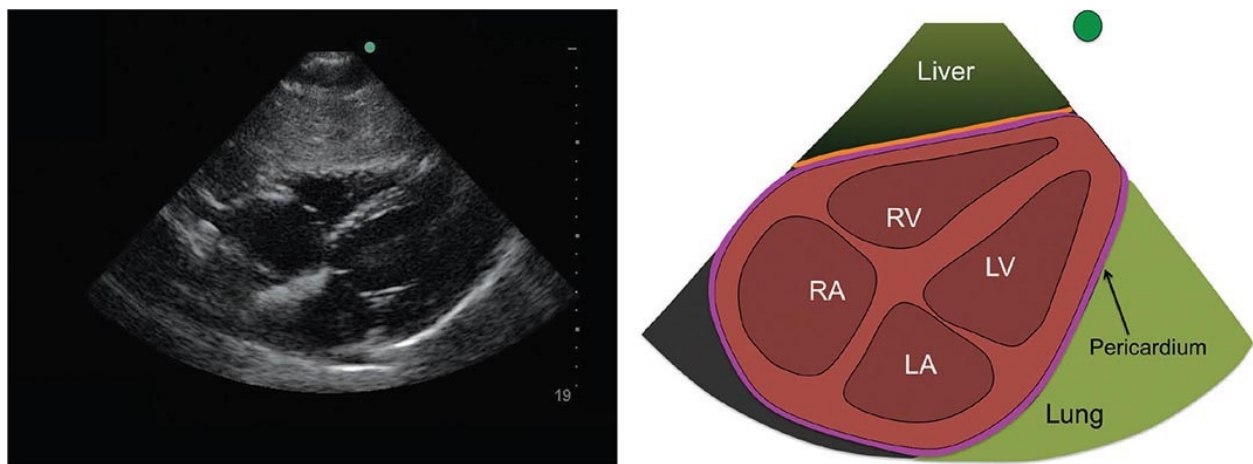


FIGURE 24.33 ■ Subxiphoid View. A normal view demonstrating the heart, four cardiac chambers, and surrounding pericardium. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Abnormal Findings

- Pericardial effusion:** Pericardial fluid will appear as an anechoic (black) region noted between the pericardium and the right ventricle. As more fluid collects, the fluid will be seen completely surrounding all four chambers of the heart. Occasionally, internal echoes representing fibrin, clot, or cardiac tissue may be present within the pericardial space ([Fig. 24.34](#)).

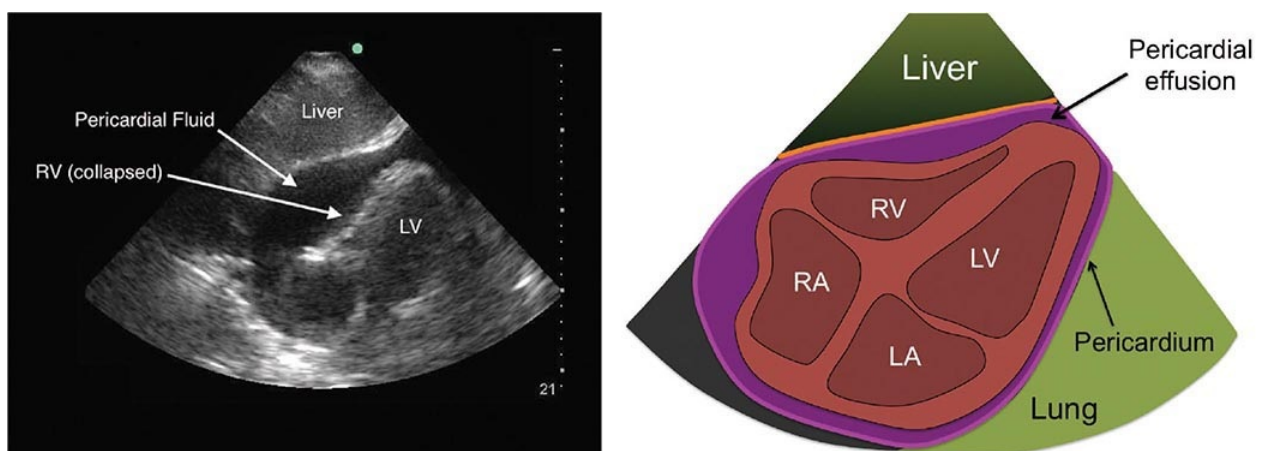


FIGURE 24.34 ■ Pericardial Effusion. Pericardial fluid will appear as an anechoic (black) region noted between the pericardium and the right ventricle. Hypotensive patients with a pericardial effusion and findings such as right ventricular collapse (illustration) and/or a distended IVC warrant consideration of emergent pericardiocentesis. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor:

Jeremy S. Boyd, MD.)

- Asystole: No cardiac activity present.
- Hyperdynamic cardiac activity: Extensive cardiac contraction with near-total or complete collapse of the cardiac chambers, often associated with tachycardia and hypovolemia.

Pearl

See “Pearls” for the “E-FAST Examination, Subxiphoid View.”

PARASTERNAL LONG-AXIS VIEW (PSLA)

Technique

- The transducer indicator is directed at the right clavicle or shoulder.
- The transducer is placed in the 4th or 5th left parasternal intercostal space and the beam is directed posteriorly ([Fig. 24.35](#)).



FIGURE 24.35 ■ Parasternal Long-Axis View. The transducer is placed in the fourth or fifth left parasternal intercostal space and the beam is directed posteriorly. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the right ventricle, left atrium, left ventricle, mitral valve, aortic valve, aortic root, aortic outflow tract, and surrounding pericardium (Fig. 24.36).

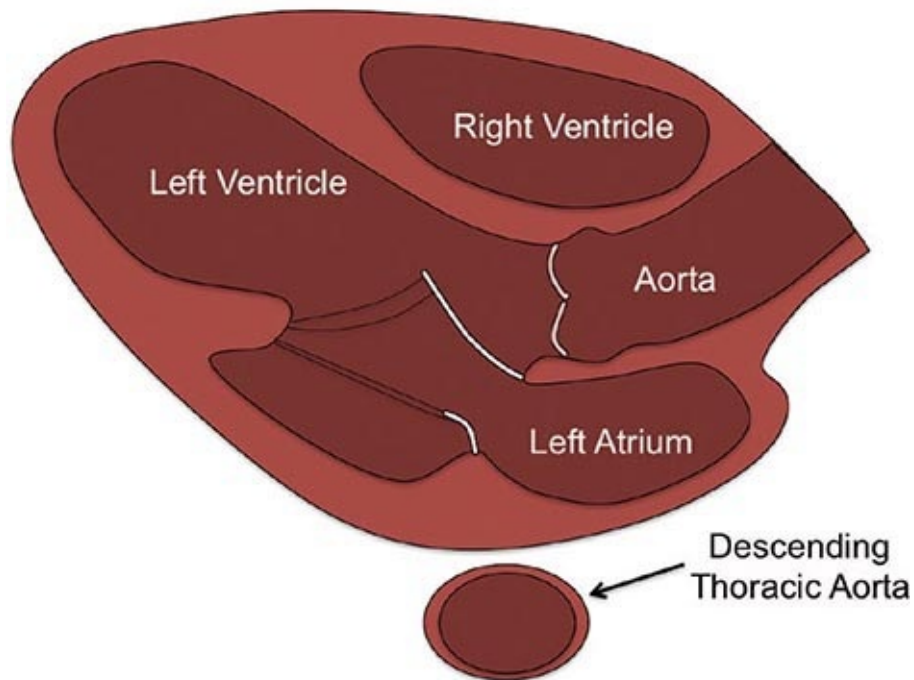
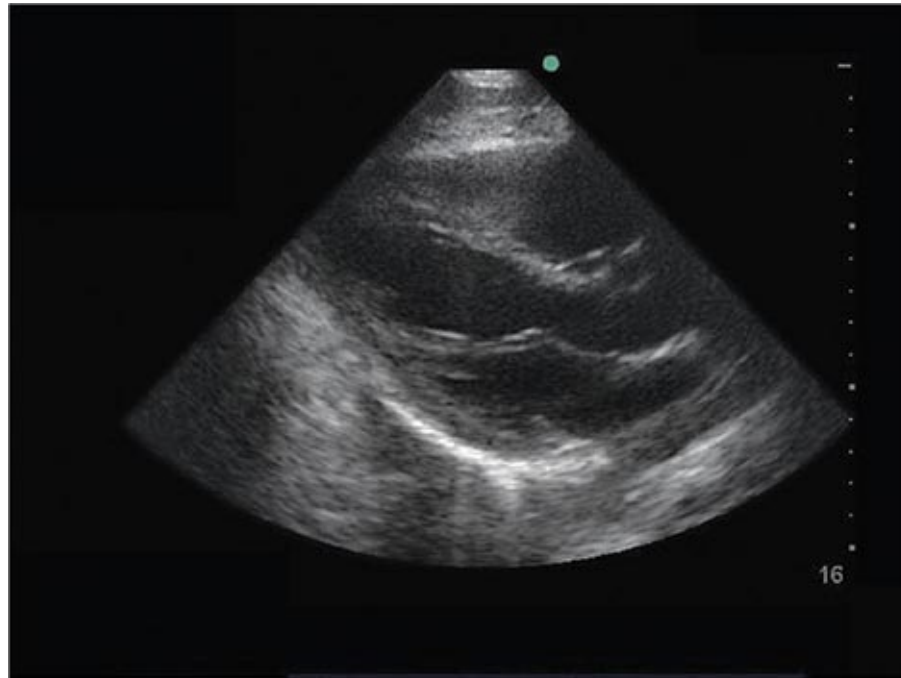


FIGURE 24.36 ■ Parasternal Long-Axis View. The right ventricle, left atrium, left ventricle, mitral valve, aortic valve, aortic root, aortic outflow tract, and surrounding pericardium are seen. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

Abnormal Findings

- Pericardial effusion: Anechoic (black) region noted between the hyperechoic (bright) pericardium and the walls of the heart (see Fig. 24.37). In this view, pericardial effusion may be distinguished from a left-sided pleural effusion by its relationship to the descending thoracic aorta.

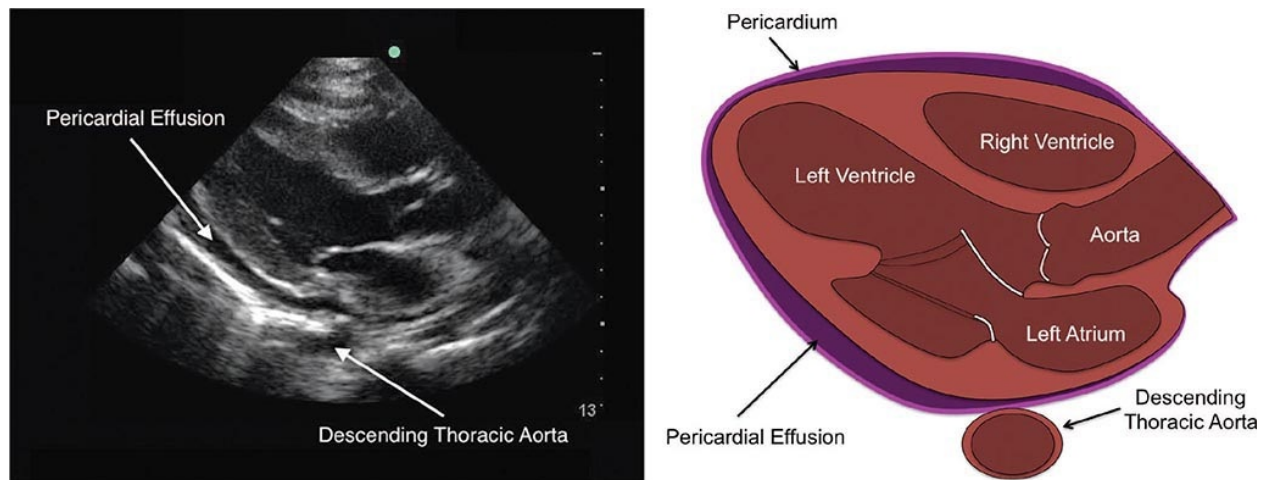


FIGURE 24.37 ■ Parasternal Long-Axis View. A pericardial effusion is seen as an anechoic (black) region between the hyperechoic (bright) pericardium and the walls of the heart. The image demonstrates a small pericardial effusion, while the illustration demonstrates the location of a larger (circumferential) effusion. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

- Aortic root dilatation: An aortic root measurement greater than 3.8 to 4.0 cm is abnormal and indicates aneurysmal dilatation that may suggest aortic dissection in the appropriate clinical setting. Further evaluation is recommended.
- Dilated descending aorta: The transverse descending thoracic aorta can be seen in the far field in this view posterior to the left atrium. A descending thoracic aorta greater than 4.0 cm is abnormal and indicates aneurysmal dilatation that may suggest aortic dissection in the appropriate clinical setting. Further evaluation is recommended.
- Poor left ventricular function: Gross estimates of left ventricular function may be made by observing the anteroposterior motion of the mitral valve, the increase in myocardial thickness with contraction, and the extent to which the anterior and posterior walls of the left ventricle approach each other in systole.

Pearls

1. A true parasternal long-axis view (a sagittal image through the heart) will visualize the aortic root within the image. If the aortic root is not present, you are likely in an oblique plane and will need to fan and/or rotate the transducer to optimize the image.
2. It is critical to make deliberate, slow, small adjustments of the transducer in imaging the heart, since even small movements at the skin surface can translate into large changes in beam angle at just 5 to 10 cm deep from the surface.
3. Normal spontaneous respiration is usually fine for cardiac imaging. Patients who are tachypneic can be very challenging, and verbally coaching the patient's breathing patterns is best. If you note a great deal of artifact due to lung interposition, place the patient in the left lateral decubitus position; have them inhale and slowly exhale while you scan. When you have an acceptable window, ask the patient to stop exhaling and hold their breath while you capture your images.
4. Remember that the parasternal long axis is approximated by a line running from the right acromioclavicular joint and the left antecubital fossa (when the arm is lying by the patient's side).

PARASTERNAL SHORT-AXIS VIEW (PSSA)

Technique

- From the parasternal long-axis position, rotate the transducer 90 degrees clockwise (to the patient's left) or place the transducer in the 4th or 5th left parasternal intercostal space in a line connecting the left clavicle/shoulder and the right hip ([Fig. 24.38](#)).



FIGURE 24.38 ■ Parasternal Short-Axis View. Place the transducer in the fourth or fifth left parasternal intercostal space in a line connecting the left clavicle/shoulder and the right hip, with the transducer rotated 90 degrees clockwise from the parasternal long-axis view. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the left ventricle (circular), right ventricle (crescent-shaped), and surrounding pericardium ([Fig. 24.39](#)).

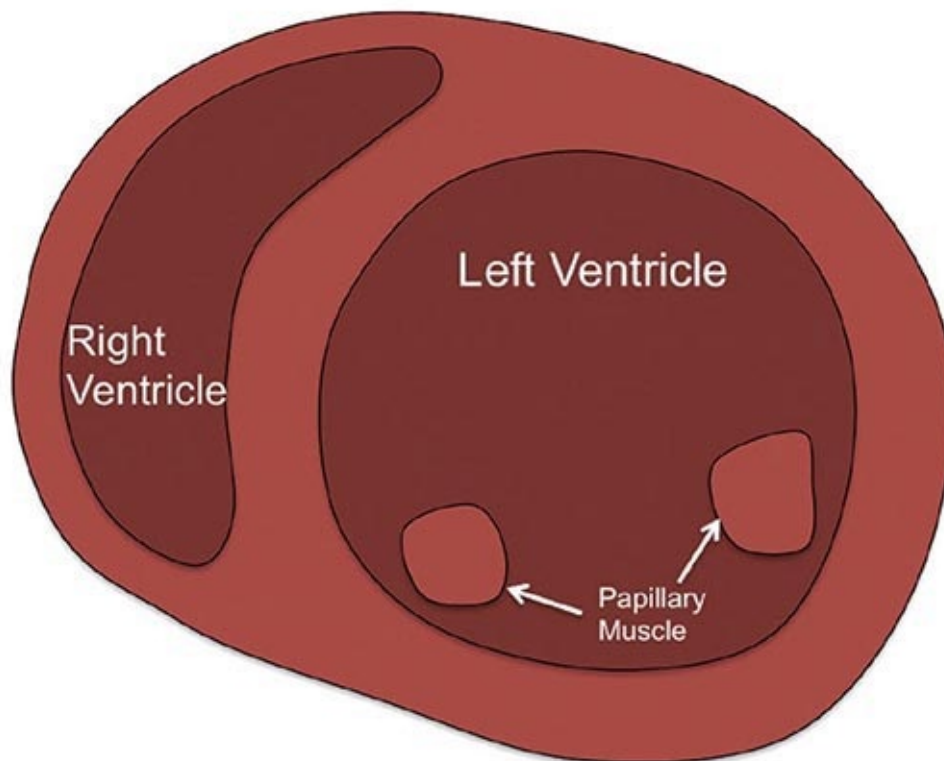
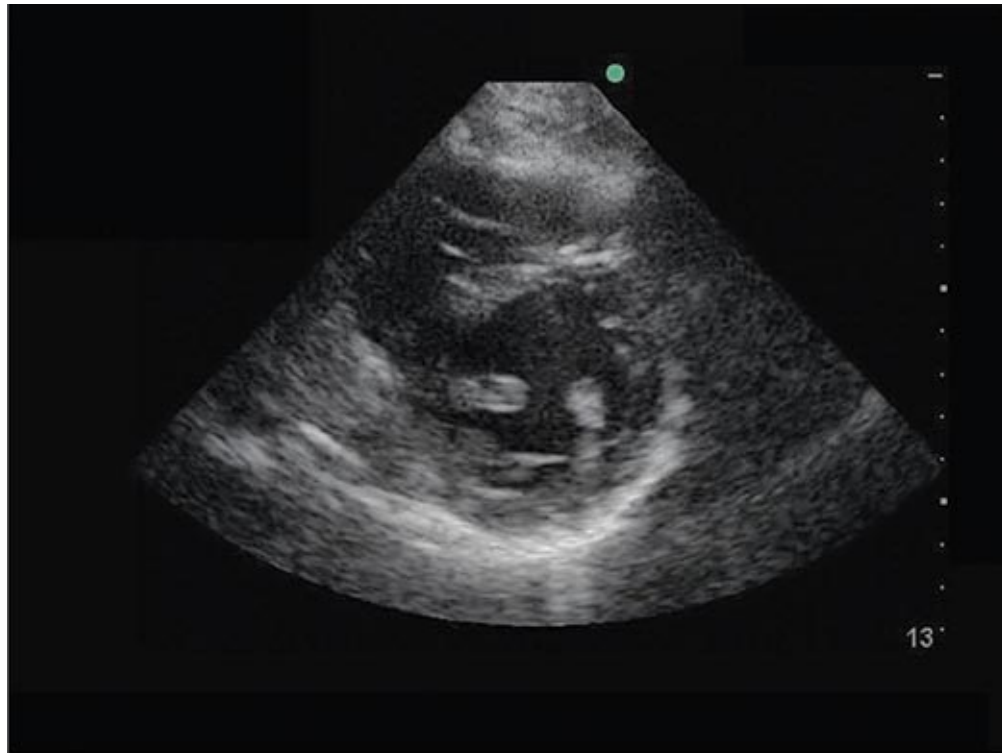


FIGURE 24.39 ■ Parasternal Short Axis. The left ventricle (circular) and right ventricle (crescent-shaped) and surrounding pericardium are seen. The papillary muscles are seen in cross-section. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

Abnormal Findings

- Pericardial effusion: Anechoic (black) region noted between the bright pericardium and the walls of the heart ([Fig. 24.37](#)).
- Dilated right ventricle: The right ventricle is normally a crescent-shaped structure. A rounded, dilated right ventricle that deforms the rounded left ventricle (creating an “OD” sign) suggests elevated right-sided pressures, as seen with pulmonary emboli and severe pulmonary hypertension ([Fig. 24.40](#)).

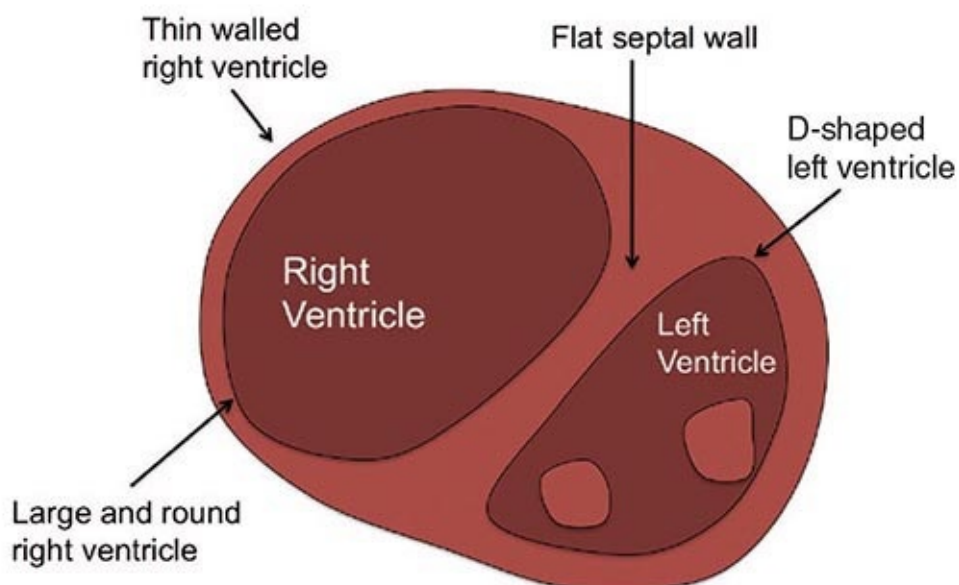
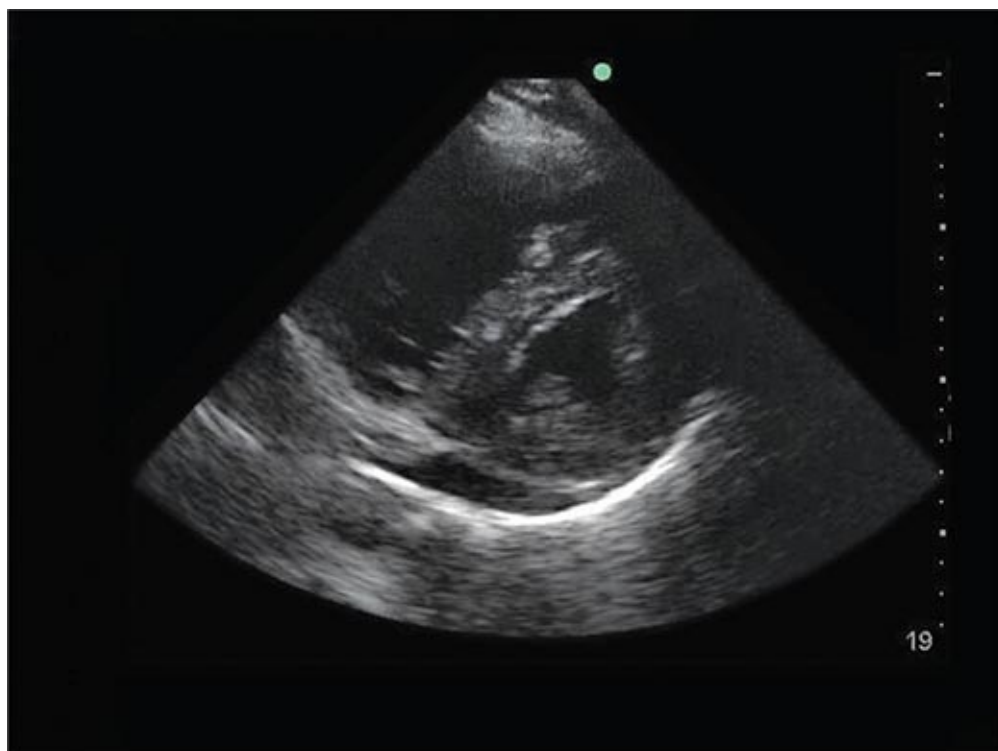


FIGURE 24.40 ■ Parasternal Short Axis. A rounded, dilated right ventricle that deforms the rounded left ventricle (creating an “OD” sign) suggests elevated right-sided pressures. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Pearl

1. The standard parasternal short-axis view is obtained with the image plane at the level of the papillary muscles. Visualization of the papillary muscles

should ensure a true transverse section through the left ventricle, and provides a prime location for the evaluation of left ventricular contraction and motion.

APICAL FOUR-CHAMBER VIEW (AP4)

Technique

- The transducer indicator is directed toward the left axilla or directly posterior to the patient.
- The transducer is placed over the cardiac apex or the point of maximal intensity with the beam directed toward the right clavicle/shoulder in a plane coronal to the heart ([Fig. 24.41](#)).



FIGURE 24.41 ■ Apical Four-Chamber View. The transducer is placed over the cardiac apex with the beam directed toward the right clavicle/shoulder in a plane coronal to the heart. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the left ventricle, right ventricle, left atrium, right atrium, tricuspid valve, mitral valve, and surrounding pericardium ([Fig. 24.42](#)).

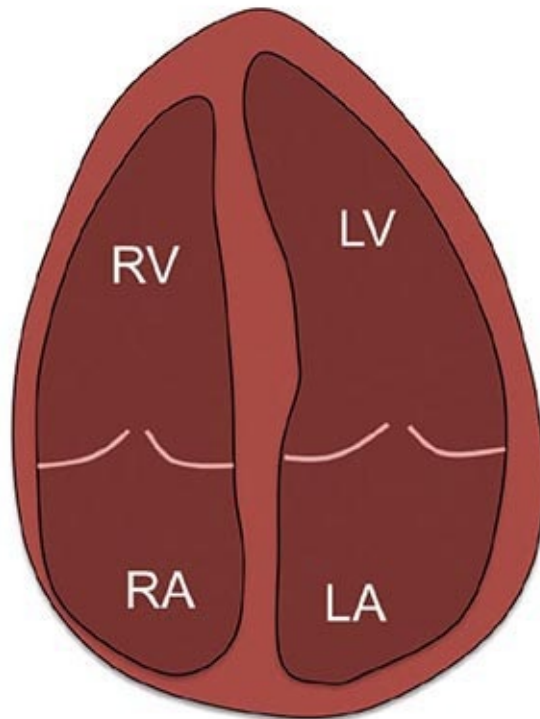
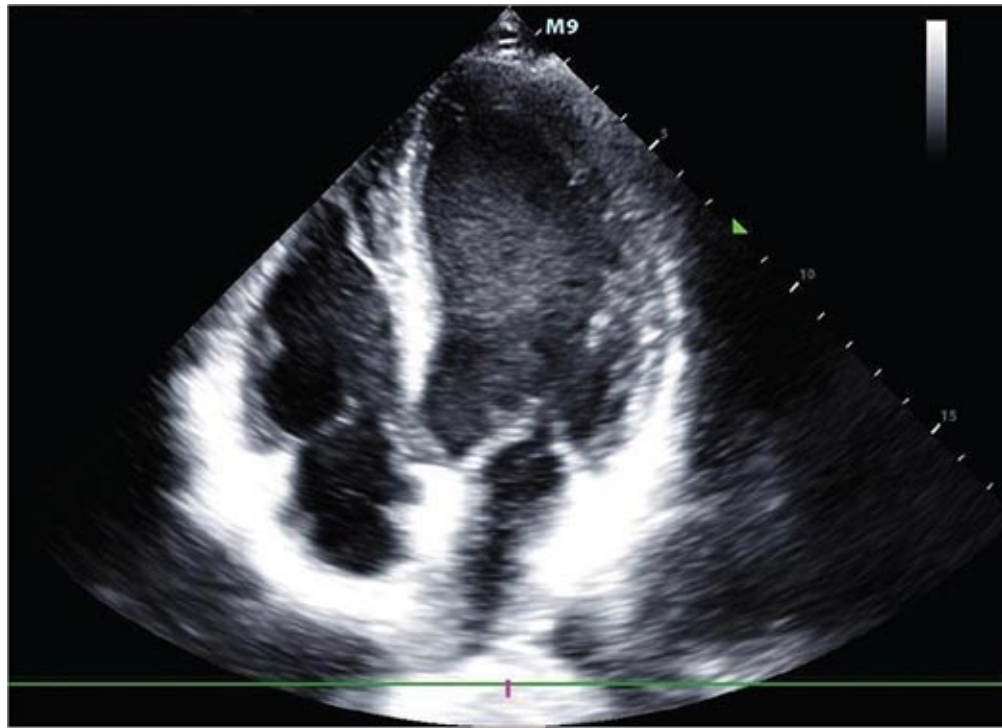


FIGURE 24.42 ■ Apical Four-Chamber View. The left ventricle, right ventricle, left atrium, right atrium, and tricuspid and mitral valves are seen. (Ultrasound contributor: Jeremy S. Boyd, MD; Illustration contributor: Robinson M. Ferre, MD.)

Abnormal Findings

- Pericardial effusion: Anechoic (black) region noted between the hyperechoic pericardium and the walls of the heart (see [Figs. 24.37](#) and [24.43](#)).

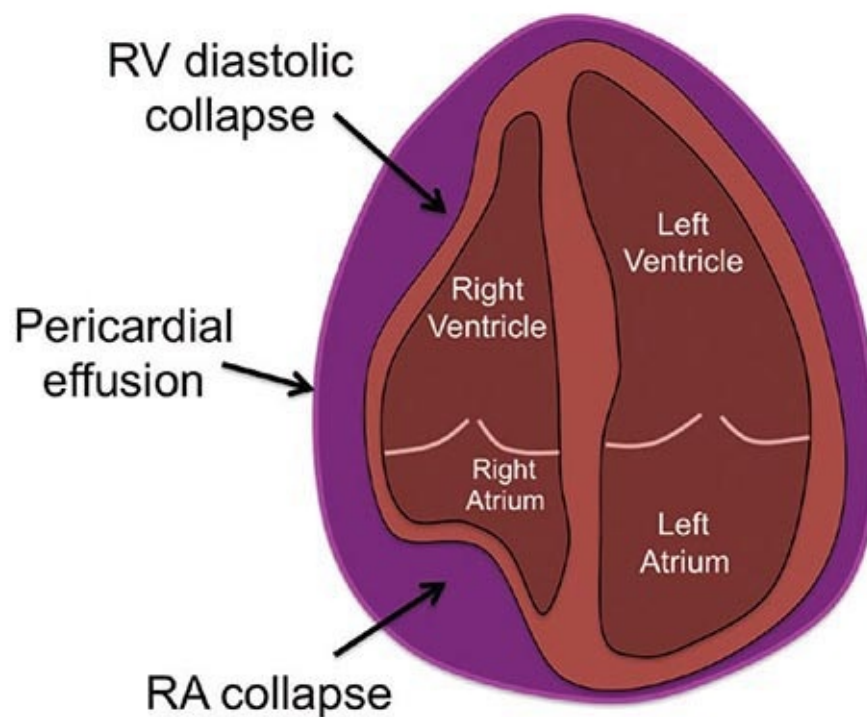
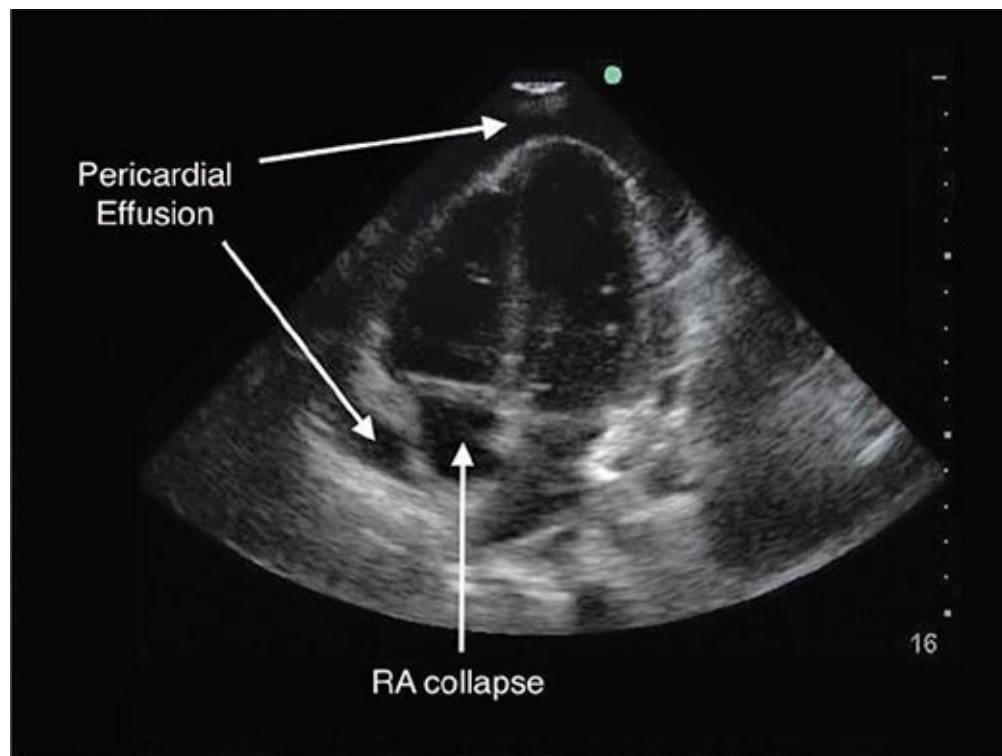


FIGURE 24.43 ■ Apical Four-Chamber View. A pericardial effusion appears as an anechoic (black) region between the hyperechoic pericardium and the walls of the heart. When tamponade physiology is

present, right atrial and right ventricular collapse may be seen. Right atrial collapse is seen in the ultrasound image. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

- Dilated right atria/ventricle: If the right ventricle is equal to or larger than the left ventricle in size, or there is poor contractility of the right heart in comparison with the left, it may suggest elevated right-sided pressures as seen with pulmonary emboli and severe pulmonary hypertension (Fig. 24.44).

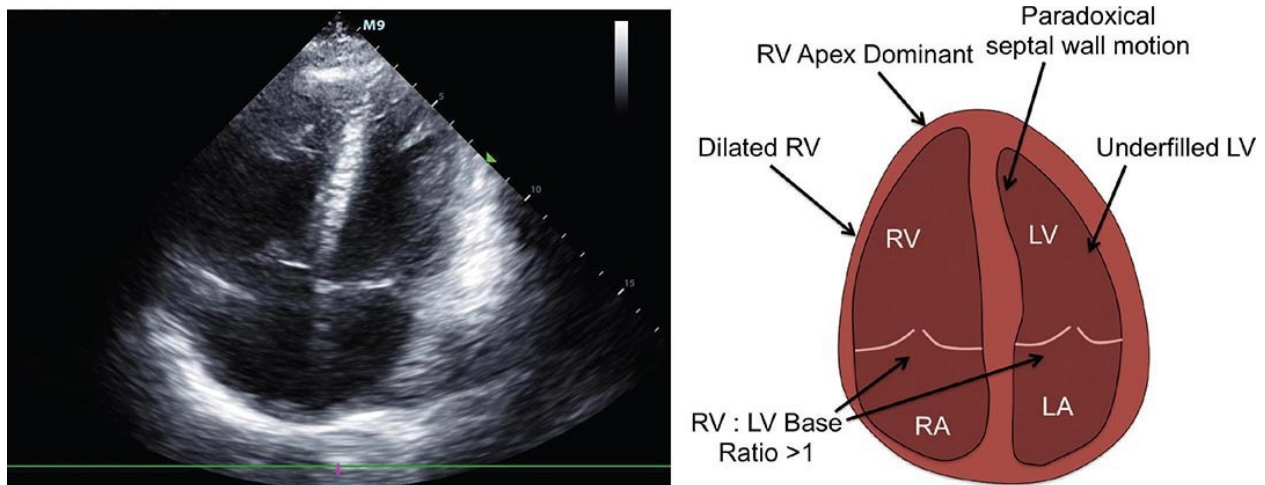


FIGURE 24.44 ■ Apical Four-Chamber View. A right ventricle that is equal in size or larger than the left ventricle suggests elevated right-sided pressures. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

- Dilated cardiomyopathy: As cardiac remodeling occurs in states of dilated cardiomyopathy, the left ventricle begins to take on more of a dilated balloon-like shape rather than its normal more streamlined bullet-like shape (Fig 24.45).

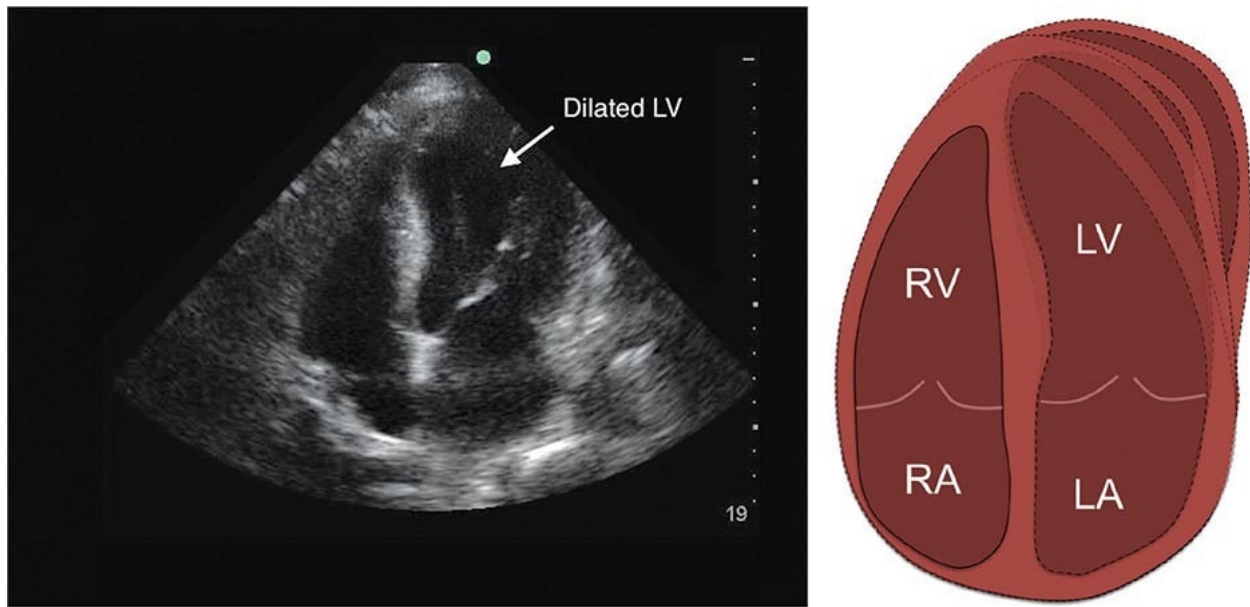


FIGURE 24.45 ■ Apical Four-Chamber View. As cardiac remodeling occurs in states of dilated cardiomyopathy, the left ventricle begins to take on more of a dilated balloon-like shape rather than its normal streamlined bullet-like shape. The diagram shows the progression of remodeling. (Ultrasound contributor: Jeremy S. Boyd, MD; Illustration contributor: Robinson M. Ferre, MD.)

2. It is critical to realize the variance in the resting position of the heart. There can be significant differences in acoustic windows from patient to patient with the four-chamber apical view.

Pearls

1. See “Pearls for the FAST Examination, Subxiphoid-Cardiac,” item 4, above.

SUBXIPHOID LONG-AXIS (IVC)

Technique

- The transducer indicator is directed toward the patient’s feet.
- The transducer is placed subxiphoid in a midline plane and tilted toward the patient’s right to identify the IVC as it enters the right atrium ([Fig. 24.46](#)).



FIGURE 24.46 ■ IVC View. The transducer is placed subxiphoid in a midline plane and tilted toward the patient's right to identify the IVC as it enters the right atrium. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the liver, IVC, right atrium, and right hepatic vein ([Fig. 24.47](#)).



FIGURE 24.47 ■ IVC View. The liver is seen with the IVC entering the right atrium on the left. Probe marker (on right of the screen) is directed inferiorly here. (Ultrasound contributor: Jeremy S. Boyd, MD.)

- Measure the IVC 2 to 3 cm caudal to the entry point to the right atrium. Measure the IVC through the respiratory cycle at end inspiration and expiration ([Fig. 24.48](#)).



FIGURE 24.48 ■ IVC View. Measure the IVC 2 to 3 cm distal to the entry point to the RA; 1.5 to 2.5 cm is considered a normal IVC diameter. Greater than 2.5 cm is considered an enlarged or “plethoric” IVC. (Ultrasound contributor: Jeremy S. Boyd, MD.)

Abnormal Findings

- IVC collapse: The IVC should partially collapse during the normal respiratory cycle in nonventilated patients. A totally collapsed IVC is indicative of low central venous pressure (CVP). More than 50% collapse is less specific but also indicative of low CVP ([Fig. 24.49](#)).



FIGURE 24.49 ■ IVC View. A totally collapsed IVC is indicative of low CVP. (Ultrasound contributor: Jeremy S. Boyd, MD.)

- **IVC size:** The size of the IVC may be measured throughout the respiratory cycle. In general, an IVC that measures 2 cm or greater with poor respiratory variation is indicative of elevated CVPs due to volume overload, RV failure, and/or tamponade physiology ([Fig. 24.50A,B](#)).

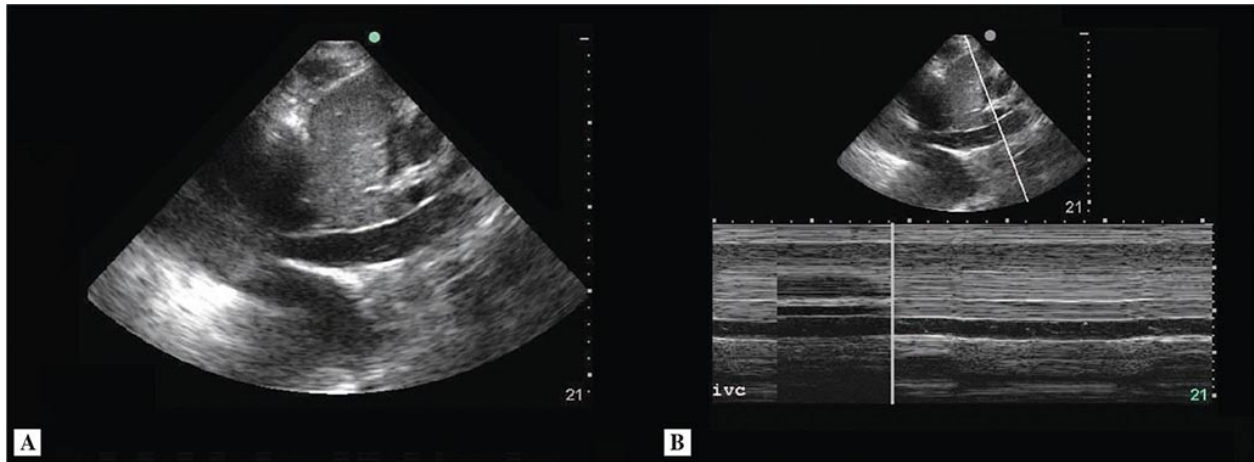


FIGURE 24.50A, B ■ IVC View. An IVC with poor respiratory variation (demonstrated in M-mode) is indicative of elevated central venous pressures due to volume overload, right ventricular failure, and/or tamponade physiology. (Ultrasound contributor: Jim Fiechtl, MD.)

- **IVC distensibility:** In patients receiving positive-pressure ventilation, the IVC will distend rather than collapse during inspiration. Measurements of IVC distensibility in ventilated patients can be used to calculate volume responsiveness to intravenous fluid challenge.

Pearls

1. Consider using M-mode through the IVC to aid in measurement for patients with high respiratory rates.
2. Some patients require a low anterior intercostal view to image the IVC as it enters the right atrium.

LUNG ULTRASOUND

Clinical Summary

Aerated lungs often interfere with sonographic visualization of the organs deep to them, such as often occurs in cardiac and biliary exams. This is due to the scattering effect of gas molecules on the ultrasound beam. For this reason, ultrasound of the lungs themselves was long thought to be of no diagnostic use. However, in recent years, researchers and clinicians have discovered that lung ultrasound can be used to assess for pathologic conditions that occur at the interface of the lung and the chest wall such as pneumothorax, pulmonary edema, and pneumonia. The evaluation of the pleura and the finding of lung sliding to evaluate for pneumothorax was described earlier in the section on trauma ultrasound. Contemporary point-of-care lung ultrasound often evaluates for the presence of alveolar interstitial syndrome, which occurs with the collection of fluid within the interlobular and intralobular lymphatics of the lung. This occurs in various pathologic states including cardiogenic pulmonary edema, acute respiratory distress syndrome, pneumonia, and diffuse parenchymal lung disease. Lung ultrasound can also effectively be used to visualize lung consolidation and pleural effusions.

Indications

- Shortness of breath and acute dyspnea
- Hypoxia
- Pleuritic chest pain
- Congestive heart failure
- Chest trauma

Required Views

- For comprehensive evaluation, current consensus recommendations include scanning the thorax in eight separate zones including the costophrenic angles of each chest ([Fig. 24.51](#)). Each hemithorax is divided into four different zones as shown in the figure.

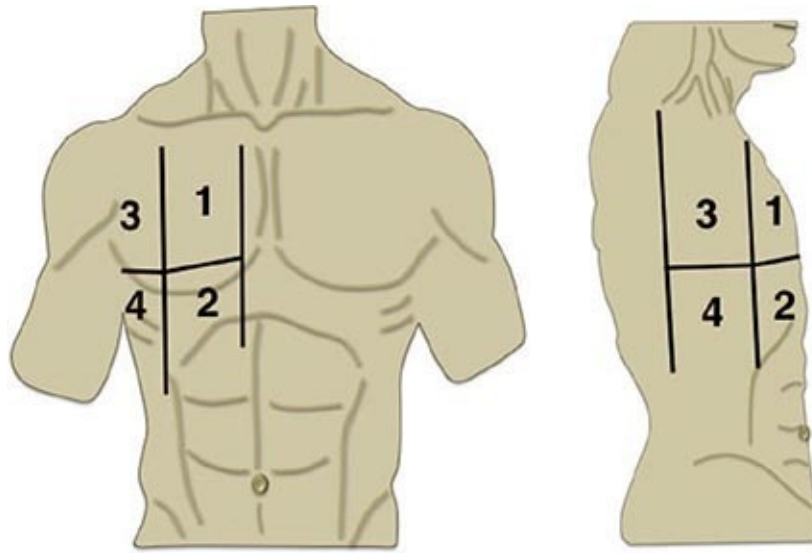


FIGURE 24.51 ■ Lung Zones. Current consensus recommendations include scanning the thorax in eight separate zones including the costophrenic angles of each chest. (Illustration contributor: Robinson M. Ferre, MD.)

- A more focused lung exam can be performed depending on the pathology suspected. For example, if assessing for a pneumothorax following a subclavian central venous line, anterior views along the midclavicular line would be sufficient.

Recommended Transducers

- Convex array (anterior and costophrenic angles)
- Phased array (costophrenic angles; poor for assessing lung sliding)
- Linear array (anterior chest only)

Patient Position

- The patient is traditionally imaged in a supine position, which is necessary if assessing for a pneumothorax.
- If assessing for conditions other than pneumothorax, a supine, semi-recumbent, or upright position may be used.

Technique

- To assess for alveolar interstitial syndrome or consolidation: With the indicator pointed toward the patient's head, each of the four zones of each

hemithorax should be imaged. The depth on the screen should be between 13 and 15 cm deep. Each zone should be imaged thoroughly for evidence of A-lines, B-lines, or consolidation.

- To assess for pleural fluid: The technique for evaluating costophrenic angles for pleural effusion is identical to that employed in the E-FAST when evaluating for hemothorax.
- To assess for a pneumothorax: The technique for evaluating costophrenic angles for pleural effusion is identical to that employed in the E-FAST when evaluating for hemothorax.

Findings

- A-lines: Normal aerated lung is indicated by the presence of “A-lines” and lung sliding. A-lines are echogenic horizontal lines deep and parallel to the pleural line that occur at regular intervals and do not move with respiration or lung sliding ([Fig. 24.52](#)).

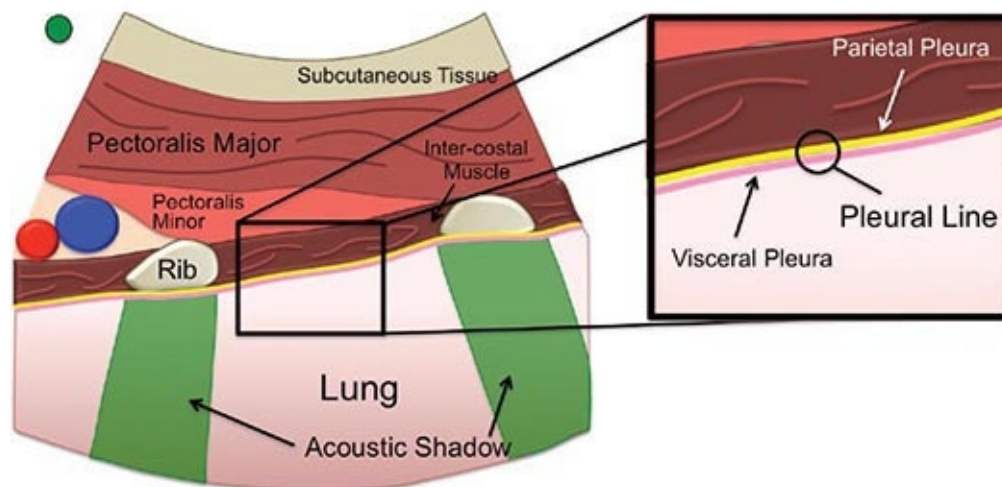
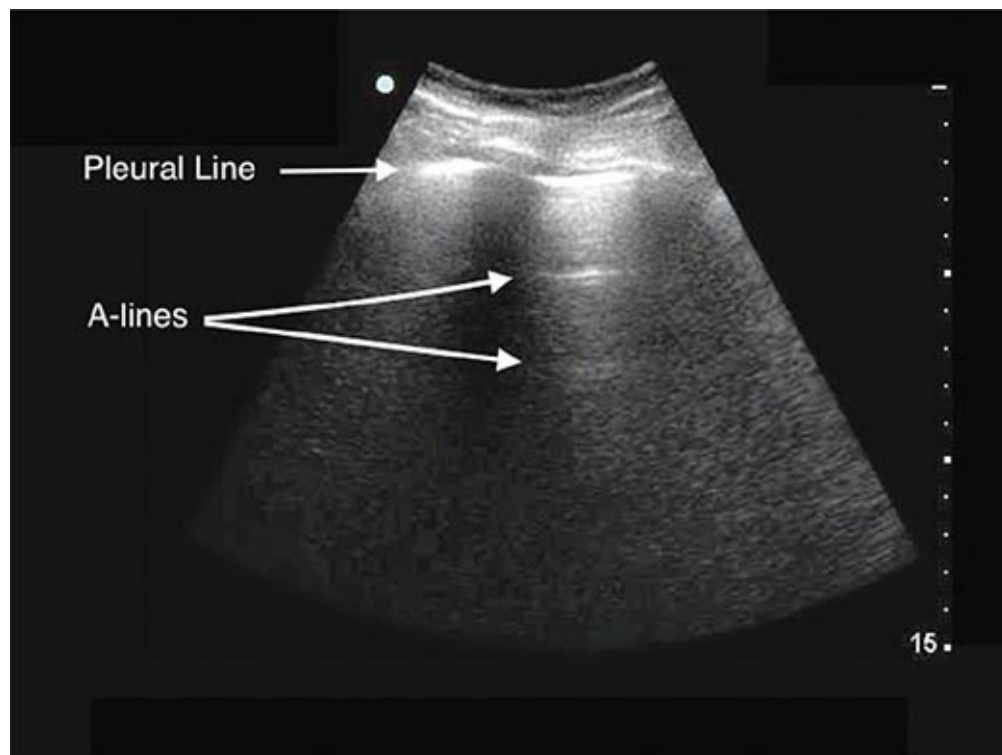


FIGURE 24.52 ■ Lung Ultrasound: A-Lines. Normal aerated lung is indicated by the presence of “A-lines” and lung sliding. A-lines are echogenic horizontal lines deep and parallel to the pleural line that occur at regular intervals and do not move with respiration. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

- **B-Lines:** The presence of “B-lines” indicates alveolar interstitial syndrome. B-lines are defined as laser-like artifacts that begin from the pleural line and extend to the bottom of the screen without fading and move in conjunction with respiration or lung sliding (Fig. 24.53).

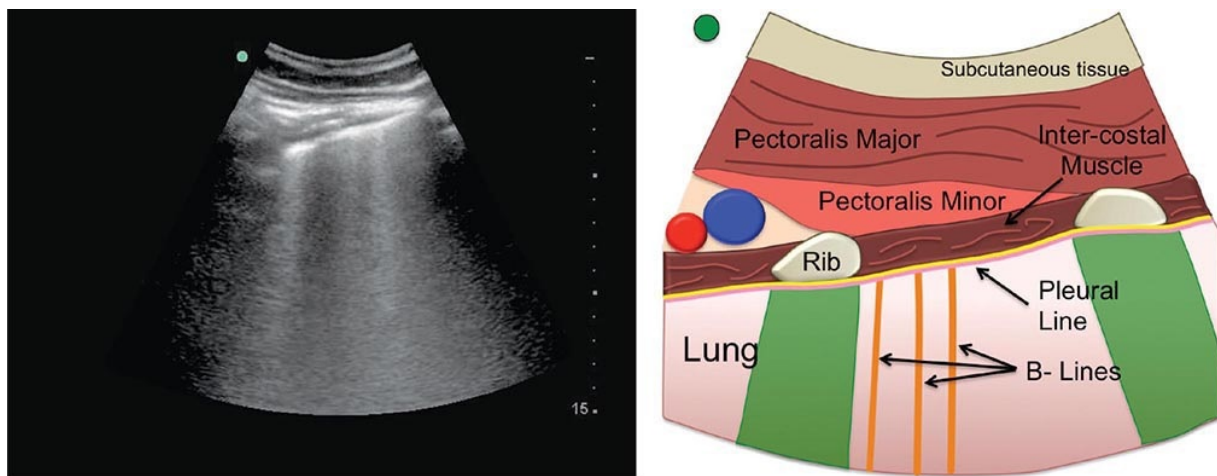


FIGURE 24.53 ■ Lung Ultrasound: B-Lines. The presence of “B-lines” indicates alveolar interstitial syndrome. B-lines are defined as laser-like artifacts that begin from the pleural line and extend to the bottom of the screen without fading and move in conjunction with respiration. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jordan Rupp, MD.)

- **Consolidation:** Consolidation of lung tissue appears as a subpleural area of tissue-like echotexture. It may have comet-tail artifacts extending from the far-field border, a pattern called the “shred sign.” Air and/or fluid bronchograms may also be noted within the consolidation ([Fig. 24.54](#)).

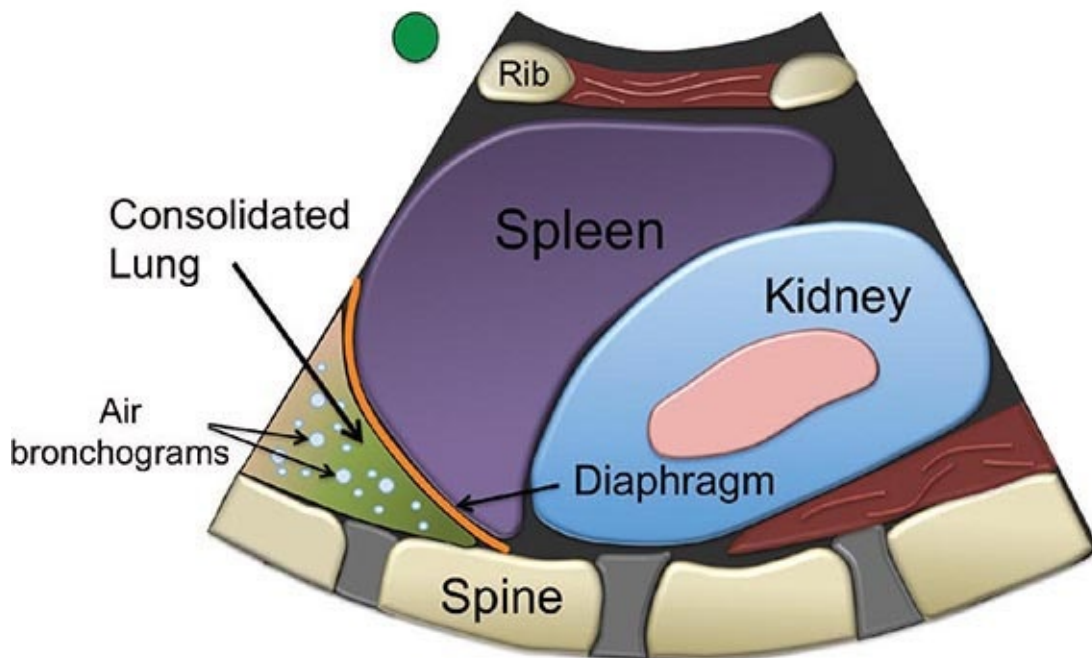
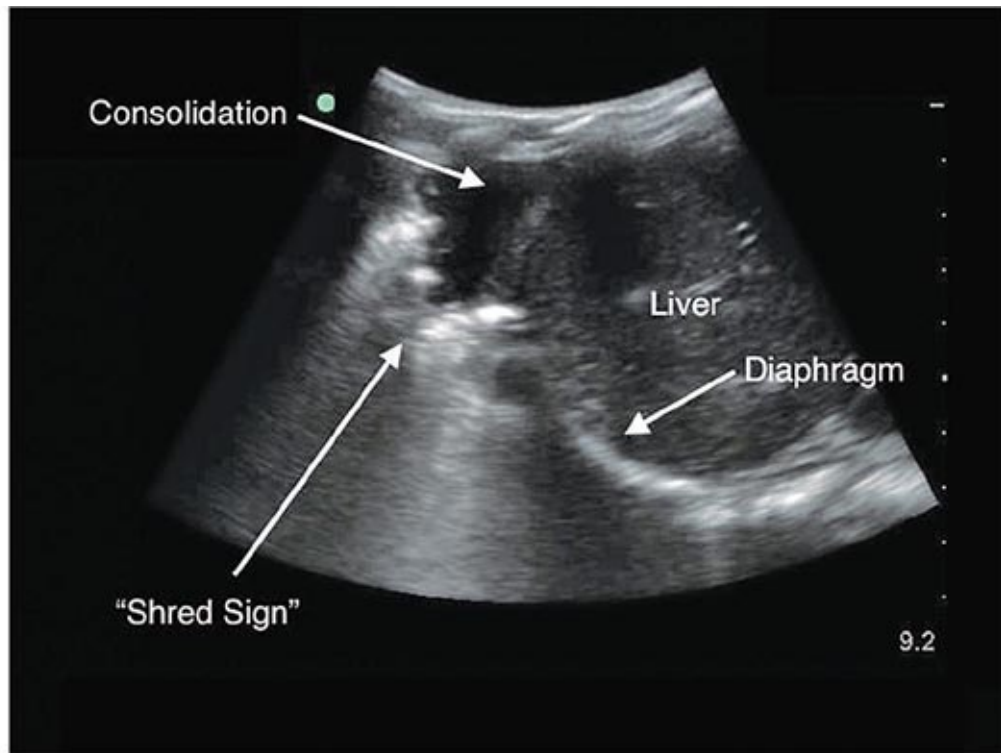


FIGURE 24.54 ■ Lung Ultrasound. Consolidation of lung tissue appears as a subpleural area of tissue-like echotexture. It may have comet-tail artifacts extending from the far field border, a pattern called the “shred sign,” seen in this image of a pneumonia. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

- Pleural effusion: A hypoechoic area located in the diaphragmatic recess superior/cephalad to the diaphragm that permits visualization of the tissues

deep to it (“spine sign”) is a pleural effusion. Consolidated lung can often be seen moving within the effusion in synchrony with the patient’s respirations (Fig. 24.55).

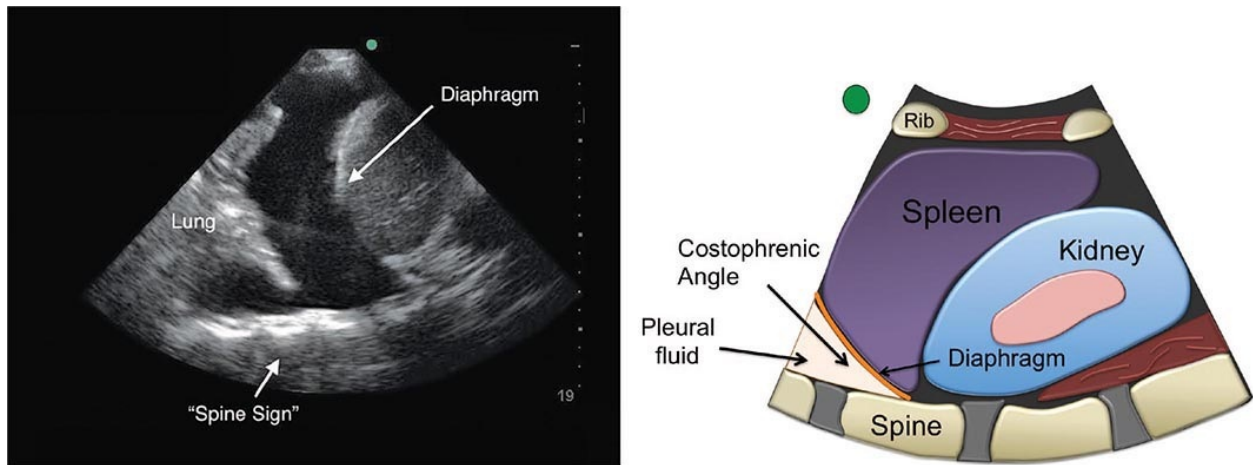


FIGURE 24.55 ■ Lung Ultrasound. A pleural effusion is seen as a hypoechoic area located in the diaphragmatic recess superior/cephalad to the diaphragm with the spine sign seen extending beyond the diaphragm into the thorax. Consolidated lung is seen within the effusion. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Pearls

- The distribution of B-lines, whether diffuse, focal, unilateral, or bilateral, in conjunction with the clinical exam and history can help to determine the etiology of the patient’s symptoms. Cardiogenic pulmonary edema will be found diffusely and bilaterally, whereas a focal pneumonia or pulmonary contusion may only present within a focal region of the ultrasound lung exam.
- In patients with cardiogenic pulmonary edema, the number and frequency of B-lines correlate with the severity of edema and can also be used to monitor the response to therapy.
- When performing an E-FAST exam, the linear probe is frequently used as it is most sensitive for lung sliding and visualization of the pleura. When using a different probe to evaluate the lungs, adjust the depth to a shallow setting to ensure adequate evaluation of the pleura for lung sliding. The remainder of the exam should be performed with a depth of at least 13 cm when assessing for B-lines and consolidation.

ABDOMINAL AORTA ULTRASOUND

Clinical Summary

EUS of the abdominal aorta is used to diagnose or exclude an abdominal aortic aneurysm (AAA). Although the sensitivity of abdominal ultrasound for the detection of aortic dissection is limited, the presence of an intra-aortic flap is diagnostic for aortic dissection.

Indications

- Abdominal, back, or flank pain
- Pulsatile abdominal mass
- Undifferentiated hypotension

When a patient is unstable, there is no bedside test superior to an EUS of the abdominal aorta to diagnose an AAA. Early diagnosis can improve patient survival. An abdominal aortic measurement greater than 3 cm in diameter is abnormal and diagnostic of an AAA. While an AAA may rupture at any size, the risk of rupture is much greater starting with measurements greater than 5 cm. AAAs occur as both fusiform (more common) and saccular types. It is essential to image the aorta in both sagittal and transverse planes.

Views for Emergency Department Abdominal Aortic Ultrasound

1. Transverse view
2. Sagittal view

Equipment: Recommended Transducer for Abdominal Aortic Ultrasound

- Convex array

Patient Position

The patient is supine.

Techniques

Transverse View

- Place the transducer in the epigastrium with the transducer indicator oriented to the patient's right (Fig. 24.56).



FIGURE 24.56 ■ Abdominal Aorta Transverse View. Place the transducer in the epigastrium with the transducer indicator oriented to the patient's right. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the acoustic shadow caused by the vertebral body. The abdominal aorta is the circular, pulsating, anechoic structure anterior and slightly to the patient's left.
- Identify and measure the aorta at the proximal (at or above the origin of the superior mesenteric artery [SMA]), middle (near the renal arteries), and distal (immediately proximal to the bifurcation) in the anteroposterior plane (Figs. 24.57 and 24.58).



FIGURE 24.57 ■ Abdominal Aorta: Transverse View. Short-axis view of the proximal aorta showing the celiac artery and its branches. (Ultrasound contributor: Jeremy Boyd, MD.)

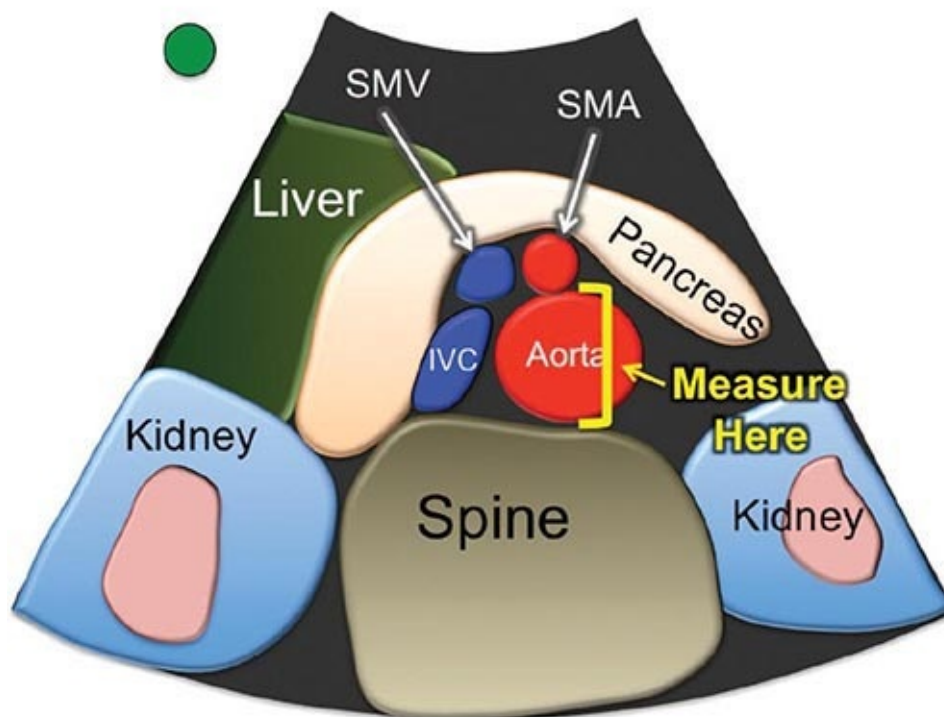
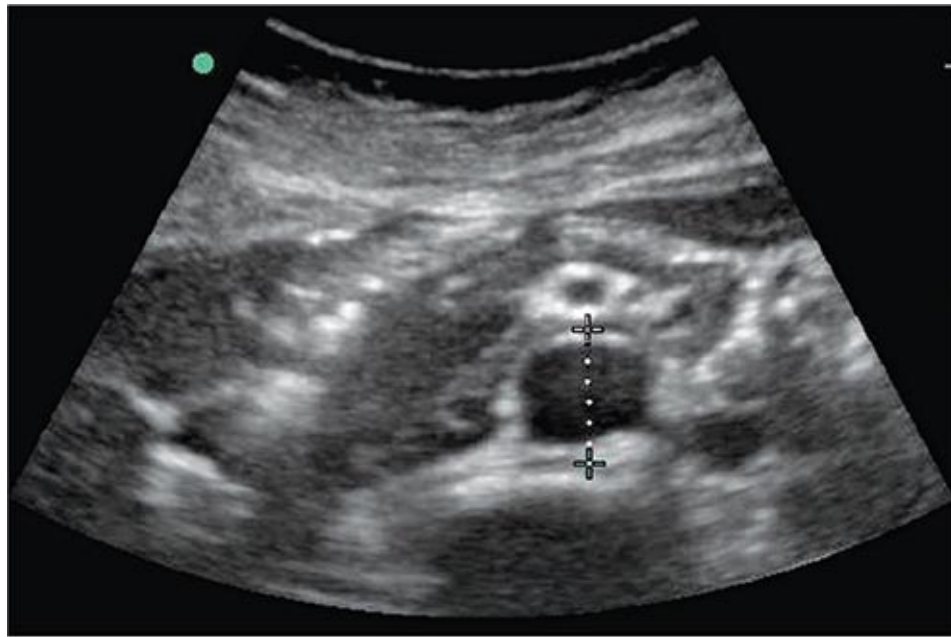


FIGURE 24.58 ■ Abdominal Aorta: Transverse View. The proximal aorta, superior mesenteric artery (SMA), and the surrounding anatomy are often compared to a mantle clock. (Ultrasound contributor: Eric Wu, MD; illustration contributor: Robinson M. Ferre, MD.)

- Identify the aorta in relation to the IVC, SMA, and splenic vein.
- Move down the abdominal aorta to the bifurcation (approximately the level of the umbilicus) (Fig. 24.59).



FIGURE 24.59 ■ Abdominal Aorta: Transverse View. Still image of the left and right iliac arteries just distal to the bifurcation of the aorta, sitting on top of the lumbar vertebra. A collapsed IVC is seen to the left. (Ultrasound contributor: Kaitlyn Works, MD.)

Sagittal View

- Place the transducer in the epigastrium with the transducer indicator oriented toward the patient's head. Move down the abdominal aorta to the bifurcation ([Fig. 24.60](#)).



FIGURE 24.60 ■ Abdominal Aorta Sagittal View. Place the transducer in the epigastrium with the transducer indicator oriented toward the patient's head. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the liver, aorta, IVC, celiac trunk, and SMA ([Fig. 24.61](#)).

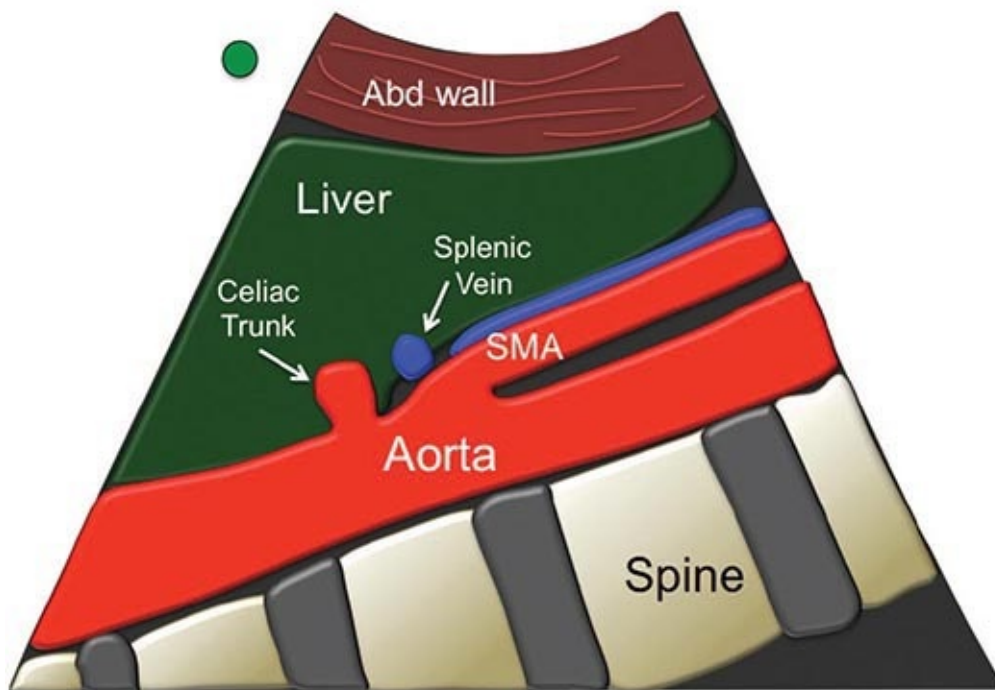
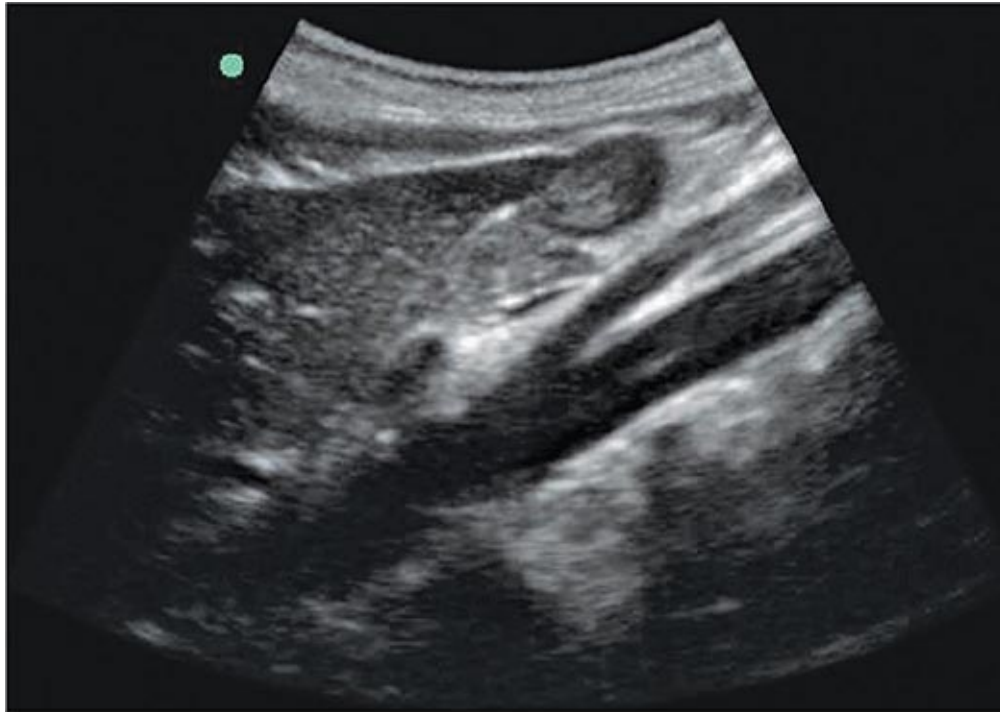


FIGURE 24.61 ■ Abdominal Aorta. Sagittal view, proximal aorta. (Ultrasound contributor: Jeremy Boyd, MD, RDMS; illustration contributor: Robinson M. Ferre, MD.)

Abnormal Findings

- AAA: The abdominal aorta is aneurysmal if at any point it measures more

than 3 cm. The aorta can also be aneurysmal if a distal segment measures more than 50% of a proximal segment ([Fig. 24.62](#)).



FIGURE 24.62 ■ Abdominal Aorta: Transverse View, AAA. Large abdominal aortic aneurysm. (Ultrasound contributor: Jeremy Boyd, MD.)

- Aortic dissection: The echogenic line of an aortic “flap” can be seen inside the lumen of the abdominal aorta when present (often moving with pulsations) ([Fig. 24.63](#)).

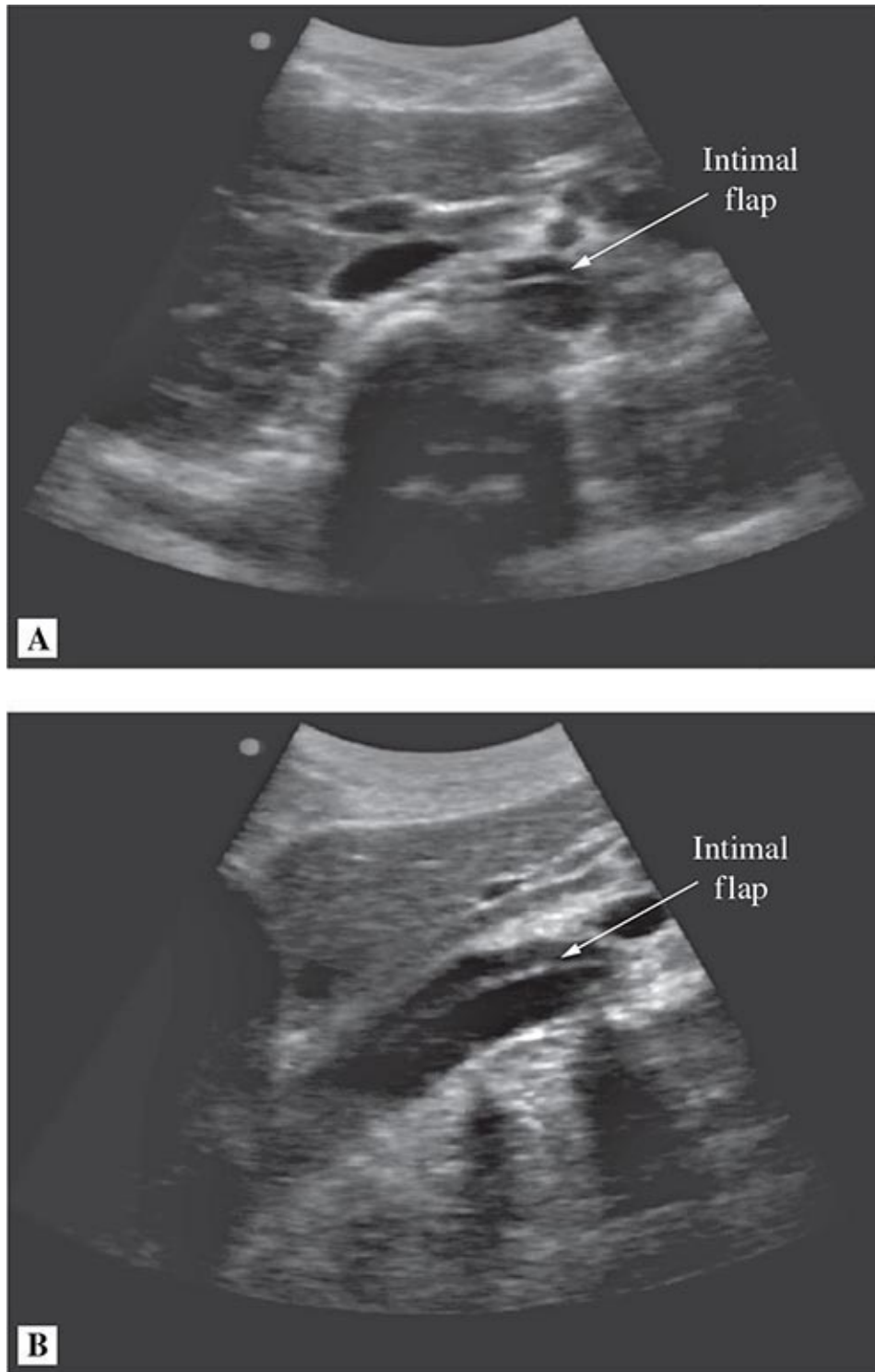


FIGURE 24.63 ■ Abdominal Aorta: Dissection. An aortic dissection flap is seen. (Ultrasound contributor: Paul R. Sierzenski, MD, RDMS.)

Pearls

1. Starting the exam in the transverse view will help orient the scanner to the anatomy.
2. Measure the entire diameter (outer wall to outer wall) of the aorta. Measuring the lumen is grossly inaccurate and will underestimate the size of an AAA when a clot is present (Figs. 24.64 and 24.65). Include measurements of the proximal, mid, and distal aorta.

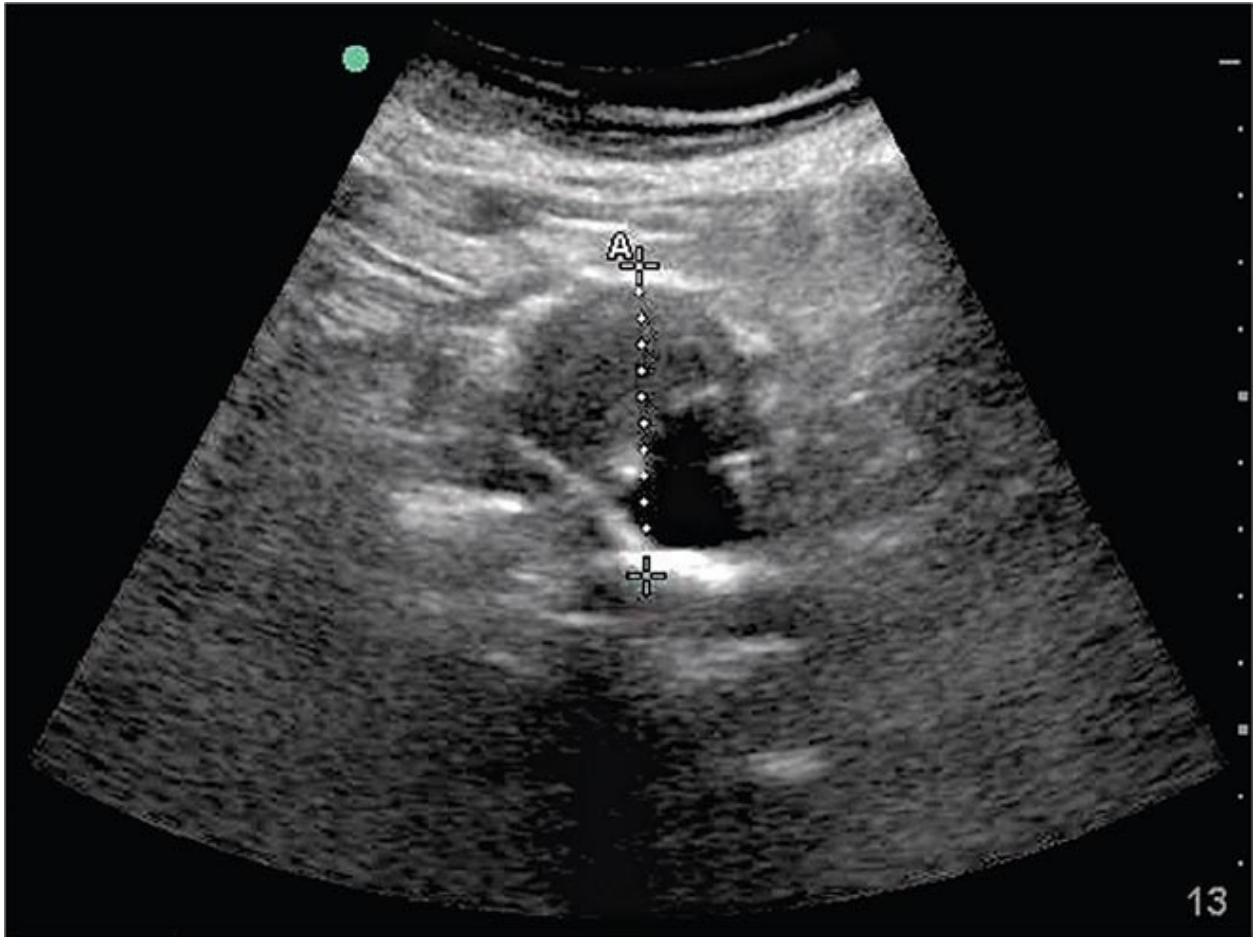


FIGURE 24.64 ■ Abdominal Aorta: Transverse View, AAA. Mid-aorta showing an abdominal aortic aneurysm with clot partially occluding the vessel. (Ultrasound contributor: Louis Frazier, MD.)

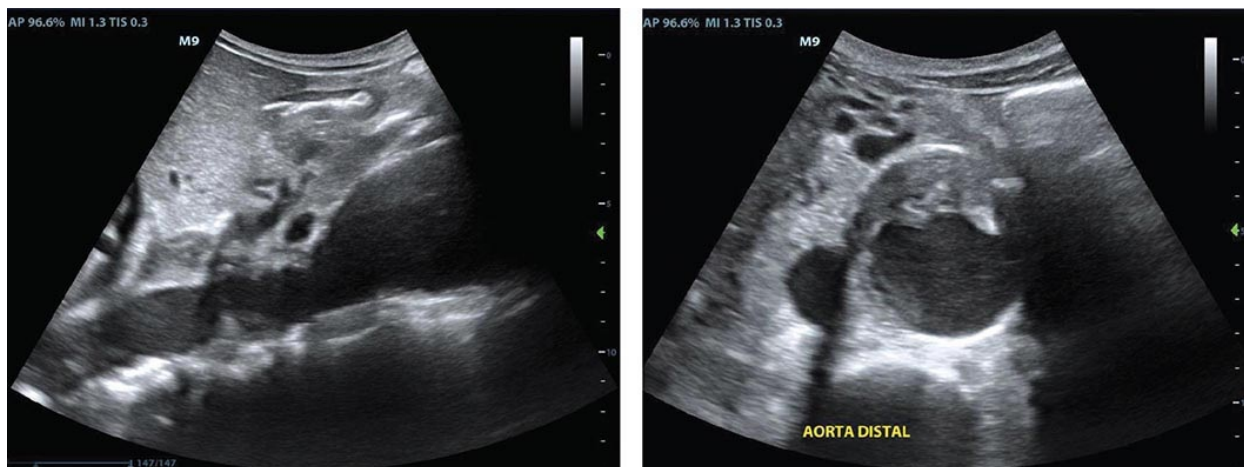


FIGURE 24.65 ■ Abdominal Aorta: Sagittal and Transverse View, AAA. Distal AAA with clot filling a portion of the vessel. (Ultrasound contributor: Sam Parnell, MD.)

3. If a significant amount of bowel gas is present, sit the patient at 45 degrees and apply constant gentle pressure.
4. The IVC may be confused for the aorta, especially in sagittal views. The “sniff test”—having the patient abruptly sniff— will cause the IVC to collapse as a result of the negative pressure transmitted to the venous system during this maneuver.
5. If spectral or color flow Doppler is available, it may be used to discriminate between the highly pulsatile flow of the aorta and the low amplitude of the IVC.
6. In patients with an aortic dissection, an intimal flap can be visible as a linear structure within the aorta. While this finding is highly specific, it is insensitive and its absence cannot be used to rule out a dissection ([Fig. 24.63](#)).

DEEP VENOUS THROMBOSIS ULTRASOUND

Clinical Summary

The presence of a deep venous thrombosis (DVT) is part of the differential diagnosis of a variety of signs and symptoms. Although clinical scoring algorithms have been developed to gauge risk, no clinical findings are conclusive of this condition, and imaging is necessary to confirm the diagnosis. Ultra-sound is a sensitive, noninvasive imaging modality that can be performed rapidly and essentially without contraindication. Radiology-performed studies

are not always readily available emergently; thus, an emergency physician properly trained in bedside ultrasonography can help guide and hasten care by performing a limited serial compression examination of the proximal leg veins. This exam has been shown to be highly sensitive for diagnosis of DVT in symptomatic ED patients when performed by properly trained individuals.

Indications

- Extremity swelling, pain, or erythema
- Dyspnea
- Chest pain

Required Views for Lower Extremity DVT Ultrasound

- Common femoral vein (CFV) ([Fig. 24.66](#))

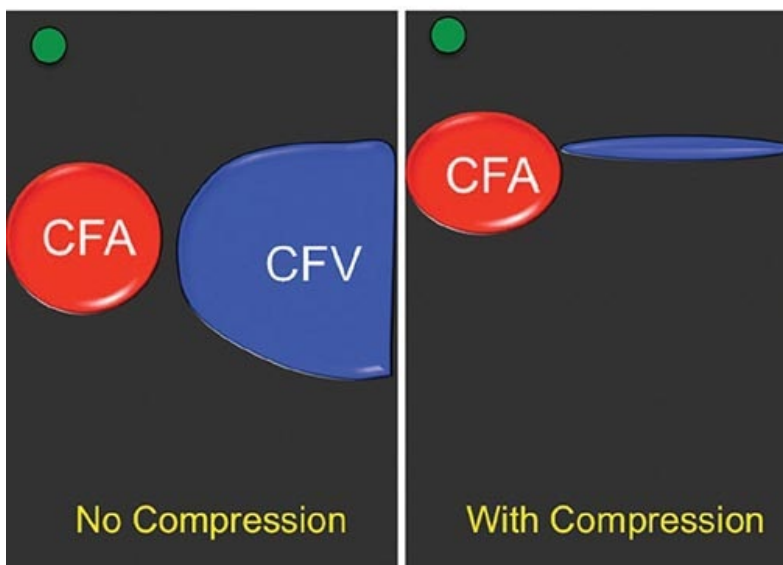
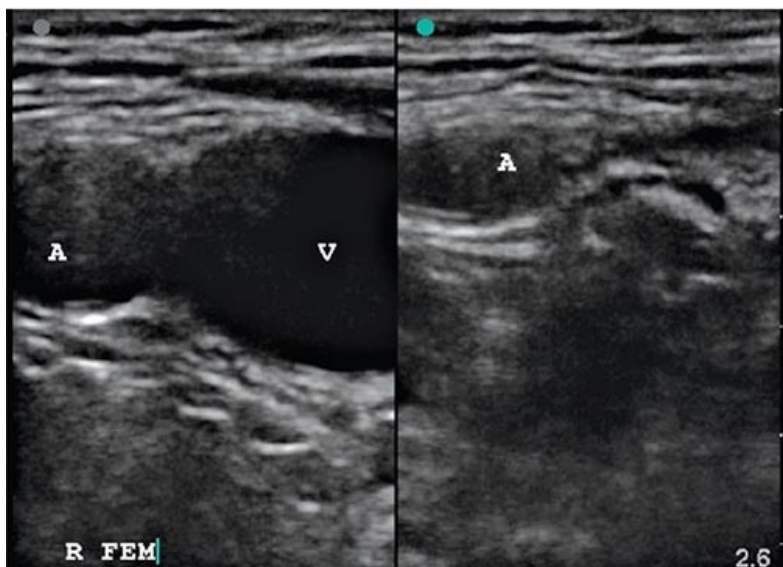


FIGURE 24.66 ■ DVT Ultrasound, CFV View. The transducer should be oriented in a transverse plane just distal to the inguinal ligament. The vein is gently but firmly compressed until the walls completely collapse. (Illustration contributor: Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Robinson M. Ferre, MD.)

- Superficial femoral vein (SFV) ([Fig. 24.67](#))

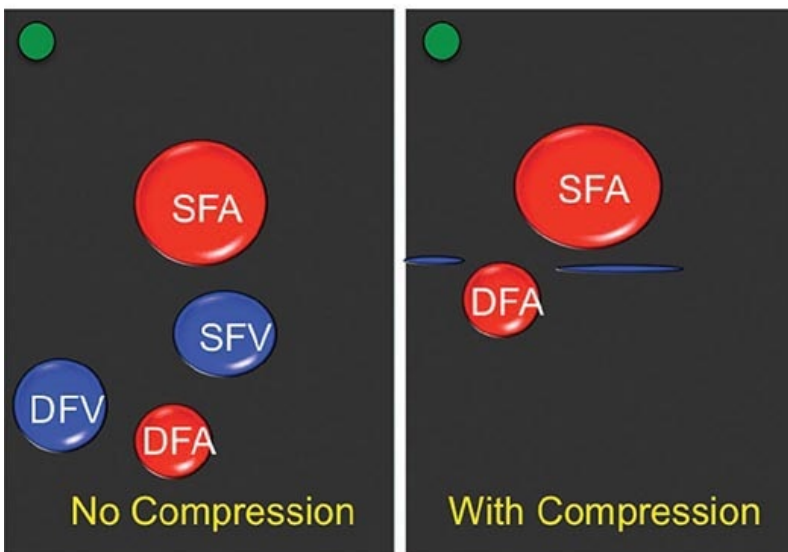
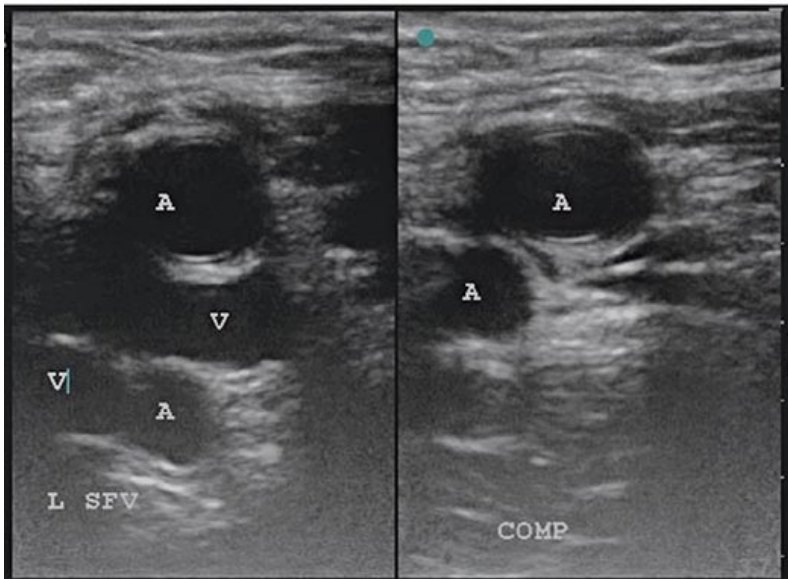


FIGURE 24.67 ■ DVT Ultrasound, SFV View. The transducer should be oriented in a transverse plane. The vein(s) is/are gently but firmly compressed until the walls completely collapse. (Illustration contributor: Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Robinson, M. Ferre, MD.)

- Popliteal vein (PV) ([Fig. 24.68](#))

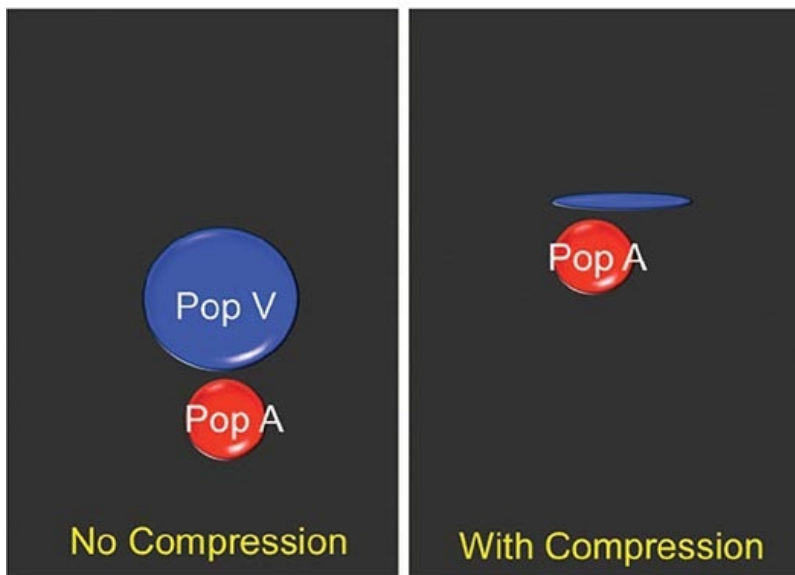
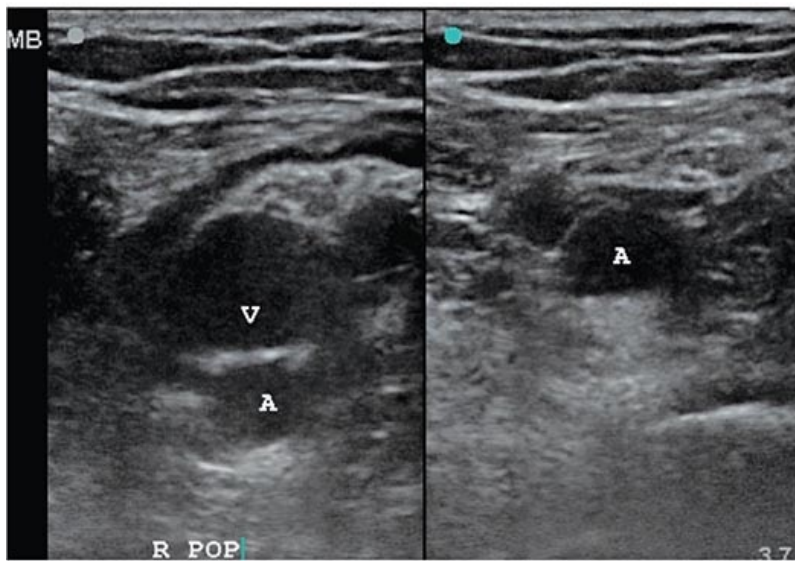


FIGURE 24.68 ■ DVT Ultrasound, Popliteal View. The transducer should be oriented in a transverse plane behind the popliteal fossa. The vein is gently but firmly compressed until the walls completely collapse. The popliteal vein is usually found superficial to the popliteal artery, ie, “pop on top.” (Illustration contributor: Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Suzanne Bryce, MD.)

Equipment: Recommended Transducers for DVT Ultrasound

- Linear
- Convex

Patient Preparation

- Ideally, the patient is recumbent with the head of the bed elevated 20 to 40 degrees for lower extremity imaging.
- External rotation of the patient’s hip with partial knee flexion will aid imaging, especially the popliteal vessels. If tolerated, having the patient lie prone may also aid in imaging the popliteal vasculature.

Technique

Lower Extremity

- The transducer should be oriented in a transverse plane through the course of the vein.
- Imaging begins at the proximal CFV moving distally to the PV. Carefully identify the CFV, SFV, and PV ([Fig. 24.69](#)).

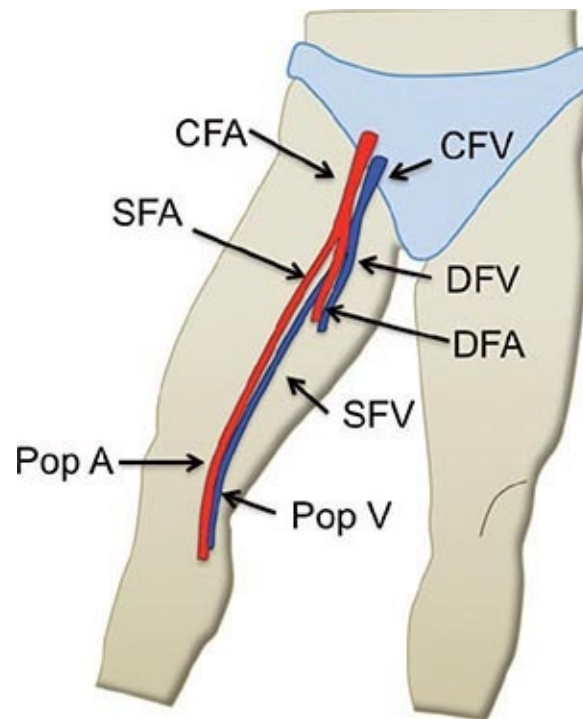


FIGURE 24.69 ■ Deep Veins of the Lower Extremity. Diagram illustrating the anatomy of the deep venous system and accompanying arterial system of the lower extremity. Note that the superficial femoral vein (SFV) is actually considered a deep vein of the extremity, despite its name. (Illustration contributor: Robinson M. Ferre, MD.)

- The vein is gently but firmly compressed until the walls completely collapse. This process is repeated every 1 to 2 cm throughout each site.
- Color or spectral Doppler can be used at the CFV and PV to document phasicity with respiration or augmentation.

Abnormal Findings

- Inability to compress the vein suggests DVT. This may be acute, chronic, or due to technical limitations (eg, unable to apply enough pressure) ([Fig. 24.70](#)).

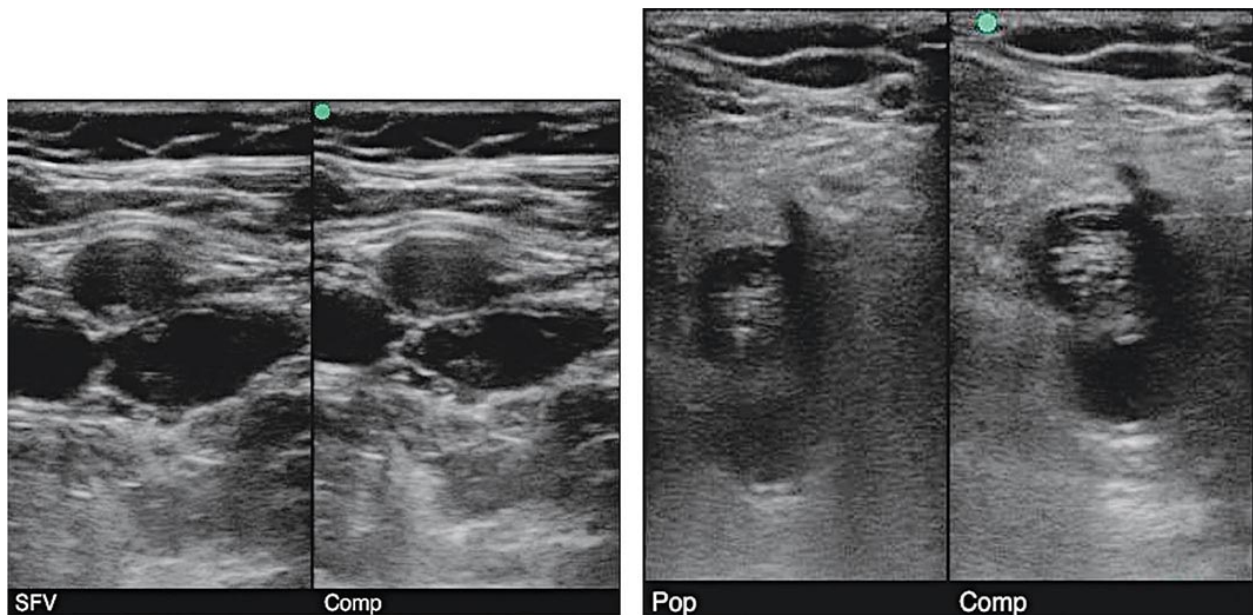


FIGURE 24.70 ■ DVT Ultrasound. A deep venous thrombosis is seen as a hyperechoic clot within the SFV (left) and popliteal vein (right), preventing compression. (Ultrasound contributor: Robinson M. Ferre, MD.)

- Poor or absent phasicity suggests occlusion at a location proximal to transducer.
- Poor or absent augmentation suggests occlusion at a location distal to transducer.

Pearls

1. The CFV is often located more proximally than new sonographers expect. Start the exam adjacent to the inguinal ligament.
2. Thoroughly evaluate the CFV at the confluence of the saphenous vein.
3. Ensure that the vascular structure is a deep vein. Deep veins are paired with arteries. Veins are easily compressible and arteries are not. Color or spectral Doppler should demonstrate characteristic arterial or venous waveforms.
4. If unable to adequately compress vessels, apply pressure from under the extremity toward the transducer. If the artery begins to distort its shape, adequate pressure has been applied.
5. Echogenic material within the vein may be an artifact and not a DVT. Visualize any abnormality in more than one plane and optimize settings to exclude artifacts.

ULTRASOUND-GUIDED RESUSCITATION

Clinical Summary

As ultrasound is integrated into the bedside evaluation of many emergent symptoms and disease states, multiple protocols have emerged for the rapid assessment of critically ill patients in shock and cardiac arrest states. By combining many of the exams described previously in this chapter, one can quickly assess for the cause of a patient's hypotension or cardiac arrest, helping to sort through what is often an extensive differential diagnosis. A common approach is termed the "RUSH" protocol—Rapid Ultrasound for Shock and Hypo-tension. Although there are distinct protocols proposed by different authors, they combine a sonographic assessment of a patient's gross cardiac function, intravascular volume status, and identification of pathologic conditions (such as AAA or pneumothorax) that threaten a patient's hemodynamic stability. One group of authors promotes the use of the heuristic "Pump, Tank, and Pipes" as the mental framework for this evaluation, while another uses the mnemonic "HI-MAP" (heart, IVC, Morison [and other FAST views], aorta, and pulmonary/pneumothorax) to outline the protocol. Regardless of which specific protocol one uses for evaluation of the hypotensive patient, when combined with a history and physical exam, bedside ultrasound can be used to guide resuscitative efforts and tailor them to the type of shock encountered.

In addition, when confronted with a patient in cardiac arrest, bedside ultrasound can be used during cardiopulmonary resuscitation (CPR) to help guide therapies and decision making. The protocols described previously can be used to search for the cause of a patient's arrest. Additionally, the subxiphoid and apical four-chamber windows can even be used during compressions or rapidly during pulse checks to determine the native function of the heart and distinguish between true PEA or "electromechanical dissociation" (where there is an electrical rhythm conducted but no cardiac activity) and "pseudo-PEA" (where the heart's contractions are so poor that they do not produce palpable pulsations). One can sometimes even distinguish between asystole and fine ventricular fibrillation on the monitor by looking closely at the mitral valve for motion during a pulse check. Alternatively, some emergency physicians are utilizing transesophageal ultrasound probes for use in cardiac arrest—obviating the need to interrupt compressions and allowing for assessment of cardiac compressions during CPR.

Indications

- Unexplained hypotension or shock
- Cardiac arrest

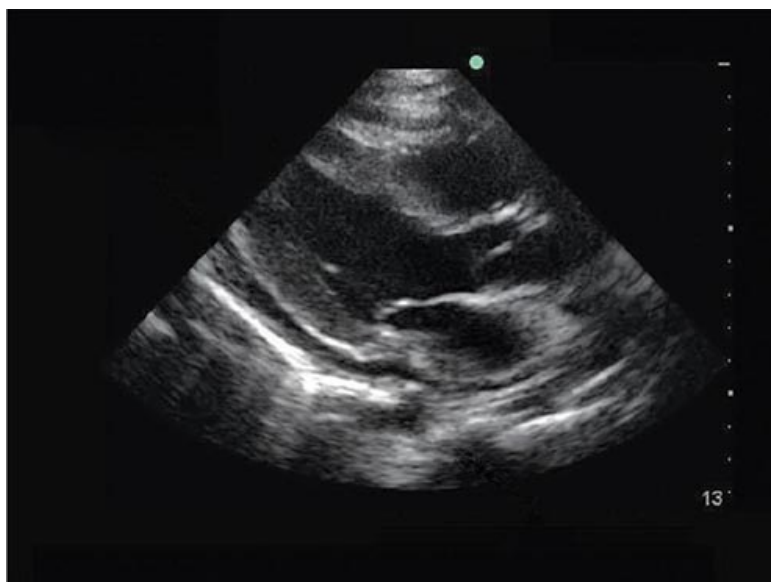


FIGURE 24.71 ■ Ultrasound-Guided Resuscitation. Representative images of anatomic areas assessed during ultrasound-guided resuscitation. The heart (“pump”), IVC + FAST (“tank”), and great vessels (“pipes”) are assessed confluent to evaluate for the etiology of hypotension and shock. (Ultrasound contributors: Jeremy S. Boyd, MD, and Lara Philips, MD.)

Once the patient has been evaluated, findings on ultra-sound may be correlated with one of the types of shock identified below. The table below lists representative findings within each shock state. Note that each finding listed below is neither sufficient nor required for the diagnosis of different types of shock, and is merely suggestive. Of course, the clinical context of each patient’s presentation must be taken into account in any resuscitation.

Pearls

1. The subxiphoid and apical four-chamber views can be obtained during CPR without interrupting chest compressions.
2. Parasternal cardiac views might be assessed during a pulse check, but should be evaluated as rapidly as possible so as to minimize pauses in chest compressions.
3. When obtaining parasternal views during an arrest, have a towel ready to wipe off any gel afterward to avoid interfering with chest compressions.
4. Tailor the resuscitation exam to the patient’s presenting symptoms. If there is trauma, start with an E-FAST exam. If the patient presents with acute dyspnea, consider starting with cardiac or lung ultrasound first.
5. In asystole, there is no spontaneous cardiac activity seen on cardiac ultrasound. Blood can often be seen swirling within the motionless ventricles when assessing after compressions.

TABLE 24.2 ■ APPROACH TO ULTRASOUND-GUIDED RESUSCITATION

	Views	Questions
“Pump”	Focused cardiac assessment: <ul style="list-style-type: none"> • Parasternal long • Parasternal short • Apical four-chamber • Subxiphoid 	<ul style="list-style-type: none"> • Is there a pericardial effusion? • What is the gross left ventricular function? • Are there signs of right heart strain?
“Tank”	Volume status and integrity: <ul style="list-style-type: none"> • IVC • E-FAST exam 	<ul style="list-style-type: none"> • Is the IVC collapsed or plethoric? • Is there hemoperitoneum? • Is there a hemothorax or pneumothorax? • Is there pulmonary edema?
“Pipes”	Vascular pathology: <ul style="list-style-type: none"> • Aorta • DVT 	<ul style="list-style-type: none"> • Is there an AAA? • Is there an aortic dissection? • Is there a DVT?

AAA, aortic abdominal aneurysm; DVT, deep vein thrombosis; E-FAST, extended focused assessment with sonography in trauma; IVC, inferior vena cava;

TABLE 24.3 ■ SONOGRAPHIC FINDINGS IN VARIOUS SHOCK STATES

	Hypovolemic/ Hemorrhagic Shock	Cardiogenic Shock	Obstructive Shock	Distributive Shock
Pump	Hyperdynamic heart	Hypocontractile heart Dilated cardiomyopathy	Pericardial effusion Right heart strain McConnell sign ^a Hyperdynamic heart	Hyperdynamic heart
Tank	Collapsed IVC Hemoperitoneum Hemothorax	Distended IVC Pulmonary edema Pleural effusions	Distended IVC Absent lung sliding/lung point (pneumothorax)	Normal/collapsed IVC Pleural fluids (empyema) Focal B-lines (pneumonia)
Pipes	AAA Aortic dissection	Normal	DVT	Normal

AAA, abdominal aortic aneurysm; DVT, deep vein thrombosis; IVC, inferior vena cava.

^aMcConnell sign is a finding seen on focused cardiac ultrasound in the apical four-chamber view. It is characterized by right ventricular (RV) free wall hypokinesis with preserved apical contractility. It is a sign of acute RV strain, whether due to massive pulmonary embolism or RV infarct.

Modified and adapted from Weingart et al. Rapid Ultrasound for Shock and Hypotension (RUSH-HIMAPP). 2009. <http://emedhome.com/>; Perera P, Mailhot T, Riley D, Mandavia D. “The RUSH exam: Rapid Ultrasound in SHock in the evaluation of the critically ill. *Emerg Med Clin North Am.* 2010;28(1):29-56; and Seif D, Perera P, Mailhot T, Riley D, Mandavia D. Bedside ultrasound in resuscitation and the rapid ultrasound in shock protocol. *Crit Care Res Pract.* 2012;2012:503254.

GALLBLADDER ULTRASOUND

Clinical Summary

Biliary ultrasound can be very rewarding. A rapid, focused exam can help determine if gallstones or gallbladder pathology is the etiology of a patient’s presenting symptoms. In turn, bedside ultrasound can expedite therapy or expedite the pursuit of an alternative diagnosis. While the sonographic identification of gallstones may seem straightforward, the sonographic findings for cholecystitis are often subtle.

Indications

- RUQ pain
- Jaundice
- Epigastric pain

Views for Emergency Department Biliary Ultrasound

- Sagittal view of the gallbladder
- Transverse view of the gallbladder

Equipment: Recommended Transducers for Gallbladder Ultrasound

- Convex array
- Phased array

Patient Positioning

- Supine or left lateral decubitus position

Techniques

Sagittal View

- Initially, the transducer is placed in the subxiphoid region with the indicator directed toward the patient's head in a sagittal view and swept below the right costal margin to approximately the midclavicular line. The transducer is then rotated (often clockwise) to obtain a view of the gallbladder in its longest axis ([Fig. 24.72](#)).



FIGURE 24.72 ■ Gallbladder: Sagittal View. Probe is placed just under the costal margin and swept across the costal margin to locate the gallbladder. The transducer is then rotated (often clockwise) to obtain a view of the gallbladder in its longest axis. (Photo contributor: Lawrence B. Stack, MD.)

- Scan through the gallbladder completely from medial to lateral borders including careful evaluation of the gallbladder neck. A thorough search for gallstones should be made, particularly at the neck where small symptomatic gallstones may be seen.
- Identify the liver, gallbladder, portal vein, hepatic artery, main lobar fissure, and the common hepatic duct ([Figs. 24.73](#) and [24.75](#)).



FIGURE 24.73 ■ Gallbladder: Sagittal View. Long-axis view of gall-bladder often reveals an

“exclamation mark” sign made of the gallbladder and the portal triad. (Ultrasound contributor: Robinson M. Ferre, MD.)



FIGURE 24.74 ■ Gallbladder: Transverse. Rotate the probe 90 degrees counterclockwise to bring the gallbladder into short axis. (Photo contributor: Lawrence B. Stack, MD.)

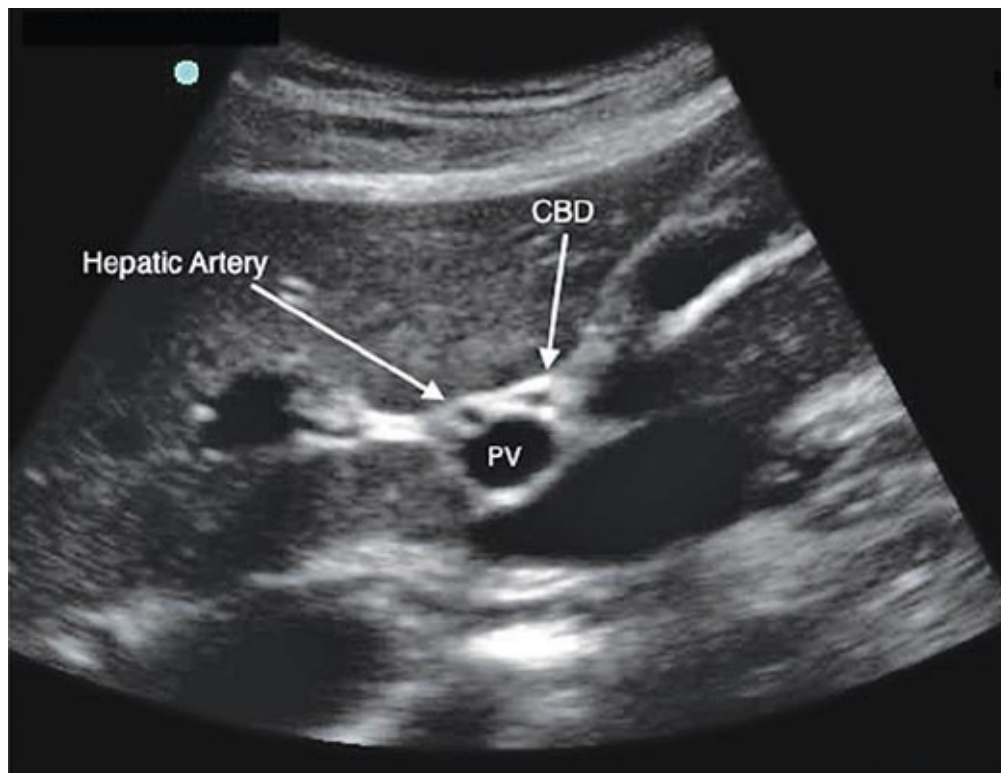


FIGURE 24.75 ■ Gallbladder: Portal Triad. The portal vein (PV), hepatic artery, and common bile duct (CBD) are readily seen in the trans-verse view. (Ultrasound contributor: Shannon Snyder, MD, RDMS.)

Transverse View

- After identifying the gallbladder in the sagittal position, rotate the transducer 90 degrees counterclockwise toward the patient's right (Fig. 24.75).
- Identify the liver, gallbladder, IVC, and common bile duct (CBD). Measure the anterior wall of the gallbladder for thickness (Fig. 24.74).

Additional Views

- If indicated, find the CBD. This can be done in two ways. First, identify the common hepatic duct at the level of the porta hepatis, and trace it down beyond the border of the liver.
- Measure the thickness of the CBD when visible from inside wall to inside wall (Fig. 24.77). Color flow Doppler will help delineate the CBD (no flow) from the hepatic artery (Fig. 24.78).

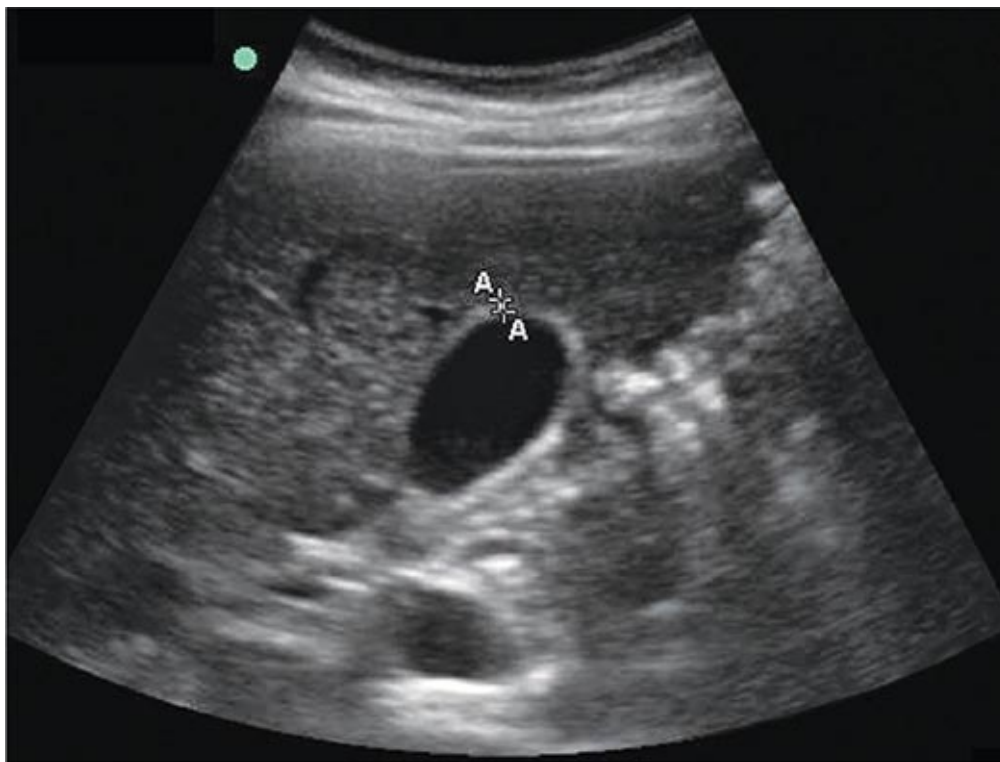


FIGURE 24.76 ■ Gallbladder: Transverse. Short-axis view of the gall-bladder measuring the anterior wall thickness. (Ultrasound contributor: Robinson M. Ferre, MD.)

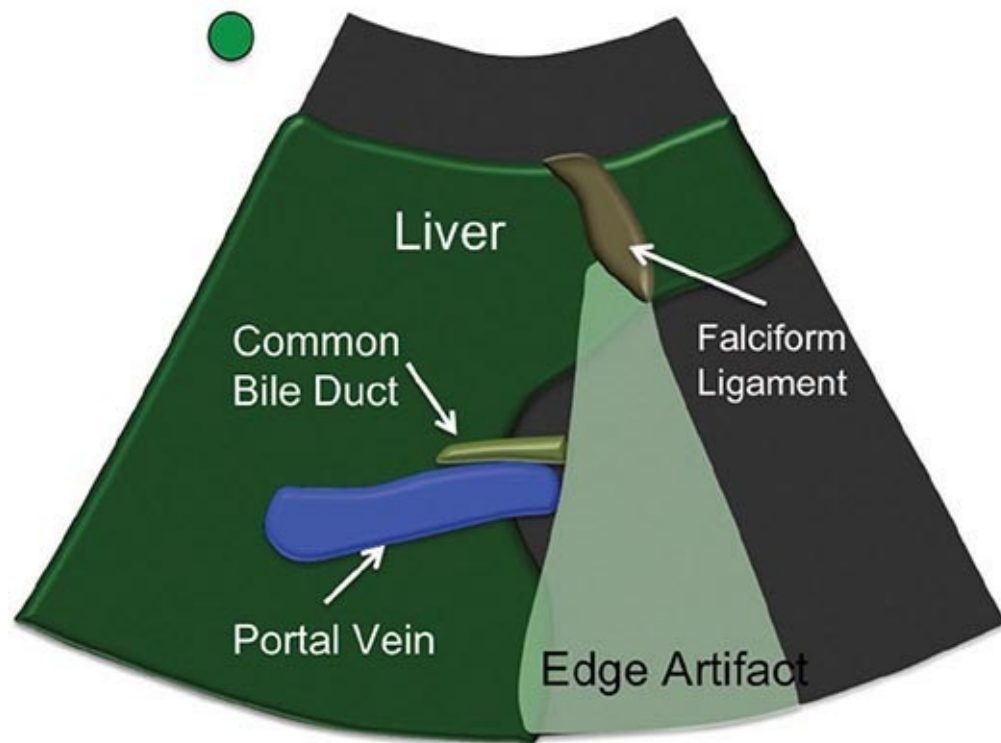
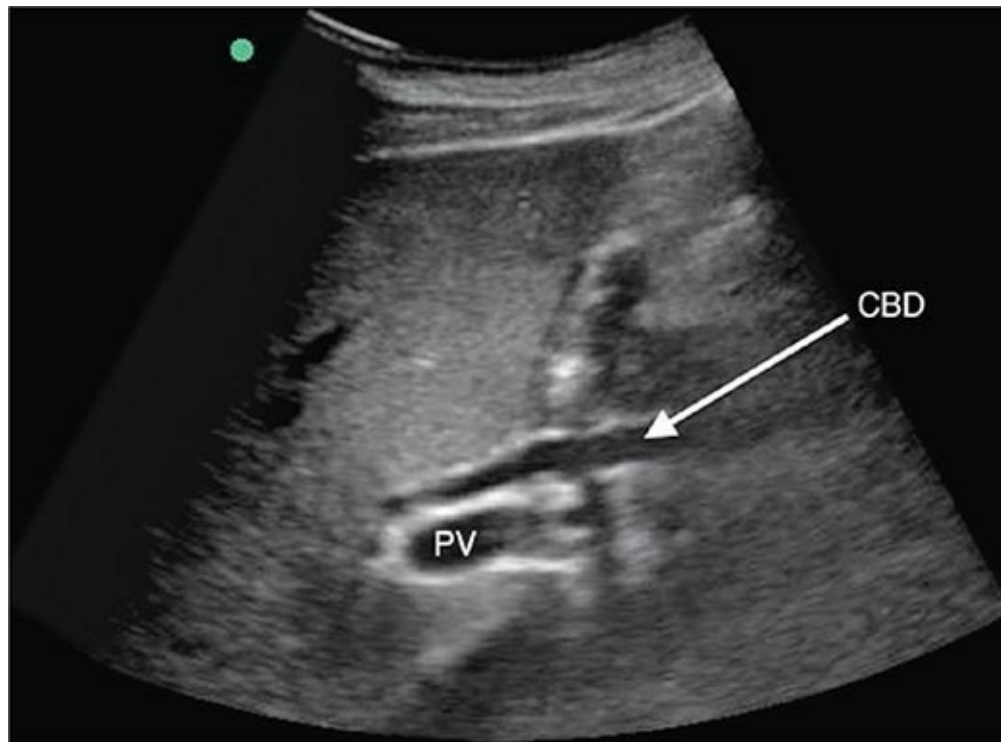


FIGURE 24.77 ■ Gallbladder: Common Bile Duct. Long-axis view of the portal vein (PV) and dilated common bile duct (CBD). The CBD is measured from inside wall to inside wall. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

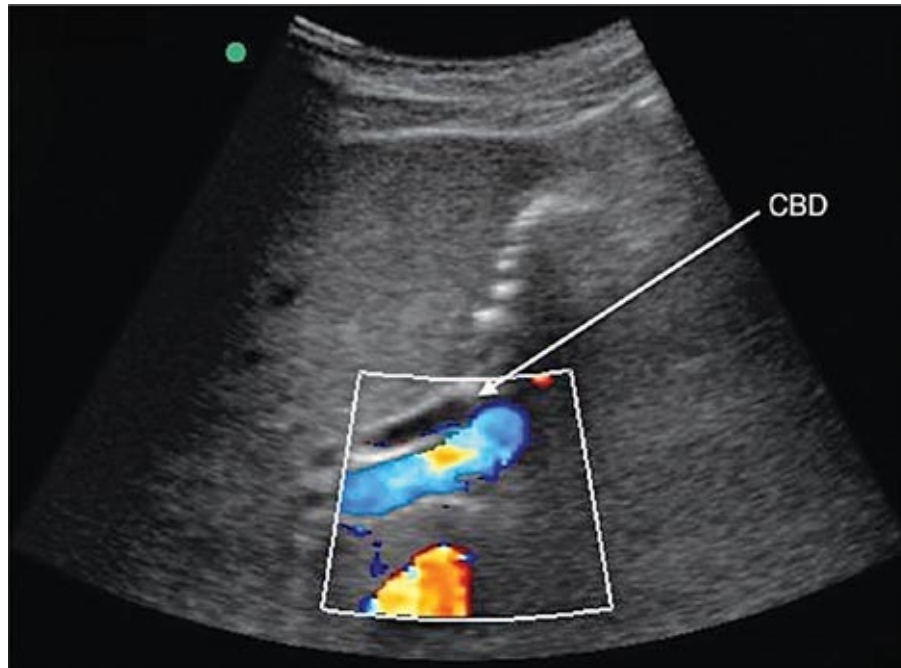


FIGURE 24.78 ■ Gallbladder: Common Bile Duct, Color Flow Doppler. Long-axis view of the portal vein (color flow) and dilated common bile duct (CBD) without color flow. (Ultrasound contributor: Max Palatnik, MD.)

Abnormal Findings

- Gallstones: Hyperechoic (bright) oval to round structure(s) within the gallbladder, often with acoustic shadowing ([Fig. 24.79A,B](#)). Gallstones should be mobile unless impacted in the neck of the gallbladder ([Fig. 24.80](#)). Rolling the patient will allow assessment of gallstone mobility.

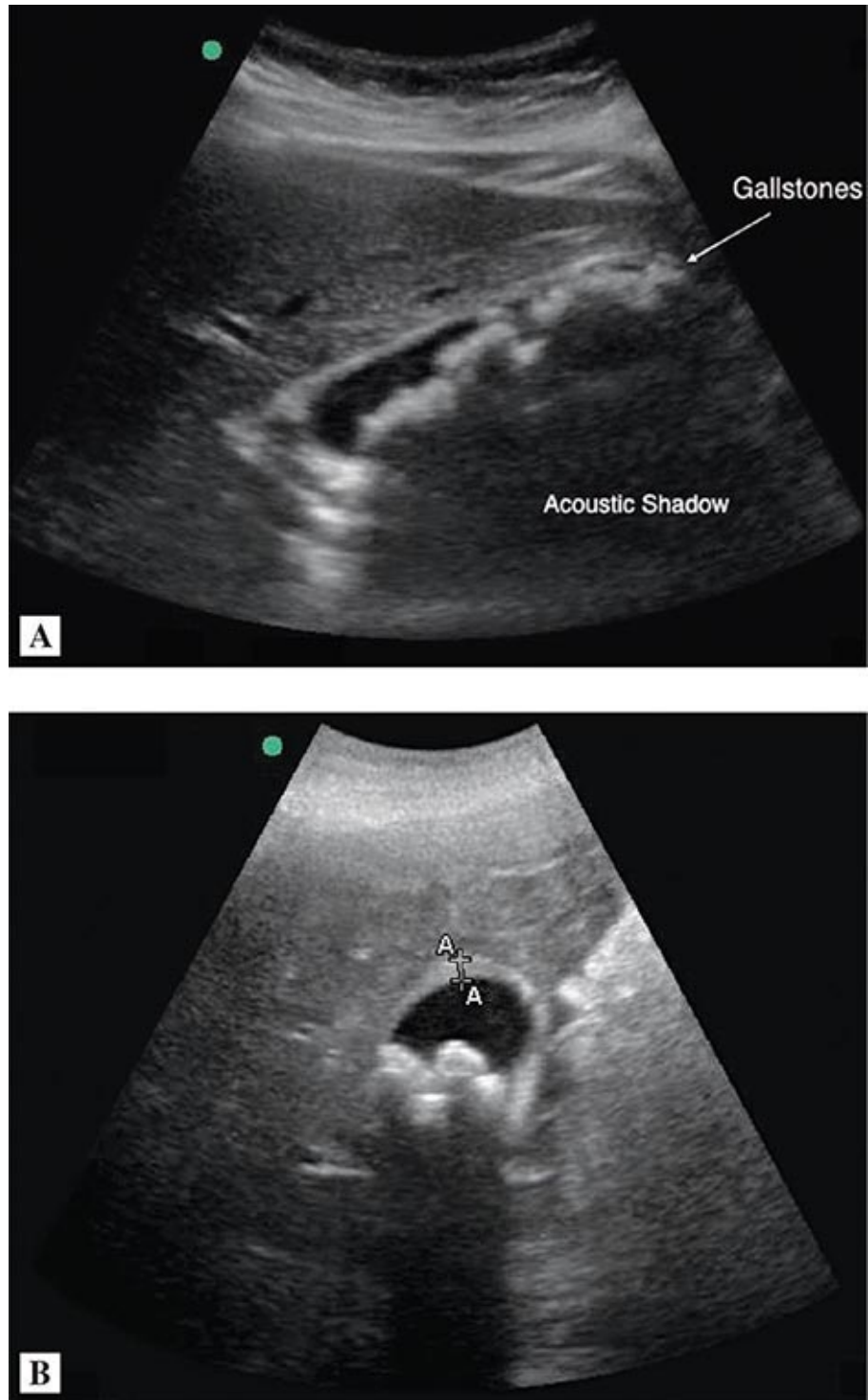


FIGURE 24.79A, B ■ Gallbladder: Gallstones. Long- and short-axis views of the gallbladder with a large number of gallstones casting an acoustic shadow posterior to the calcified stones. (Ultrasound contributor: Shannon Snyder, MD, RDMS.)



FIGURE 24.80 ■ Gallbladder: Sagittal View, Gallstone. Long-axis view of the gallbladder with a single gallstone lodged in the gallbladder neck. (Ultrasound contributor: Robinson M. Ferre, MD.)

- Pericholecystic fluid: An anechoic (dark) stripe that borders the outer gallbladder wall and the liver. It will be visible in two views ([Fig. 24.81A,B](#)).

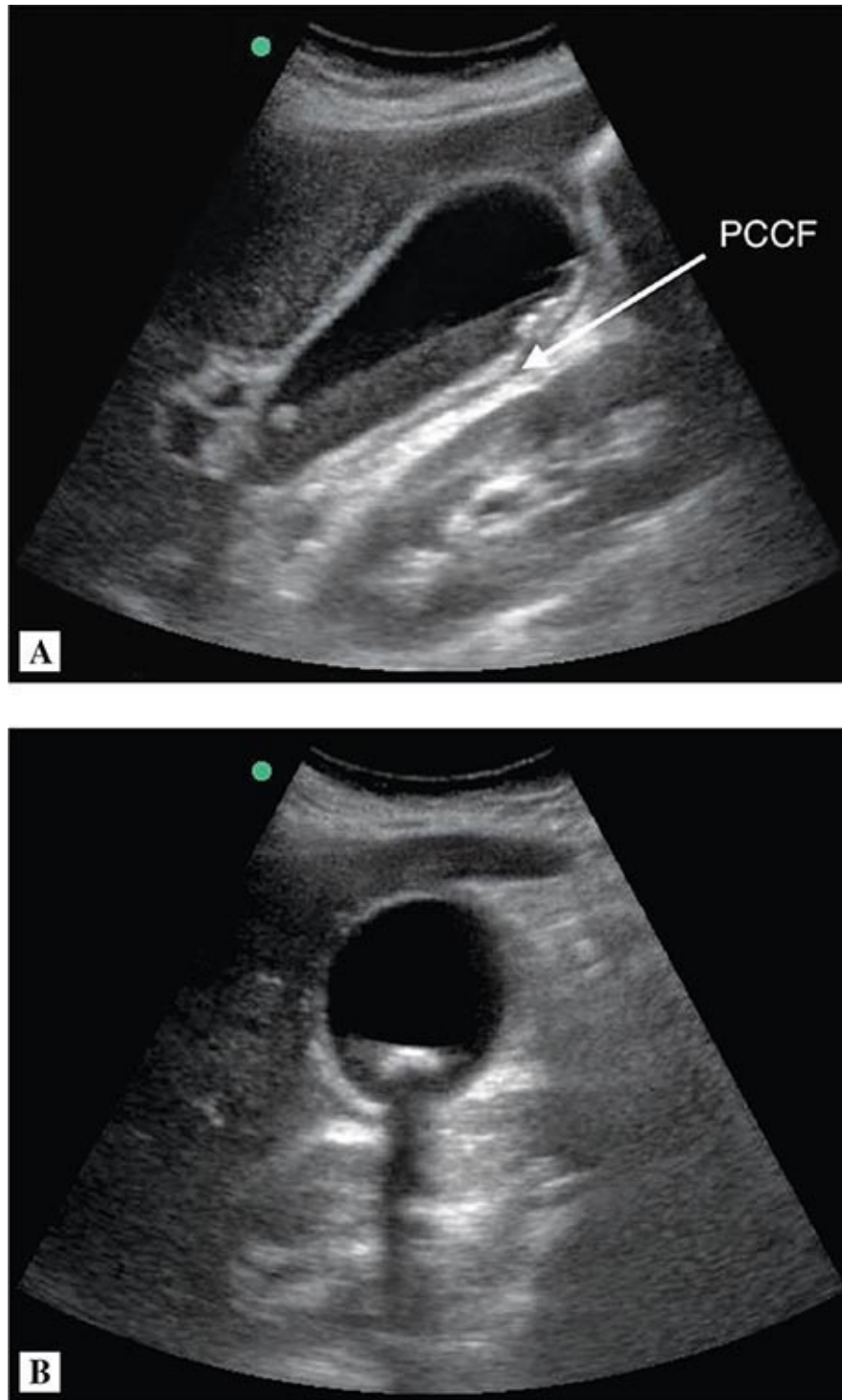


FIGURE 24.81A, B ■ Gallbladder. Gallbladder sludge, gallstone, and pericholecystic fluid (PCCF). Long- and short-axis view of the gallbladder showing a gallstone and biliary sludge. There is also a thin stripe of PCCF. (Ultrasound contributor: Jeremy Boyd, MD.)

- Thickened gallbladder wall: A gallbladder wall that measures greater than 3 mm is considered abnormal ([Fig. 24.82](#)).

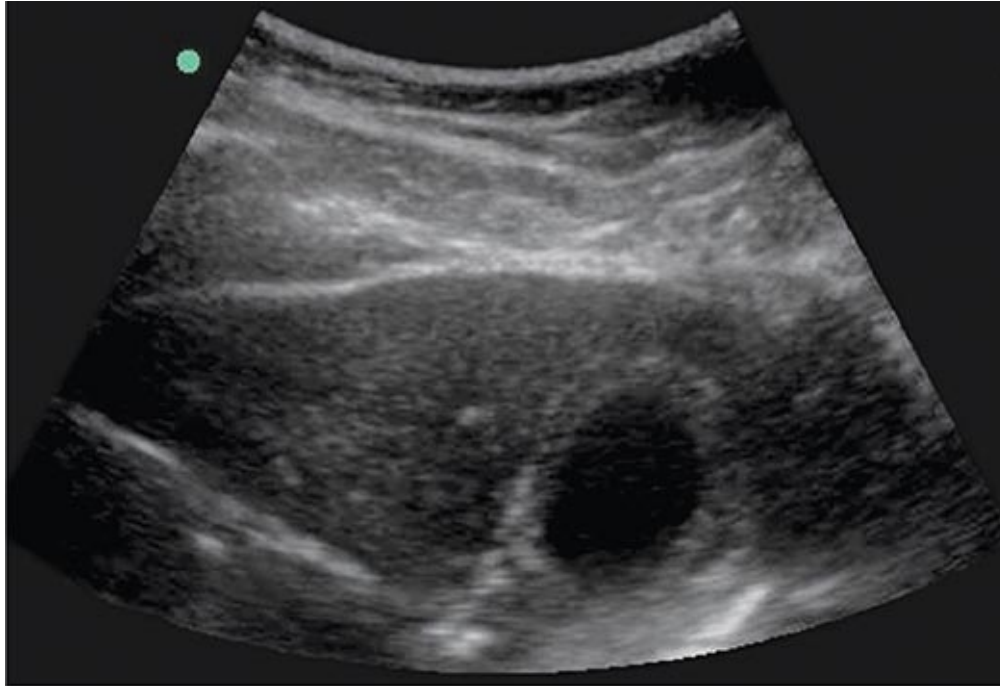


FIGURE 24.82 ■ Gallbladder: Transverse View, Cholecystitis. Short-axis view of the gallbladder with thickening of the wall. (Ultra-sound contributor: Jordan Rupp, MD.)

- Sonographic Murphy sign: When direct visualized compression of the gallbladder by the ultrasound probe reproduces the patient's symptoms of abdominal pain and this pain is not reproduced by compression with the ultrasound probe elsewhere in the surrounding area.
- Dilated CBD: A CBD with an internal diameter greater than 6 mm is dilated; however, documented measurements up to 8 to 9 mm can be normal in the elderly. One rule of thumb is that the CBD can measure up to 6 mm up to age 60 and thereafter an increase of 1 mm per decade of life represents the normal range.

Pearls

1. Other positions—including prone, right lateral decubitus, semi-erect, and standing—may be helpful in scanning the gallbladder and are patient specific.
2. Having the patient hold their breath after deep inspiration may help the gallbladder descend into view below the ribcage.
3. Always measure the anterior wall of the gallbladder. The thickness of the posterior wall often appears falsely thickened by posterior acoustic enhancement.
4. Small gallstones (1-3 mm) may not be large enough to cause acoustic

shadowing. Increasing the frequency and ensuring that your focal zone is on the area of interest will increase the likelihood that shadowing will be seen.

5. The CBD lies just anterior to the portal vein, a structure with markedly hyperechoic walls compared to the hepatic veins. Placing the patient in the left lateral decubitus position may help visualize the CBD.

RENAL AND BLADDER ULTRASOUND

Clinical Summary

Ultrasound of the kidneys and bladder can yield helpful diagnostic information for patients presenting with symptoms of urinary retention, decreased urinary output, or abdominal/flank pain consistent with renal colic. Obstructive uropathy due to kidney stones is the principal pathology identified with emergency physician–performed renal ultrasound. Bladder volume is easily calculated by viewing and measuring the bladder in two planes. In focused renal ultrasound, the goal is not to identify renal or ureteral calculi; rather, the kidneys are evaluated for hydronephrosis. The presence of hydronephrosis in the patient with renal colic is presumed to be a direct result of ureteral obstruction. There are no accurate means of determining the degree of obstruction by the presence of hydronephrosis.

Indications

- Flank/abdominal pain
- Renal colic
- Hematuria
- Urinary retention
- Decreased urinary output

The recommended sonographic approach to the kidney is similar to that for the RUQ and LUQ windows in the E-FAST examination previously discussed, with the probe angled toward the retroperitoneum for better views of the kidneys. The coronal view allows the sonographer to visualize the right or left kidney from the superior to inferior poles. Trans-verse or axial views of the entire kidney should be obtained as well. Renal EUS is most easily interpreted when comparative images are obtained between the right and left kidneys. The

bladder view is also nearly identical to that of the E-FAST exam described earlier. However, rather than focusing on the potential spaces of the peritoneum, attention is focused on the bladder and its contents.

Views for Emergency Department Renal and Bladder Ultrasound

1. Coronal or sagittal kidney view
2. Transverse kidney view
3. Sagittal bladder view
4. Transverse bladder view

Equipment: Recommended Transducers for Renal Ultrasound

- Convex array
- Phased array

Patient Position

- The patient is supine.
- Alternatively, the patient may be placed in a prone position with the probe placed in the midscapular line and the transducer directed toward the abdomen.

Technique

Kidney: Right and Left Coronal Views

- The transducer indicator is oriented toward the patient's head.
- The transducer is directed as a coronal section through the body in the mid to posterior axillary lines ([Fig. 24.83](#)). Begin scanning between the 9th and 11th ribs on the right and the 8th and 11th ribs on the left.



FIGURE 24.83 ■ Renal Ultrasound. The transducer is directed as a coronal section through the body in the mid to posterior axillary lines. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the liver, right kidney, renal cortex (with pyramids), and central renal sinus ([Fig. 24.84](#)).

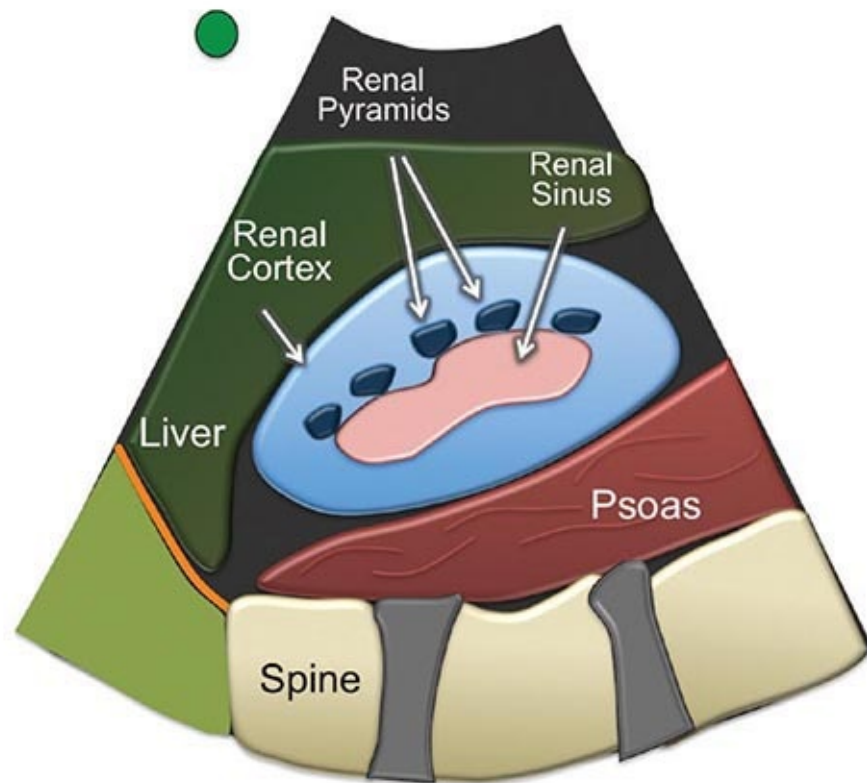


FIGURE 24.84 ■ Renal Ultrasound, Right Coronal View. The liver, right kidney, renal cortex (with pyramids), and central renal sinus are seen. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

- Identify the spleen, left kidney, renal cortex (with pyramids), and central renal sinus.

Kidney: Right and Left Transverse Views

- Obtain the coronal view as directed above, but rotate the transducer 90 degrees counterclockwise to visualize the renal parenchyma and central renal sinus in a cross-sectional view ([Fig. 24.85](#)).

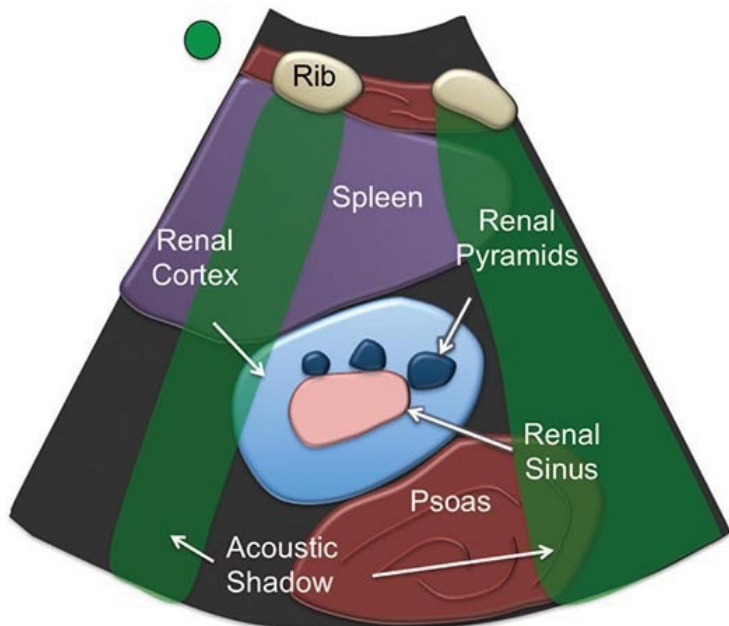
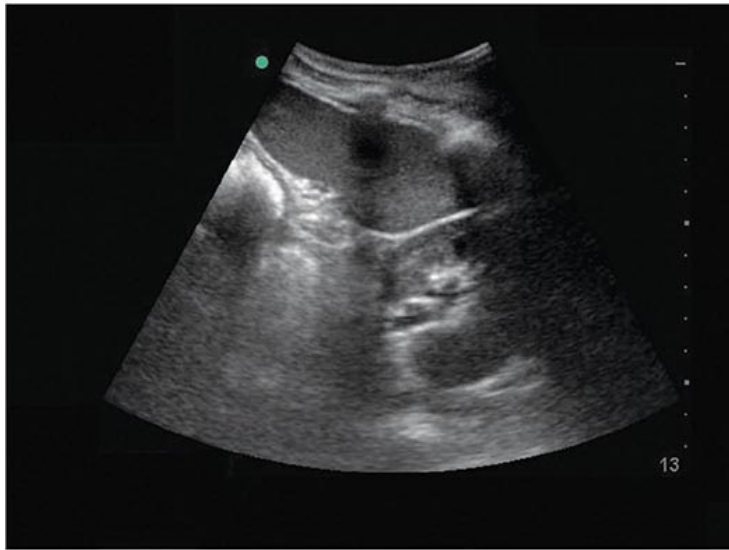


FIGURE 24.85 ■ Renal Ultrasound, Left Transverse View. Rotate the transducer 90 degrees counterclockwise to visualize the renal parenchyma and central renal sinus in a cross-sectional view. (Illustration contributor: Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

Bladder: Sagittal View

- With the indicator oriented toward the patient's head, the transducer is placed just above the symphysis pubis and is directed into the pelvis (see [Fig. 24.17](#)).
- Identify the bladder (triangular in this view when fully dis-tended), uterus (pear-shaped if present), prostate, seminal vesicles, and rectum (see [Figs. 24.18](#) and [24.19](#)).

Bladder: Transverse View

- With the indicator oriented toward the patient's right, the transducer is placed about 1 to 2 cm above the symphysis pubis, with the transducer angled into the peritoneum (see [Fig. 24.20](#)).
- Identify the bladder (rectangular in this view when fully dis-tended), uterus (oval hyperechoic structure if present), prostate, seminal vesicles, and rectum (see [Fig. 24.21](#)).

Findings

- Hydronephrosis: Dilatation of the bright renal sinus with black, anechoic fluid ([Fig. 24.86](#)). Hydronephrosis is graded based on its extension into the secondary calyces and whether there is accompanying cortical thinning ([Fig. 24.87](#)).

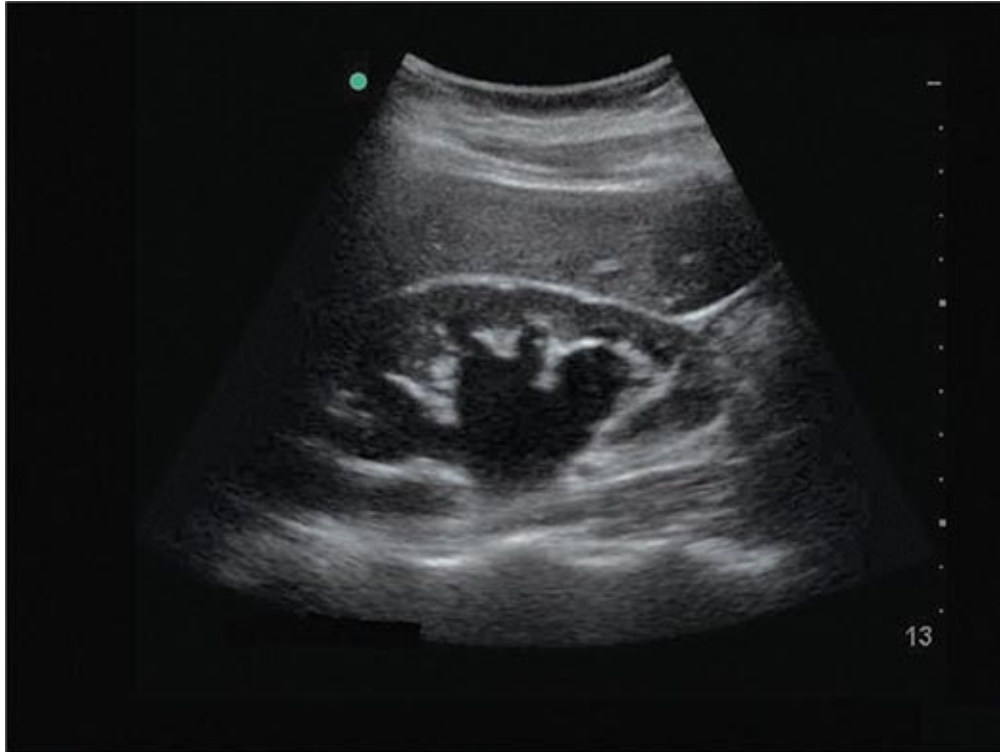


FIGURE 24.86 ■ Renal Ultrasound: Hydronephrosis. Moderate hydronephrosis is seen as dilatation of the bright renal sinus and secondary calyces by anechoic (black) fluid. (Ultrasound contributor: Shannon Snyder, MD, RDMS.)

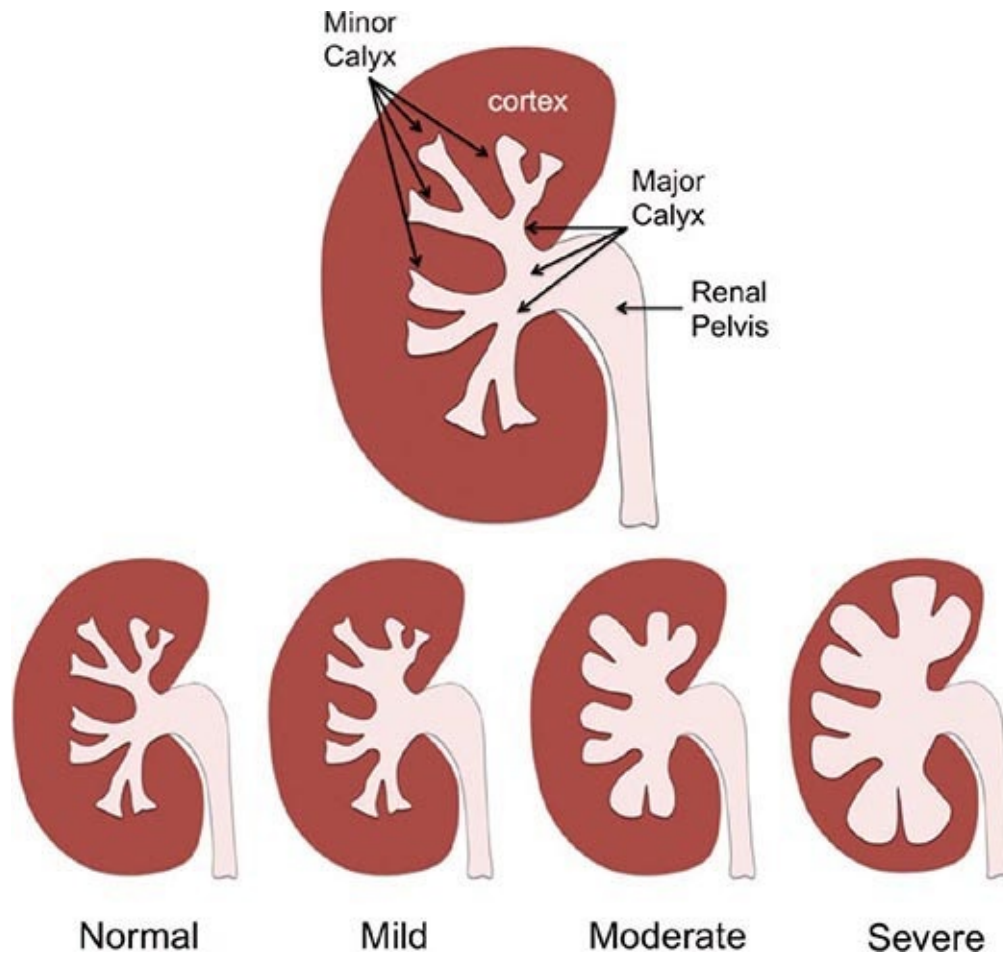


FIGURE 24.87 ■ Hydronephrosis Grading. Hydronephrosis is graded based on its extension into the secondary calyces and whether there is accompanying cortical thinning. Mild hydronephrosis does not involve the secondary calyces, moderate does, and severe hydro-nephrosis is accompanied by cortical thinning. (Illustration contributor: Robinson M. Ferre, MD.)

- Renal calculi: Bright hyperechoic oval/round structures within the cortex or renal sinus (acoustic shadowing is often present). With color flow Doppler, uroliths will often demonstrate twinkling artifact deep to their reflective echogenic surface ([Fig. 24.88](#)).

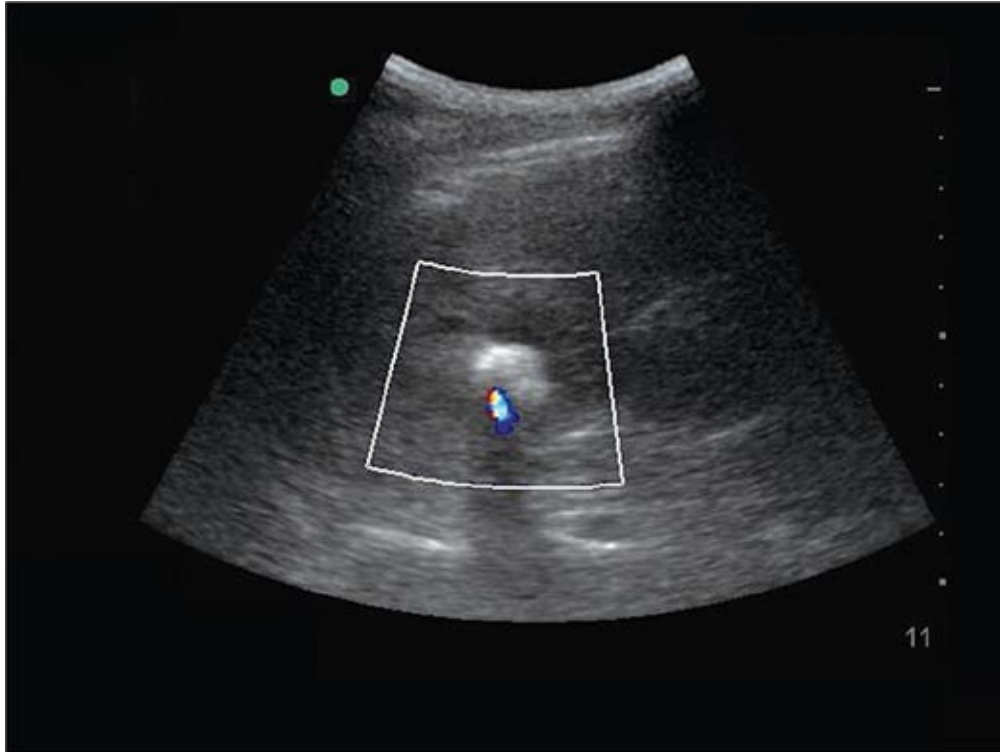


FIGURE 24.88 ■ Renal Calculi, Twinkle Artifact. With color flow Doppler, uroliths will often demonstrate twinkling artifact deep to their reflective echogenic surface. (Ultrasound contributor: Jeremy S. Boyd, MD.)

- Urinary retention: By calculating the volume of the bladder, one may determine the presence of urinary retention. Greater than 100 mL of retained postvoid urine is diagnostic.

Pearls

1. Rib shadows will be evident with the coronal view and trans-verse views. Have the patient hold his or her breath in inspiration and move the transducer a rib space higher or lower to visualize the kidney from the superior to the inferior pole.
2. The presence of a ureteral jet confirms the passage of urine into the bladder from a specific side. Its absence is sensitive, but not specific, for complete ureteral obstruction ([Fig. 24.89](#)).

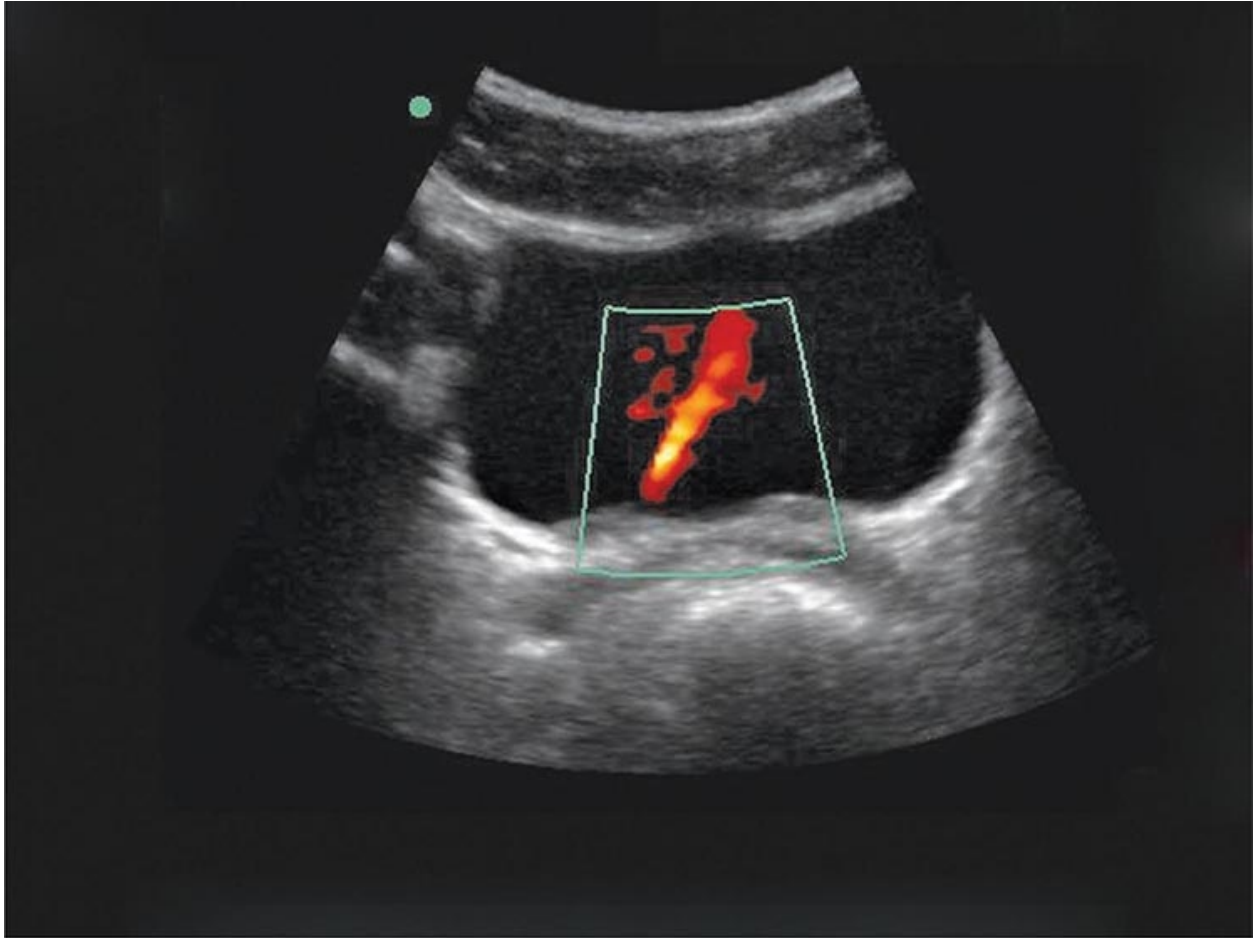


FIGURE 24.89 ■ Ureteral Jet. The presence of a ureteral jet confirms the passage of urine into the bladder from a specific ureter. (Ultrasound contributor: Jeremy S. Boyd, MD.)

3. When evaluating for renal colic, stones may frequently be seen on the bladder views when they are located at the ureterovesicular junction (UVJ) ([Fig. 24.90](#)).

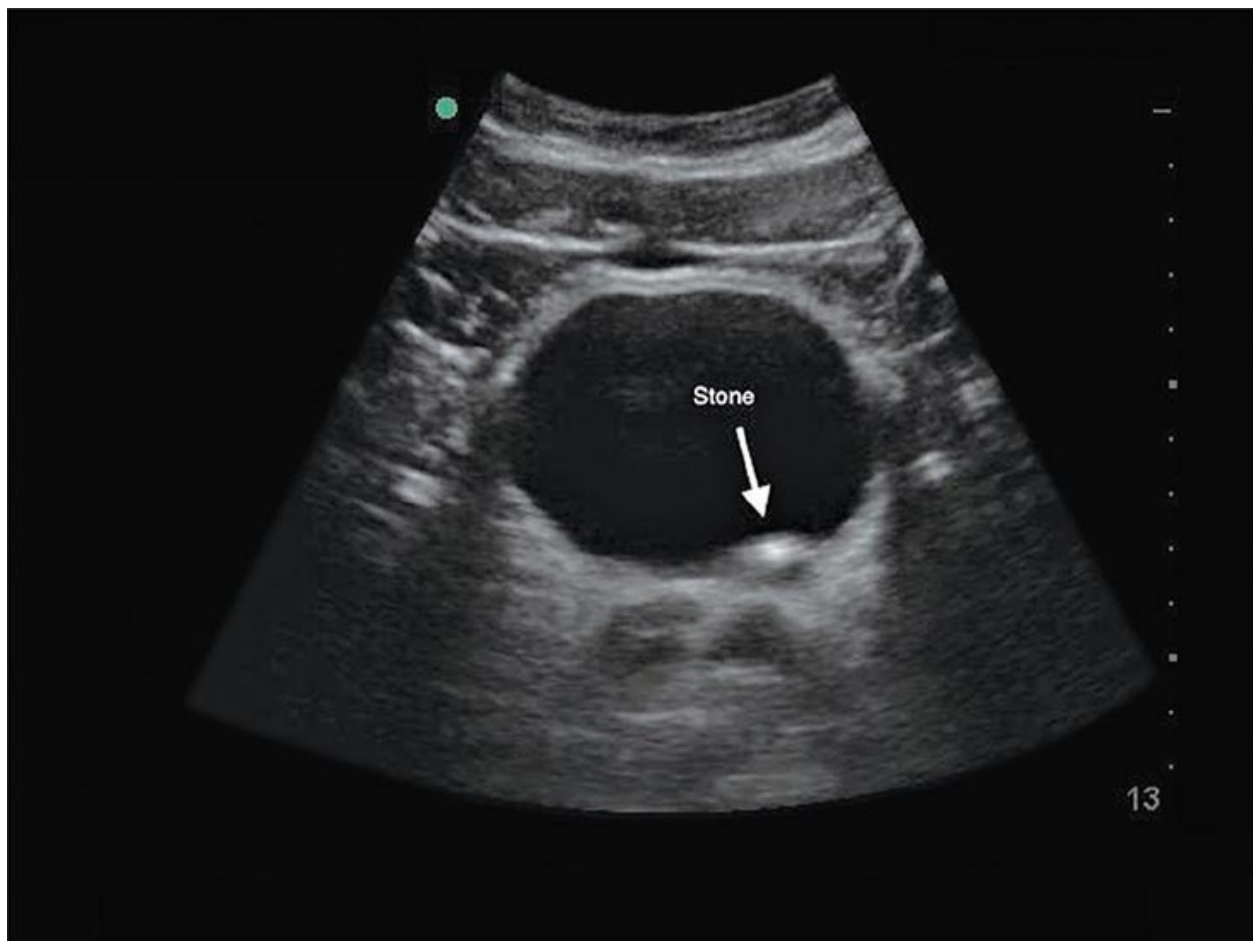


FIGURE 24.90 ■ Uterovesicular Junction (UVJ) Stone. Stones may frequently be seen on the bladder views when they are located at the UVJ. (Ultrasound contributor: Robinson M. Ferre, MD.)

PREGNANCY ULTRASOUND

Clinical Summary

Ultrasound is frequently used to evaluate the patient presenting with pelvic pain and/or vaginal bleeding and may aid in the diagnosis of numerous forms of pathology such as urinary retention, molar pregnancies, and ovarian torsion. Pelvic EUS is most commonly used in the evaluation of pregnant patients who present with signs concerning for ectopic pregnancy or miscarriage. The primary goal of pelvic EUS is to evaluate for the presence of an intrauterine pregnancy (IUP) and assess for risk factors that are concerning for an ectopic pregnancy. Ultrasound of pregnant patients is accomplished with two different scanning

techniques: trans-abdominal and transvaginal.

Pregnant patients presenting with abdominal pain or vaginal bleeding during the 1st trimester must be evaluated for the presence of an ectopic pregnancy. This is commonly accomplished in the ED setting by identifying an IUP in a patient who is at low risk for a heterotopic pregnancy. Patients not receiving infertility treatments with evidence of an IUP can in many circumstances be safely discharged from the ED.

Indications

- Pelvic/abdominal pain
- Vaginal bleeding
- Suspected pregnancy

Required Views for the Emergency Department Pelvic Ultrasound

- Sagittal view
- Transverse view

Equipment: Recommended Transducers for Pelvic Sonography

- Convex array (transabdominal)
- Endocavitary probe (transvaginal)

Patient Positioning

- The patient is supine for transabdominal views.
- The patient is supine and preferably in the lithotomy position for transvaginal views.

Techniques

Transabdominal Sonography: Sagittal View

- With the indicator pointed toward the patient's head, place transducer superior to the symphysis pubis ([Fig. 24.91](#)).



FIGURE 24.91 ■ Transabdominal Sagittal View. With the indicator pointed toward the patient's head, place the transducer superior to the symphysis pubis. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the bladder, uterus, rectum, ovaries, and the rectouterine pouches (pouch of Douglas). Identify an IUP if present ([Fig. 24.92](#)).

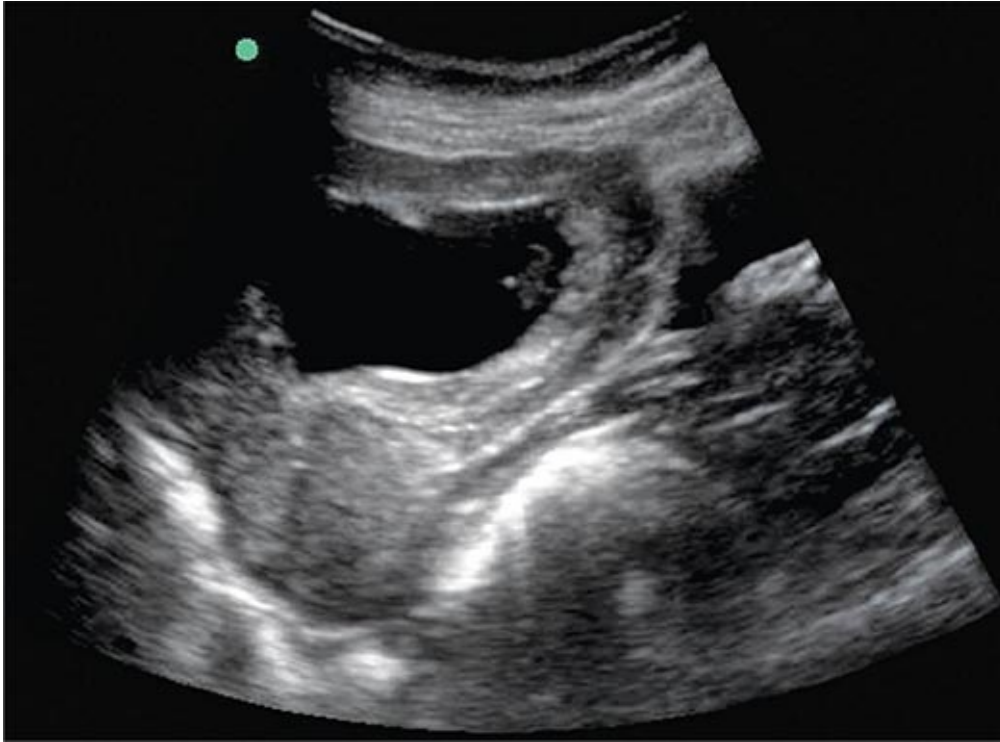


FIGURE 24.92 ■ Transabdominal Sagittal View. Gravid uterus showing cervix and vagina posterior to the bladder. (Ultrasound contributor: Robinson M. Ferre, MD.)

Transabdominal Sonography: Transverse View

- From the sagittal view, rotate the transducer 90 degrees counterclockwise so the transducer indicator points to the patient's right. The transducer should be superior to symphysis pubis ([Fig. 24.93](#)).



FIGURE 24.93 ■ Transabdominal Transverse View. From the sagittal view, rotate the transducer 90 degrees counterclockwise so the transducer indicator points to the patient's right. (Photo contributor: Lawrence B. Stack, MD.)

- Identify the bladder, uterus, rectum, ovaries, and the vesicouterine and rectouterine (pouch of Douglas) areas ([Fig. 24.94](#)).

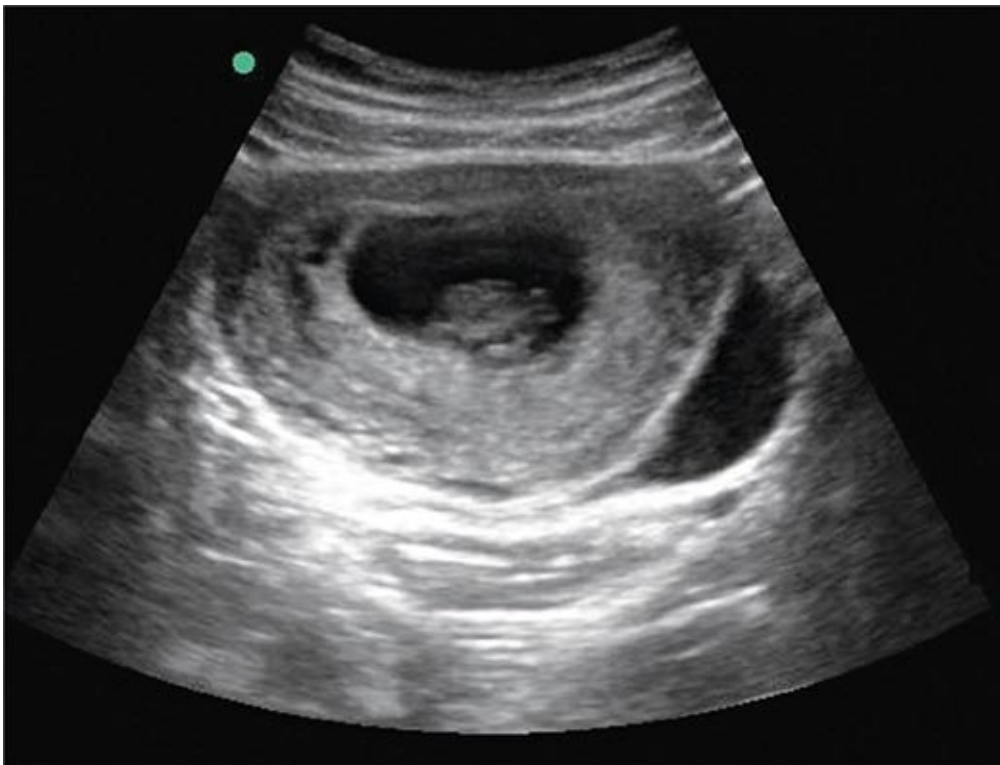


FIGURE 24.94 ■ Transabdominal Transverse View: Uterus. Firsttrimester pregnancy showing uterus with gestational sac and fetal pole. Some of the urinary bladder is seen deep and lateral (right on the image) to the uterus. (Ultrasound contributor: Robinson M. Ferre, MD.)

Transvaginal Sonography: Sagittal View

- With a condom or commercially available endocavitary probe cover covering the transducer, insert the transducer with the marker directed toward the ceiling. The transducer should be inserted until it rests in the anterior fornix of the vagina ([Fig. 24.95](#)).



FIGURE 24.95 ■ Transvaginal Sagittal View. The transducer is directed toward the anterior fornix in a line through the umbilicus (**A**); it is placed into the vagina (**B**). The probe is advanced gradually. (Photo contributor: Michael J. Lambert, MD.)

- Identify the uterus, rectum, ovaries, and rectouterine pouch ([Fig. 24.96](#)). Identify an IUP if present.

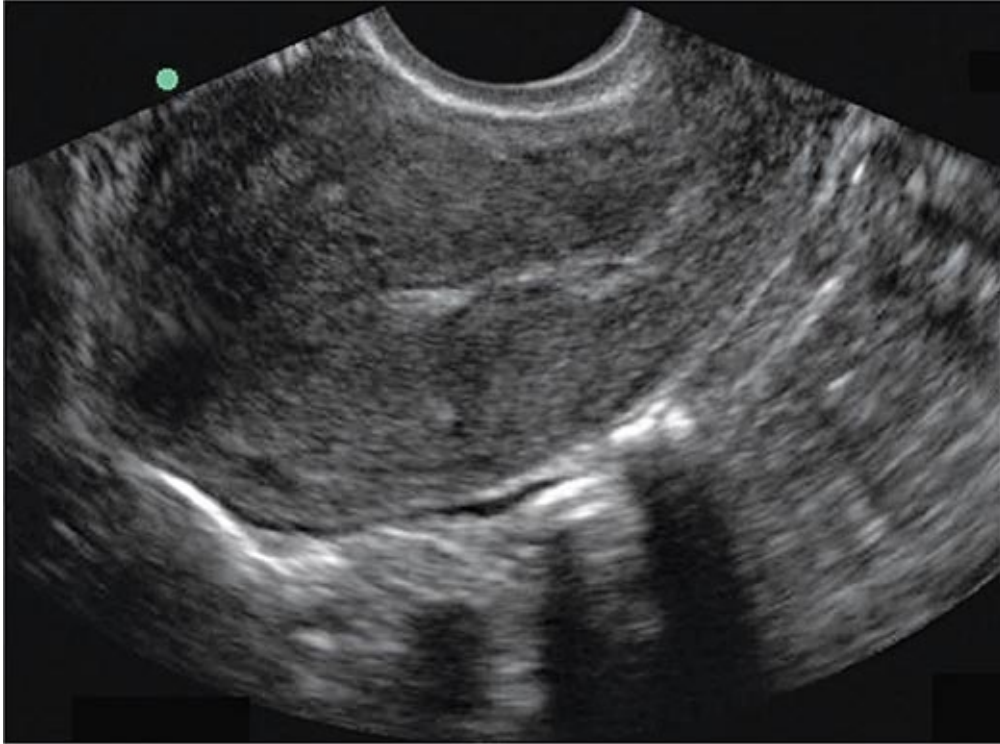


FIGURE 24.96 ■ Transvaginal: Sagittal View. The uterus with endometrial stripe. (Ultrasound contributor: Robinson M. Ferre, MD.)

Coronal View

- From the sagittal view, rotate the transducer counterclockwise 90 degrees so that the indicator is directed toward the patient's right ([Fig. 24.97](#)).

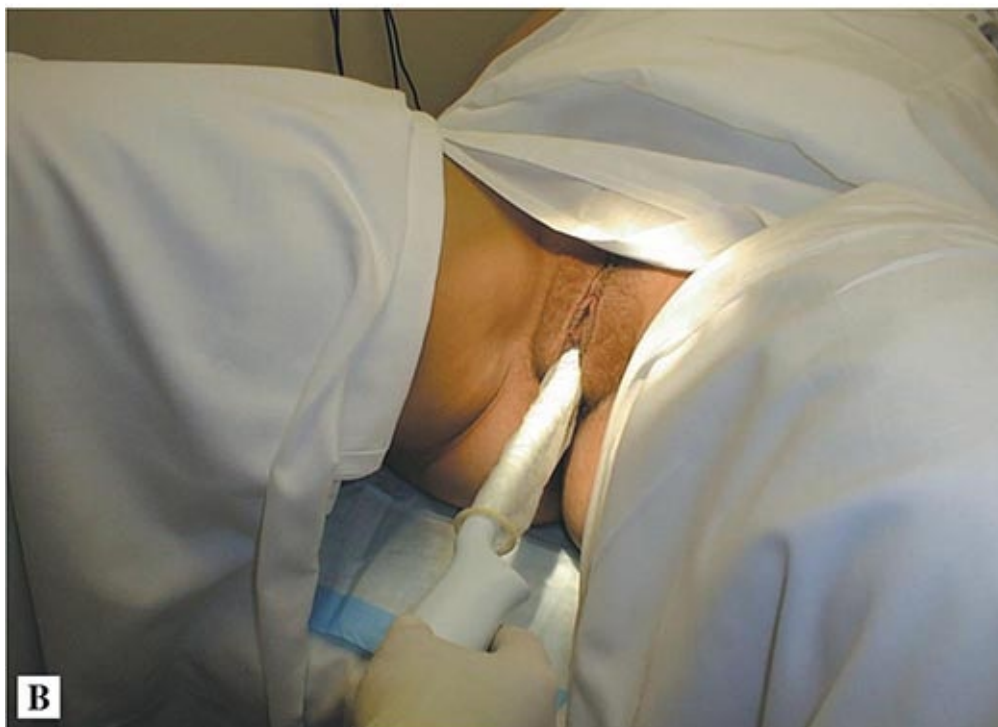


FIGURE 24.97 ■ Transvaginal Coronal View. From the sagittal view, the transducer is rotated counterclockwise 90 degrees (**A**) and directed toward the posterior fornix in a line through the umbilicus (**B**). (Photo contributor: Michael J. Lambert, MD.)

- Identify the uterus, rectum, ovaries, and rectouterine pouch ([Fig. 24.98](#)).



FIGURE 24.98 ■ Transvaginal: Coronal. The uterus with endome-trial stripe. Left ovary visualized adjacent to the uterus on the right of the image. (Ultrasound contributor: Robinson M. Ferre, MD.)

Abnormal Findings

- Free intraperitoneal fluid: Anechoic (black) bands of fluid located in the rectouterine pouch (pouch of Douglas).
- Ectopic pregnancy: Most commonly, an ectopic pregnancy is identified as an adnexal mass with an associated empty uterus ([Figs. 10.39](#) and [24.99](#)). At times, an extrauterine gestational sac with or without a fetal pole may be seen. Rarely, a pseudogestational sac (an anechoic fluid collection < 1 cm in diameter within the uterus) may mimic an early gestational sac.



FIGURE 24.99 ■ Transvaginal: Right Adnexa. Ectopic pregnancy located in the right adnexa. (Ultrasound contributor: Robinson M. Ferre, MD.)

- **IUP:** The 1st evidence of an IUP is a gestational sac with two thick, concentric echogenic rings within the body or fundus of the uterus. Because this may be confused with a pseudo-gestational sac, for EUS, the 1st definitive evidence of an IUP is a gestational sac with a visualized yolk sac and/or fetal pole. A live IUP has both a fetal pole and cardiac activity. (See [Figs. 10.41](#) and [10.42](#).)
- **Abnormal IUP:** A fetal pole greater than 7 mm without cardiac activity is diagnostic of a fetal demise. A gestational sac greater than 16 mm without a fetal pole and an enlarged yolk sac greater than 7 mm are concerning for failed IUP, but are not diagnostic. A mean gestational sac diameter of 25 mm or more without a fetal pole is diagnostic of failed IUP. (See [Figs. 10.45](#) and [10.46](#).)
- **Interstitial or cervical ectopic pregnancy:** An IUP must be within the body or fundus of the uterus. A gestational sac that is within the cornua or cervix of the uterus, although technically within the uterus, is still considered an ectopic pregnancy. A gestational sac within 5 to 7 mm of the border of the myometrium is concerning for interstitial ectopic pregnancy. (See [Figs. 10.41](#) and [10.42](#).)

Pearls

1. A full bladder improves visualization of structures posterior to the bladder in the transabdominal approach. An empty bladder is necessary for transvaginal ultrasound.
2. A small amount of free fluid found in the pouch of Douglas may be physiologic.
3. The bladder is a good landmark to find initially for orientation to the anatomy when scanning transabdominally.
4. When scanning transvaginally in the sagittal view, identify the endometrial stripe from the fundus of the uterus to the cervix. This is accomplished by tilting the probe (anteriorly to posteriorly) while maintaining a sagittal plane of the uterus.
5. Return to the fundus of the uterus in a sagittal view and slowly evaluate the right and then left borders of the uterus in the longitudinal axis.
6. Turn the transducer counterclockwise 90 degrees to enter the coronal plane. The uterus usually appears as an oval. Scan posteriorly to the cervix and then superiorly to the fundus of the uterus to exclude the presence of a bicornuate uterus.
7. If a pregnancy or intrauterine sac is identified with fetal cardiac activity, M-mode, not Doppler, should be used for documentation of fetal heart rate.
8. Evaluate both adnexa/ovaries individually by scanning each side in both coronal and sagittal planes. The ovaries ideally will be located anteromedially to the external iliac vessels. This is often only a guide to their location, and a methodical approach is often required to visualize both ovaries.
9. If the uterus is difficult to identify on transvaginal scan, withdraw the transducer slightly. A common error is insertion of the transducer too far, thus bypassing the uterus and imaging only bowel.

PROCEDURAL GUIDANCE: ULTRASOUND- GUIDED VENOUS ACCESS

Clinical Summary

Vascular access is a necessity for evaluation and treatment of many patients in

the ED. Traditionally, knowledge of common venous anatomy in experienced hands was the standard method of obtaining venous access. Factors such as obesity, prior access, and volume depletion can increase the difficulty of the conventional approach. This increased difficulty not only leads to failure to obtain access, but also increases the risk of complications.

Ultrasound-guided vascular access reduces the variables associated with the traditional landmark-based approach for central venous access and has become the standard of care. Additionally, ultrasound is useful for both difficult peripheral venous and arterial access.

Indications

- Need for venous access
- Obscured landmarks (obesity, trauma, etc)
- Cardiac arrest

Equipment

- Linear
- Convex

Patient Preparation

- Position and prepare the patient as per convention for access and use a sterile sheath for the transducer.
- For central venous access, the patient is supine with the access site in a dependent position.

Technique

There are two main approaches to all venous access.

Short-Axis Approach

- In a transverse view, identify the structures of interest:
 - Vein
 - Corresponding artery
 - Others (nerve, trachea, etc)

- Confirm the vein as the collapsible vascular structure, and trace the vessel to ensure there are no anatomic variants that may disrupt your procedure.
- In the short-axis approach, maintain your transverse view and position the vein in the center of the image.
- Introduce the needle immediately distal to the transducer's edge at approximately 45-degree angle to the patient's skin and perpendicular to the long axis of the probe ([Fig. 24.100](#)).

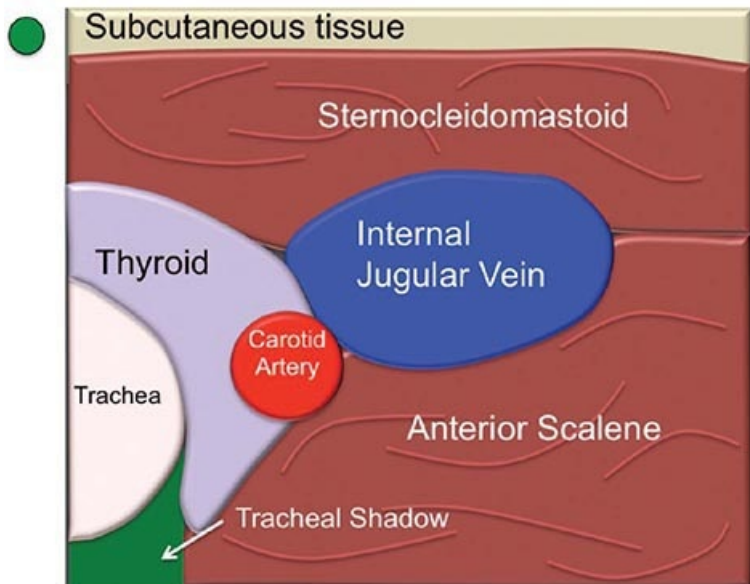
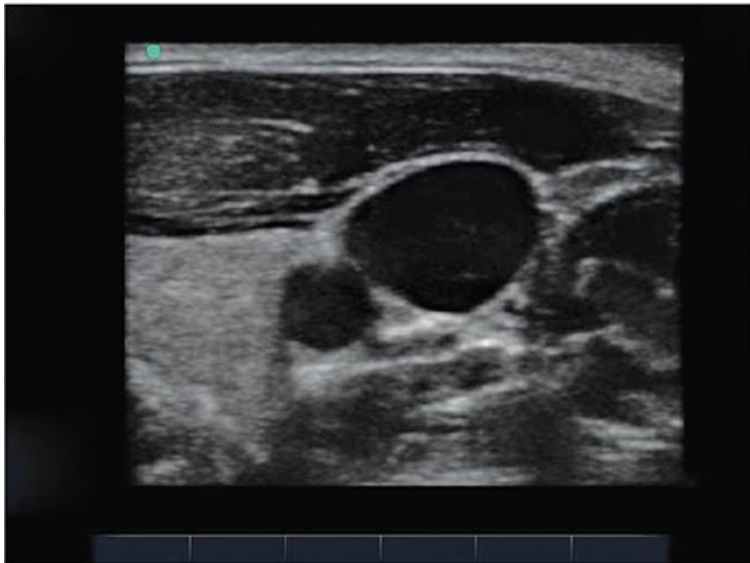


FIGURE 24.100 ■ Internal Jugular Vein Cannulation, Short-Axis Approach. The internal jugular vein and carotid artery are identified in cross-section. Introduce the needle immediately distal to the transducer's edge at approximately a 45-degree angle to the patient's skin and perpendicular to the long axis of the probe. (Photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Jeremy S. Boyd, MD; illustration contributor: Robinson M. Ferre, MD.)

- Maintaining the probe's orientation to the vessel, slide the probe away from the puncture site to maintain visualization of the tip of your needle by observing:
 - The needle tip
 - Tissue movement
 - Ring-down artifact from the needle
- Follow the tip of the needle until it contacts the anterior surface of the vein.
- Continue to advance until a venous flash is seen and the needle has reached the intended depth. If no flash is seen at the appropriate depth in the correct path, slowly withdraw the needle and observe for venous return.
- Continue catheter placement as per common practice.

Long-Axis Approach

- In a transverse view, identify the structures of interest and confirm the vein as the collapsible vascular structure as above.
- Once the vein of interest is identified and centered on the screen, rotate the probe so that the transducer is in the same axis as the vein.
- Introduce the needle at the distal edge of the transducer.
- Direct the needle along the long axis of the probe, careful to keep the needle completely in line with the probe and ultra-sound beam ([Fig. 24.101](#)).

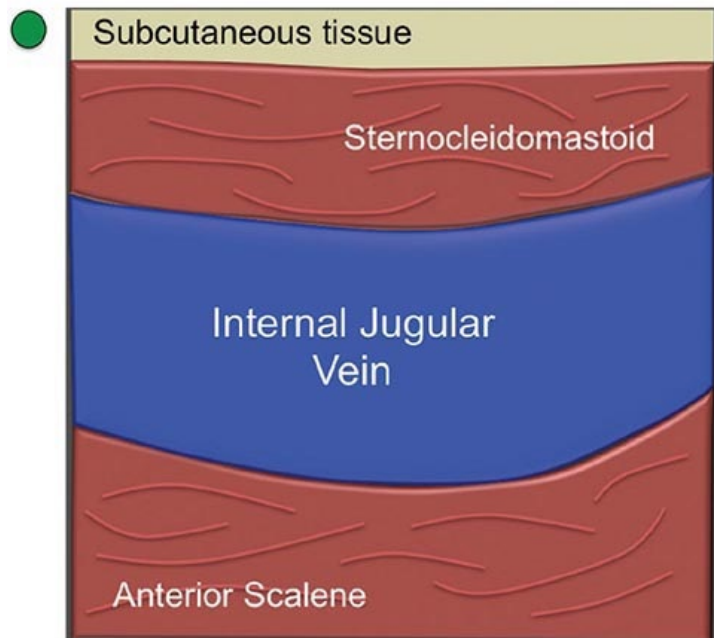
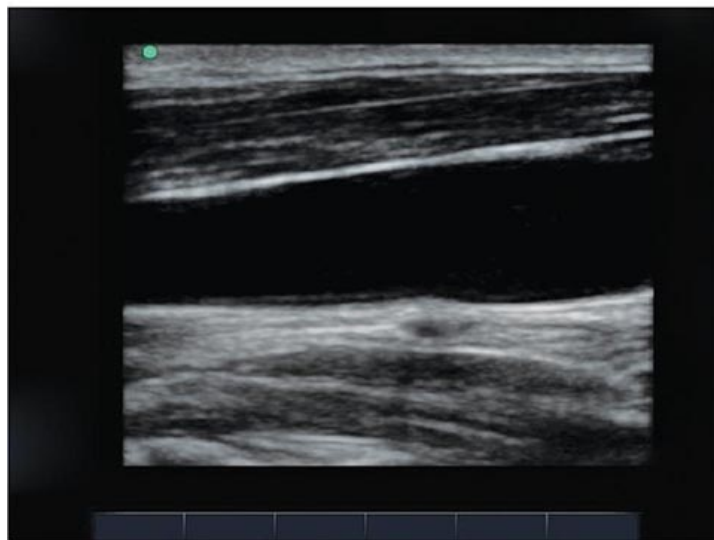


FIGURE 24.101 ■ Internal Jugular Vein Cannulation, Long-Axis Approach. The internal jugular vein is identified in its long axis. Introduce the needle immediately distal to the transducer's edge at approximately a 45-degree angle to the patient's skin and parallel to the long axis of the probe. (Photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Jeremy S. Boyd, MD; illustration contributor: Robinson M. Ferre, MD.)

- Identify the path of the advancing needle as indicated by observing:
 - The needle tip
 - Tissue movement
 - Ring-down artifact from the needle
- Follow the tip of the needle until it contacts the anterior surface of the vein.
- Continue to advance until a venous flash is seen or the needle has reached the intended depth.
- If no flash is seen at the appropriate depth in the correct path, slowly withdraw the needle and observe for venous return.
- Continue catheter placement as per common practice.

Central Venous Access

Special considerations for central access:

- Typical sites include the internal jugular vein ([Figs. 24.100](#) and [24.101](#)) and the femoral vein, but the subclavian/axillary vein can also be accessed with ultrasound guidance ([Fig. 24.102](#)).

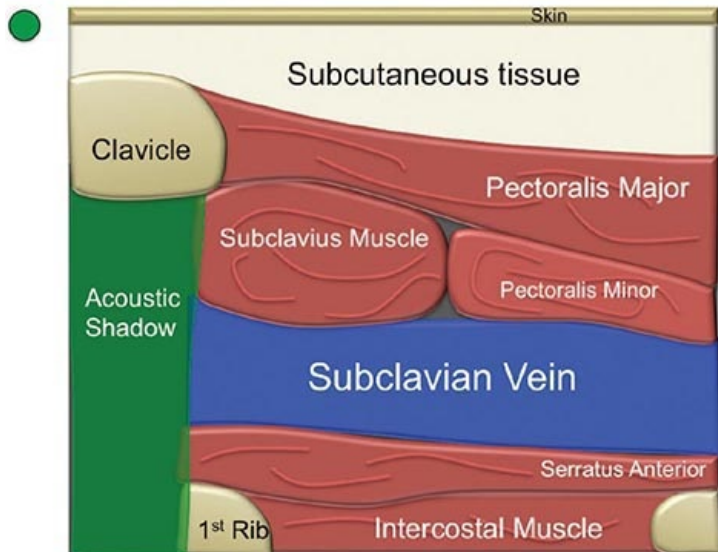
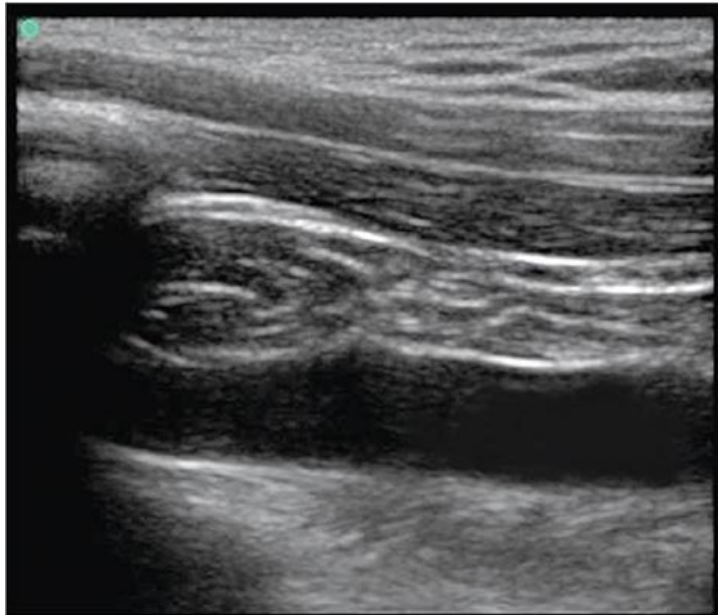


FIGURE 24.102 ■ Subclavian Vein Cannulation, Long-Axis Approach. The subclavian vein is identified in its long axis with the proximal edge of the transducer overlying the clavicle. Introduce the needle immediately distal to the transducer's edge at approximately a 30- to 45-degree angle to the patient's skin and parallel to the long axis of the probe. The needle should be visualized throughout its entire course. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD.)

- Be careful to maintain sterile precautions including a sterile transducer cover.

Peripheral Venous Access

Special considerations for peripheral access:

- Apply a tourniquet if needed.
- Ensure that both the needle tip and the plastic catheter are within the vein before advancing the catheter overlying the needle. This often requires advancing the needle just beyond the depth where an initial flash is obtained.
- The veins of the upper arm (basilic, deep brachial, and cephalic) are good targets for ultrasound-guided peripheral venous access ([Figs. 24.103](#) and [24.104](#)).

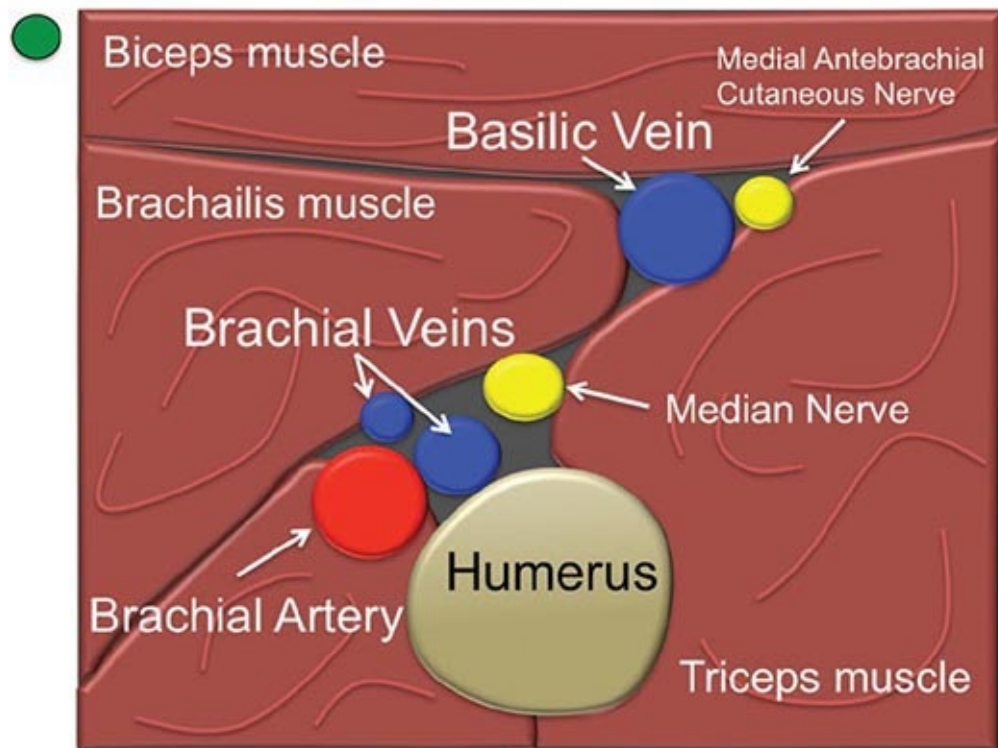
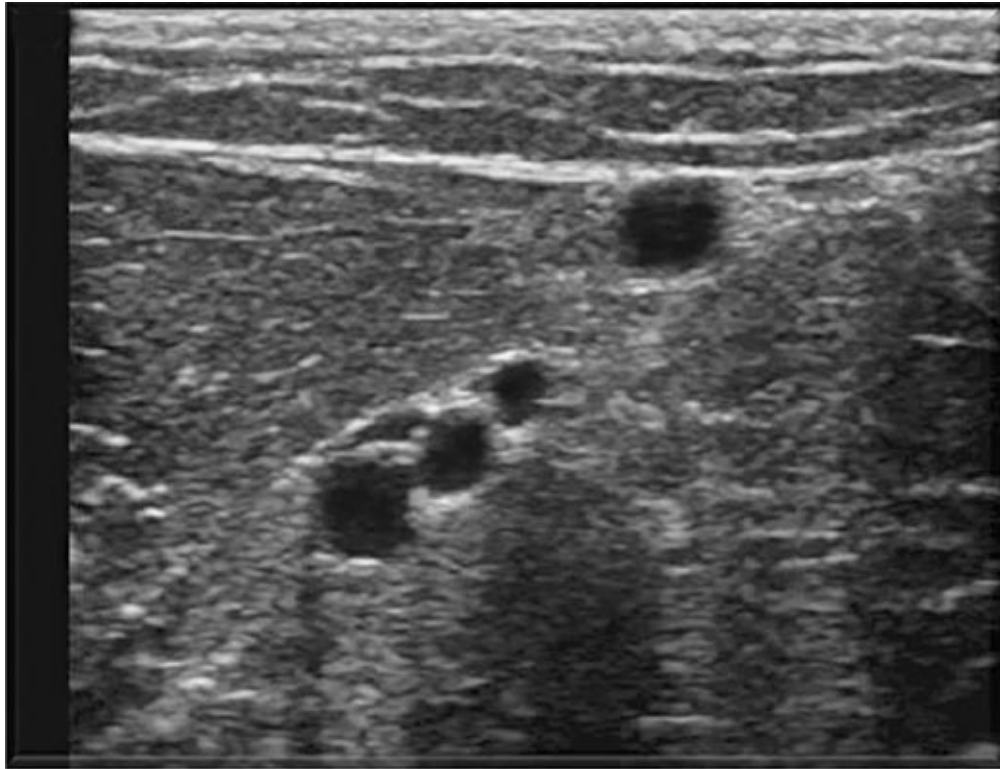


FIGURE 24.103 ■ Deep Brachial Veins. Transverse anatomy of the deep veins of the upper arm. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Steven Downie, MD.)



FIGURE 24.104 ■ Peripheral IV Insertion. Approaches to ultrasound-guided peripheral venous access of the upper extremity: short-axis (**A**), long-axis (**B**), and “backscratcher” approach (**C**). The backscratcher approach facilitates easy access to the more medial basilic vein while allowing improved patient comfort. It is easily performed from the head of the bed. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Ultrasound identifies the relevant anatomy, but care must be used to follow the typical procedures for venous access.
2. When using a sterile sheath (or glove), use conductive media both inside and outside the sheath. The gel outside the sheath should also be sterile.
3. Venous access may be performed by a single provider or with an assistant.
4. Having the patient cough or perform the Valsalva maneuver may distend the internal jugular vein to facilitate venous access.

PROCEDURAL GUIDANCE: ULTRASOUND- GUIDED NERVE BLOCKS

Clinical Summary

Managing pain due to painful conditions or procedures is a core component of emergency care. While landmark-based nerve blocks have traditionally been used, the use of ultrasound at the bedside can enhance the safety and effectiveness of nerve blocks. Patients should be monitored both during and after the procedure to evaluate for any signs of toxicity due to the anesthetic of choice.

Femoral Nerve Block and Fascia Iliaca Compartment Block

Nerve Anatomy

The femoral nerve provides sensory innervation via two branches. The anterior branch of the femoral nerve provides sensation to the anteromedial thigh via two additional branches, the medial cutaneous nerve of the thigh and the intermediate

cutaneous nerve. The posterior branch of the femoral nerve provides sensation to the anteromedial portion of the lower leg and the region overlying the patella via two additional branches, the saphenous nerve and the infrapatellar nerve. These four nerves (two in the anterior branch and two in the posterior branch) are blocked with the femoral nerve block. The fascia iliaca compartment (FIC) block is a planar block that includes the femoral nerve and the lateral femoral cutaneous nerve (LFCN). The LFCN, as its name would suggest, provides sensation to the anterolateral thigh. The FIC block can also affect the obturator, ilioinguinal, accessory obturator, and genitofemoral nerves.

Indications

- Femur fracture
- Proximal tibia fracture
- Analgesia for incision and drainage of leg abscess within affected sensory distribution
- Analgesia for laceration closure on leg within affected sensory distribution

Equipment

- Skin disinfection agent and sterile gloves
- Sterile ultrasound probe cover
- Skin marker
- 27-gauge needle and 10-mL syringe for skin wheal of anesthetic of choice
- 20-gauge Tuohy epidural needle (for block) (or other blunt-tipped needle)
- 6- to 24-inch pressure tubing (flushed with saline and connected to block needle)



FIGURE 24.105 ■ Supplies for Ultrasound-Guided Peripheral Nerve Blocks. Sterile skin disinfectant, sterile saline flushes, short IV tubing, block needles, Luer lock syringes, sterile transducer sheath, and sterile lubricant. Not pictured: local anesthetic. (Photo contributor: Lawrence B. Stack, MD.)

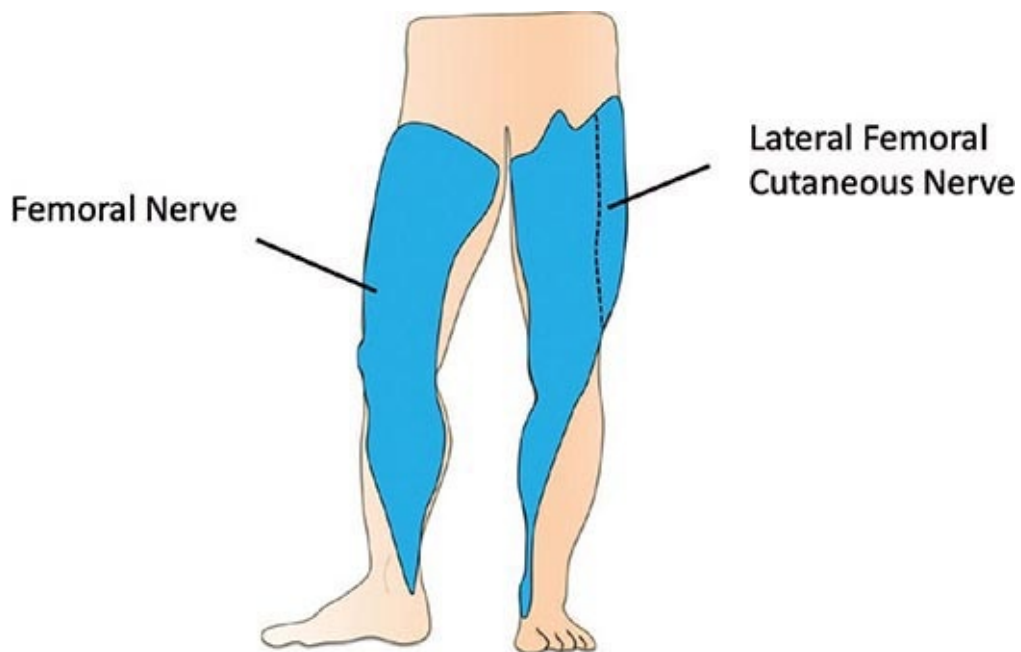


FIGURE 24.106 ■ Sensory Distribution of Fascia Iliaca Compartment Nerve Block. (Illustration

contributor: Robinson M. Ferre, MD.)

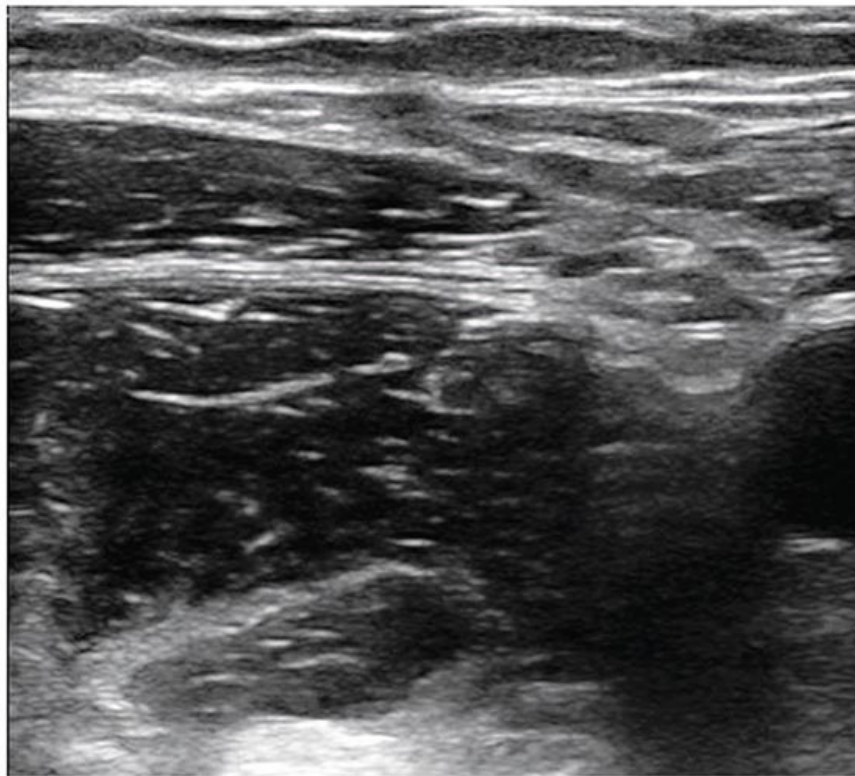
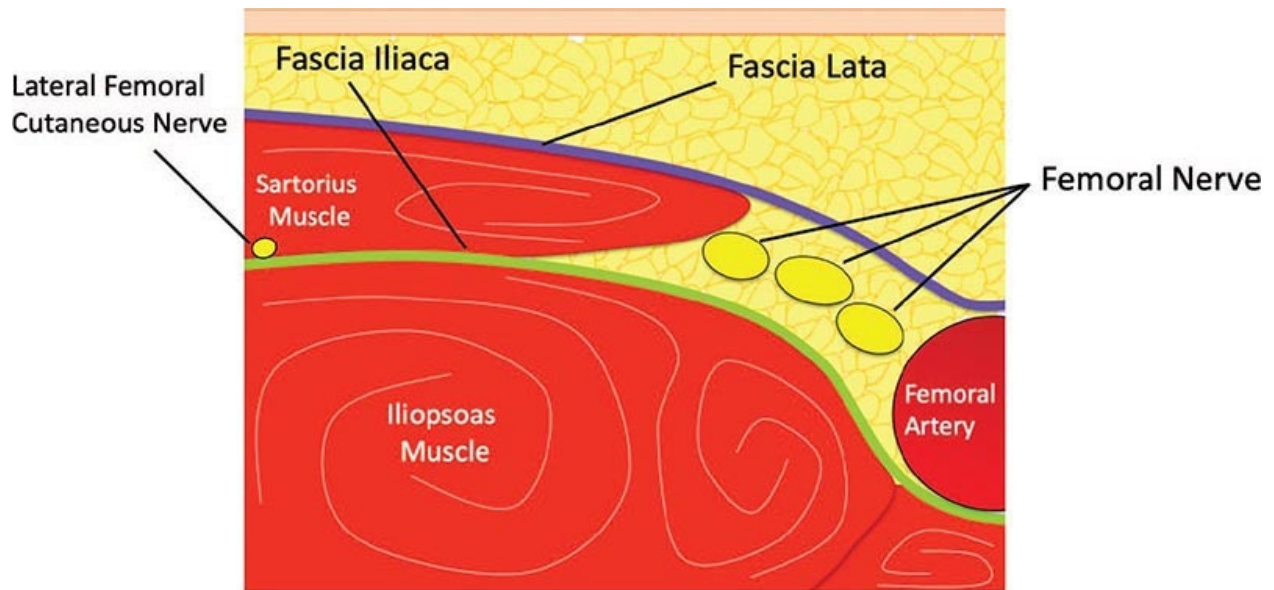


FIGURE 24.107 ■ Femoral Nerve Block Anatomy. (Ultrasound and illustration contributor: Robinson M. Ferre, MD.)

- Saline syringes (10 mL × 2) for hydrodissection
 - Empty syringe (20 mL × 2) for injection of diluted local anesthetic
 - Local anesthetic of choice

Patient Preparation

- Supine and in Trendelenburg if pannus covers inguinal crease (to assist in pannus retraction)

Technique

For FIC block:

- Probe is placed parallel to and distal to the inguinal ligament.
- The femoral artery is identified medially with the femoral nerve just lateral and inferior to the artery.

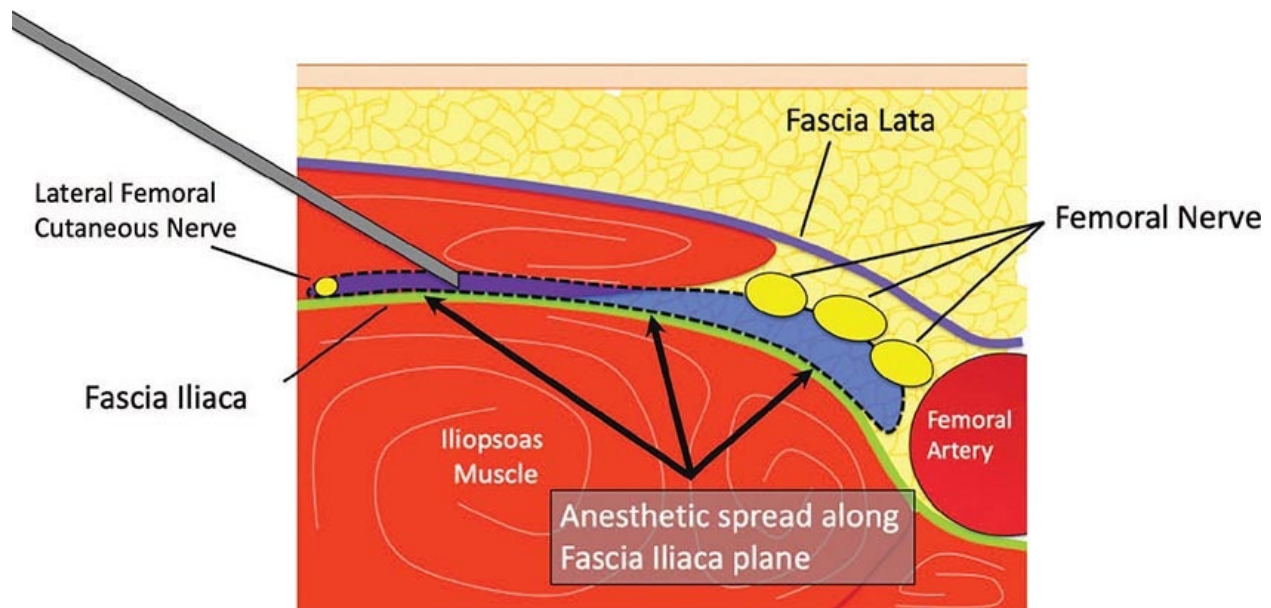


FIGURE 24.108 ■ Fascia Iliac Compartment Nerve Block. In this block, the anesthetic is distributed throughout the fascial plane defined by the fascia iliaca. Note that this distribution includes the lateral femoral cutaneous nerve as well as the femoral nerve. (Illustration contributor: Robinson M. Ferre, MD.)

- The femoral nerve and LFCN run under the fascia iliaca, so the plane to target for anesthetic spread is the potential space between the iliopsoas muscle and the overlying fascia iliaca.
- Mark selected insertion site with pen.
- Skin is disinfected and sterile techniques are applied (sterile gloves, probe cover, etc).
- Skin wheal of local anesthetic is placed at selected site of needle insertion.
- Under ultrasound guidance in long axis, needle is visualized as it penetrates fascia iliaca comfortably lateral to femoral nerve (it is not necessary to be near femoral nerve for this planar block to work).

- Aspirate to ensure no vascular penetration.
- Hydrodissection with sterile saline injected through flushed tubing attached to block needle is used to ensure correct plane has been penetrated.
- Anesthetic is pushed through tubing with continuous visualization of needle in long axis.

For femoral nerve block:

- The same landmarks and approach noted for FIC block are used, but the needle tip should be targeted just lateral to the femoral nerve, being careful not to damage the nerve. The goal is to surround the nerve in anesthetic.

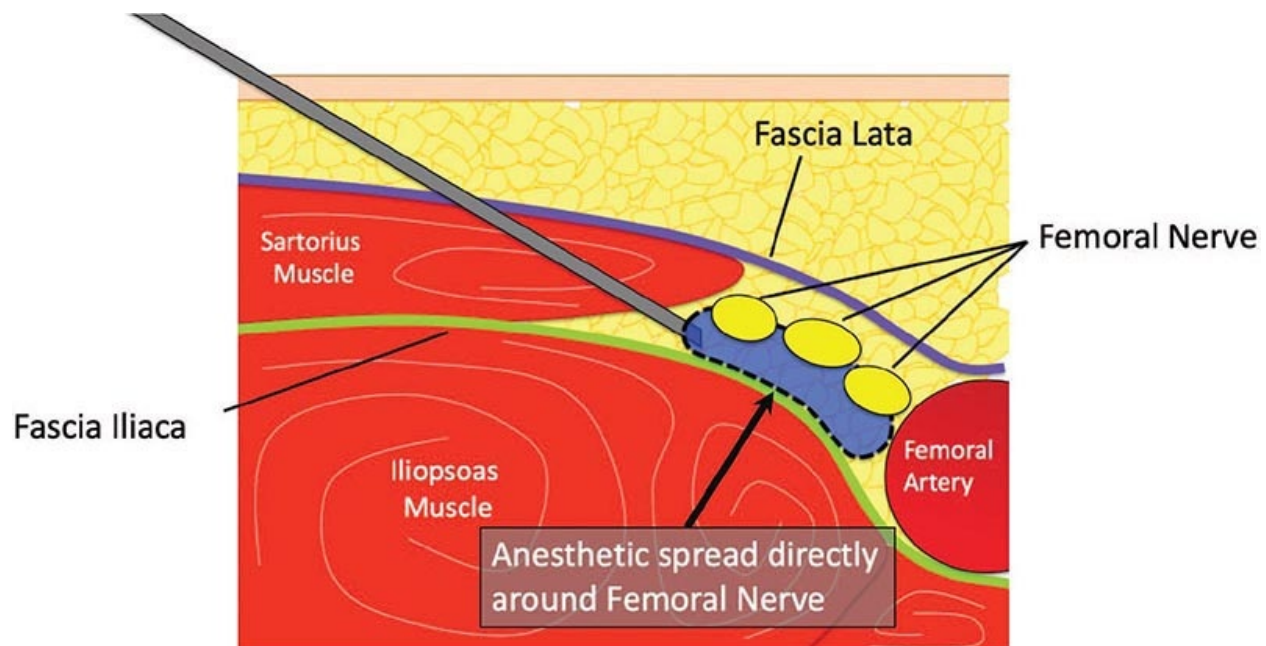


FIGURE 24.109 ■ Femoral Nerve Block. The femoral nerve block distributes anesthetic at the site of the femoral nerve and thus often uses a smaller volume of anesthetic than the fascia iliaca block. (Illustration contributor: Robinson M. Ferre, MD.)

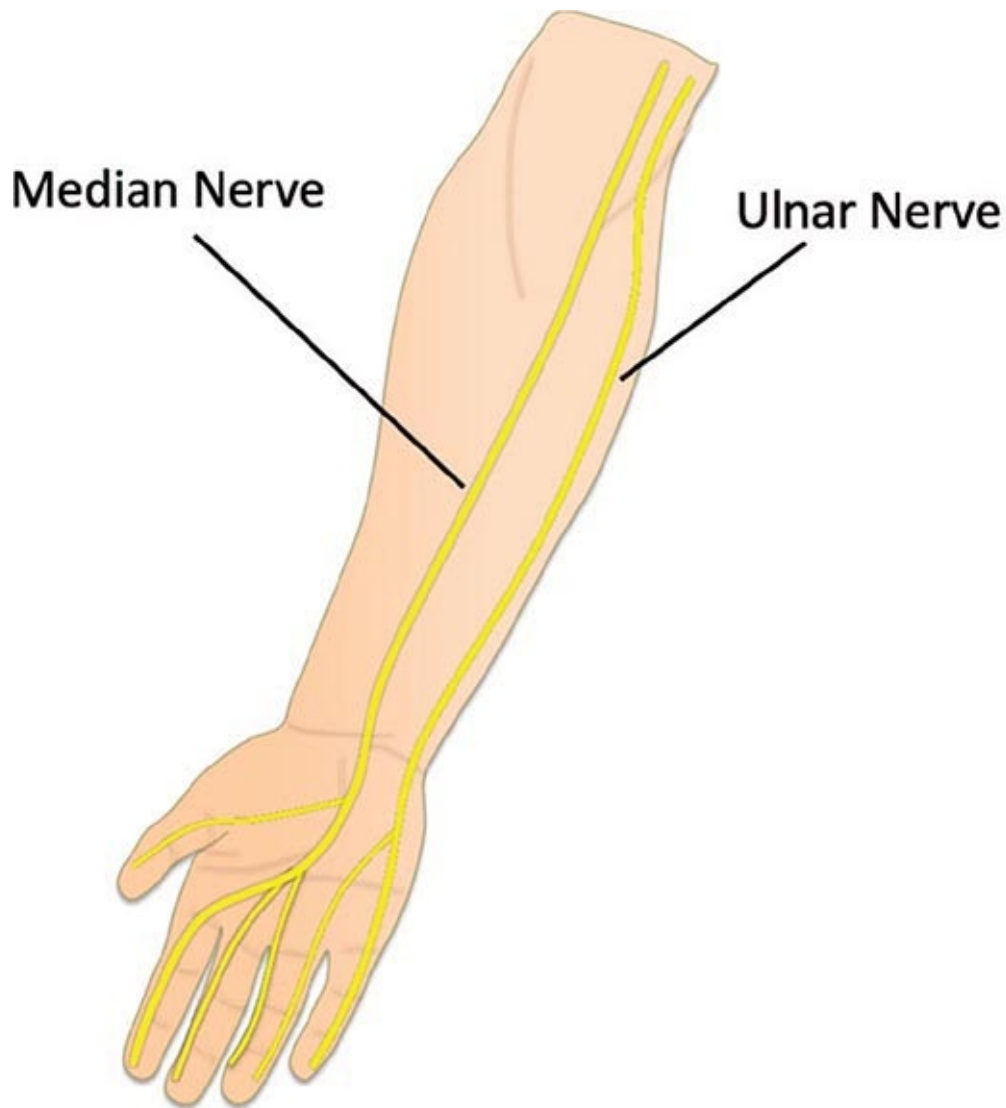


FIGURE 24.110 ■ **Forearm Nerve Anatomy.** (Illustration contributor: Robinson M. Ferre, MD.)

Forearm Blocks

Nerve Anatomy

The median nerve is visible with ultrasound in the midportion of the forearm on the volar surface between the flexor digitorum profundus and flexor digitorum superficialis. The median nerve is also visible with ultrasound on the medial portion of the elbow crease just medial to the brachial artery.

The radial nerve is visualized in the mid-forearm just lateral to the radial artery. The forearm portion of the radial nerve is visualized after the nerve splits into a superficial sensory branch and deep motor branch at the elbow crease.

This superficial sensory branch can be difficult to visualize in the mid-forearm. Thus, the radial nerve is often blocked at its more easily identified region above the elbow crease at the anterolateral portion of the upper arm between the brachialis and brachioradialis muscles.

Finally, the ulnar nerve is visualized in the mid-forearm just medial to the ulnar artery. The ulnar nerve can also be visualized in the posteromedial portion of the elbow proximal to the elbow crease overlying the triceps muscle.

Sensory Innervation of the Hand

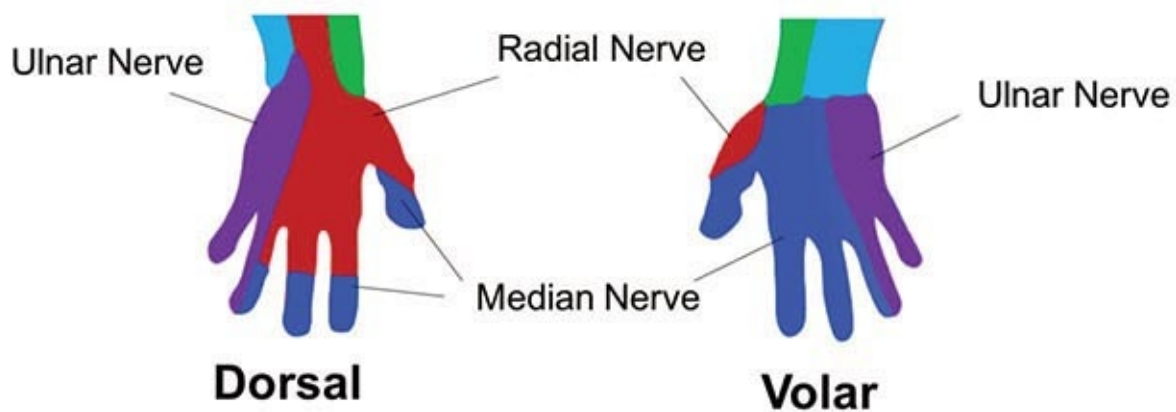


FIGURE 24.111 ■ Sensory Innervation of the Hand for Forearm Nerve Block. (Illustration contributor: Robinson M. Ferre, MD.)



FIGURE 24.112 ■ Median Nerve Block. Example of an approach that might be used for dynamic guidance of a mid-forearm median or radial nerve block. Needle placement for forearm median or radial nerve block with dynamic ultrasound guidance. (Photo contributor: Lawrence B. Stack, MD.)

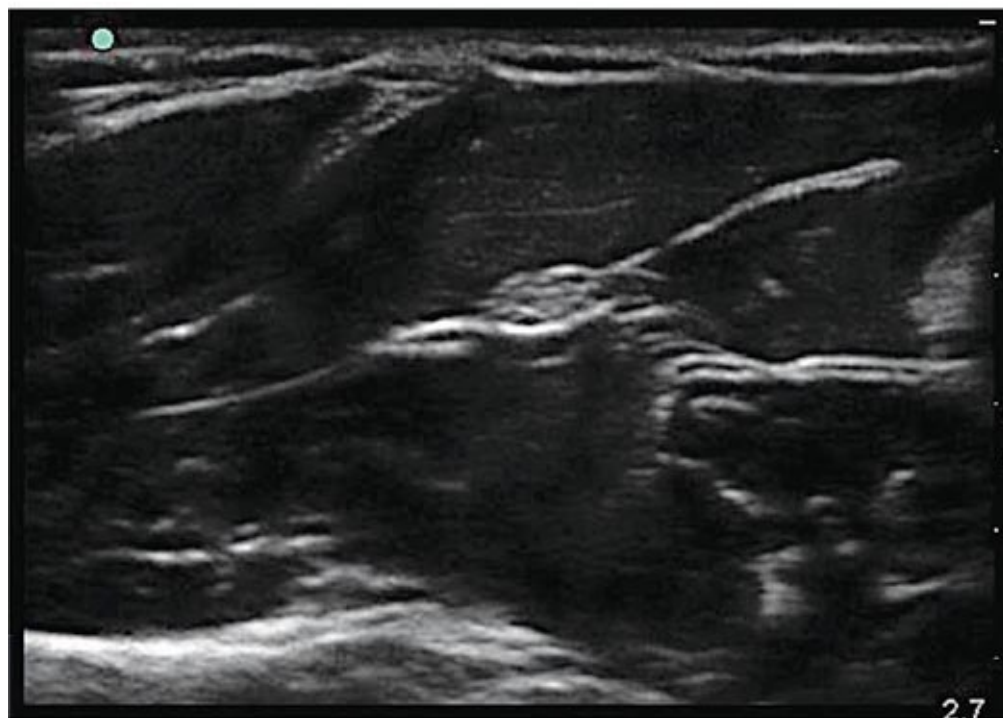
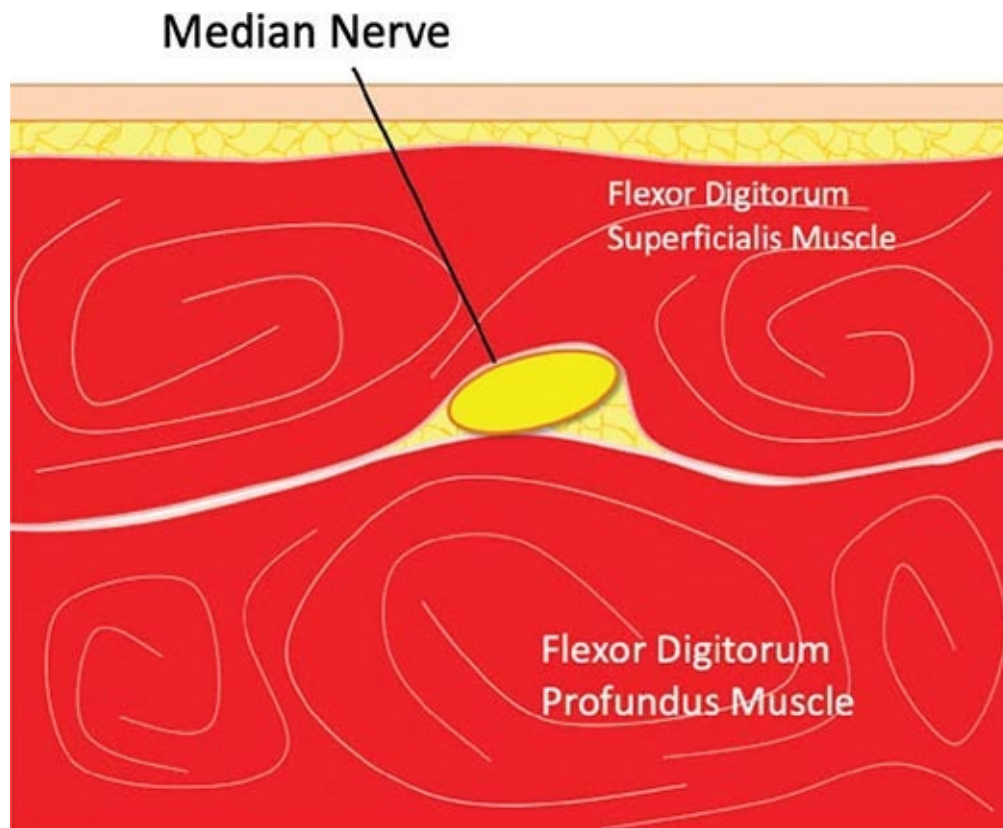


FIGURE 24.113 ■ Median Nerve Anatomy. The median nerve can be identified in the mid-forearm between the muscle bellies of the flexor digitorum superficialis and the flexor digitorum profundus. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Indications

- Hand fractures (does not work well for distal forearm fractures or carpal fractures)
- Analgesia for incision and drainage of hand abscess within affected sensory distribution
- Analgesia for laceration closure on hand within affected sensory distribution

Equipment

- Skin disinfection agent and sterile gloves
- Sterile ultrasound probe cover
- Skin marker
- 27-gauge needle and 10-mL syringe for skin wheal of anesthetic of choice

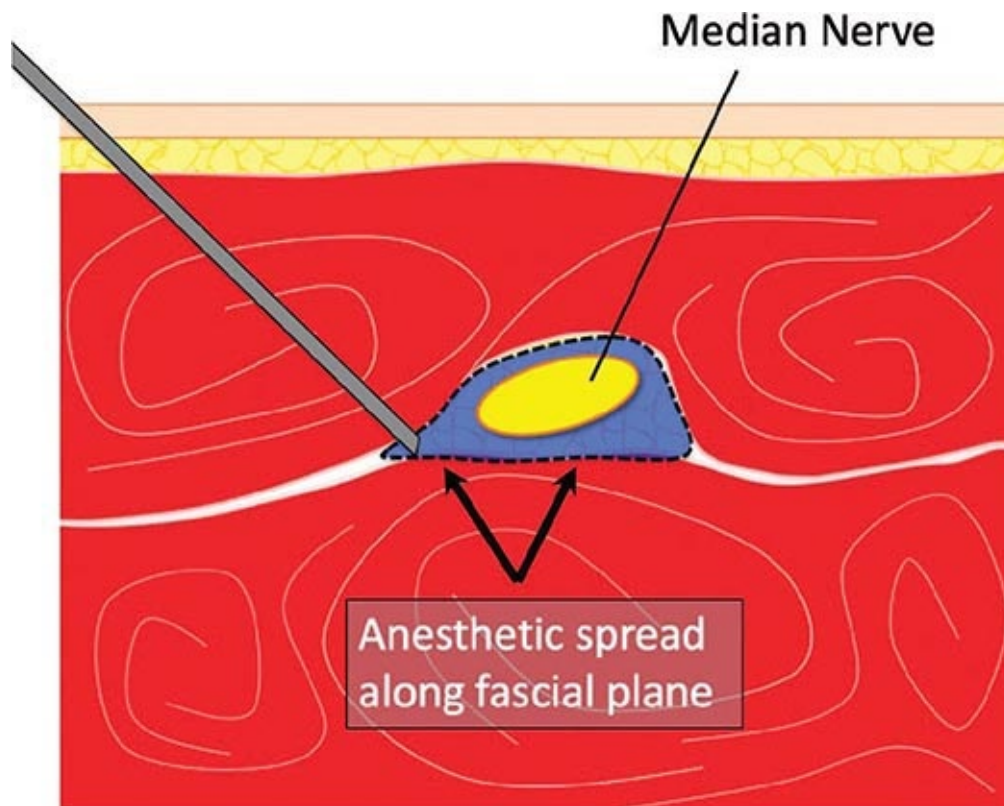


FIGURE 24.114 ■ **Median Nerve Infiltration.** (Illustration contributor: Robinson M. Ferre, MD.)



FIGURE 24.115 ■ Ulnar Nerve Block. Example of needle placement for forearm ulnar nerve block with dynamic ultrasound guidance. (Photo contributor: Lawrence B. Stack, MD.)

- 22-gauge blunt tip block needle or 22-gauge Quincke spinal needle
- Empty syringe (10 mL) for injection of local anesthetic
- Local anesthetic of choice

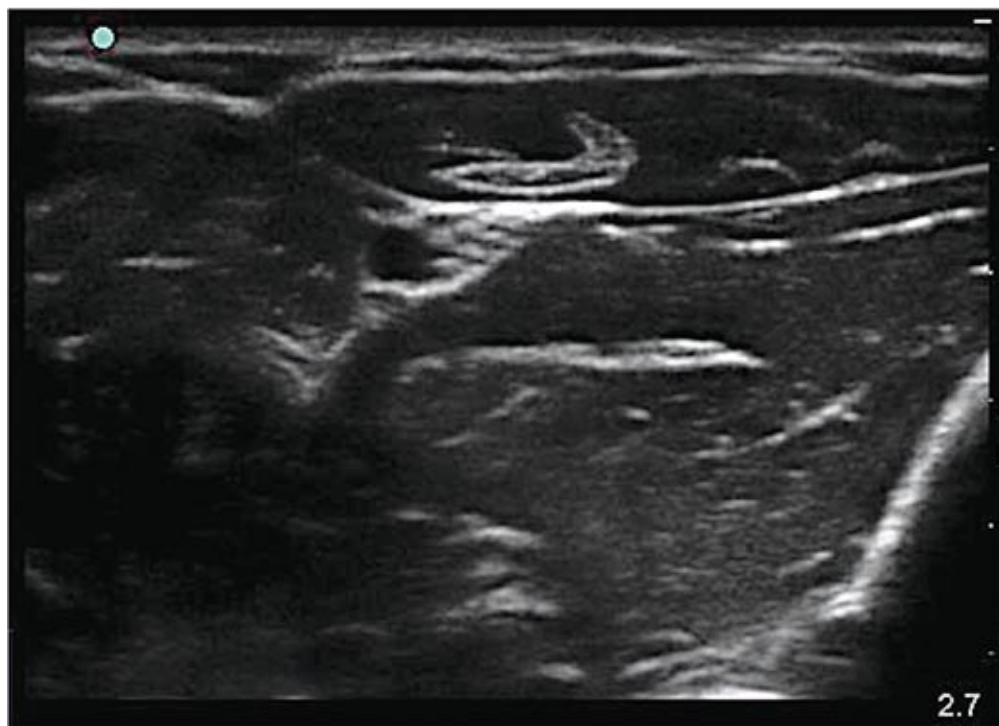
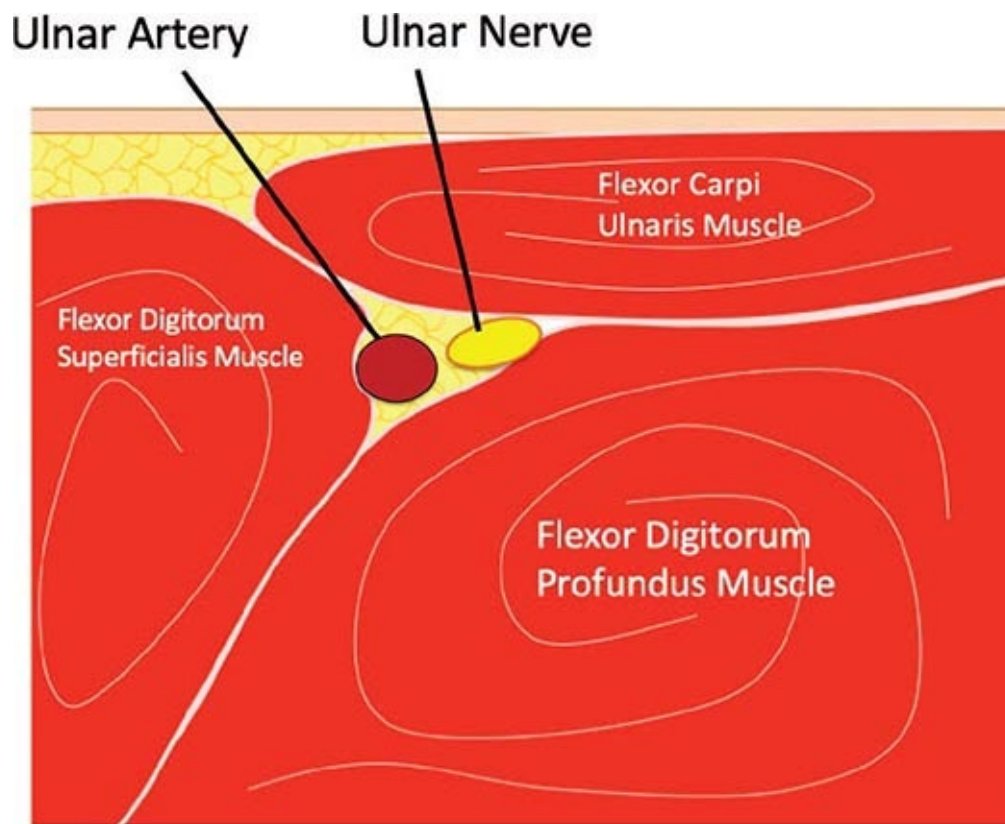


FIGURE 24.116 ■ Ulnar Nerve Anatomy. The ulnar nerve travels with the easily identifiable ulnar artery between the muscles of the flexor carpi ulnaris, flexor digitorum profundus, and flexor digitorum superficialis. (Ultrasound and illustration contributor: Robinson M. Ferre, MD.)

Patient Preparation

- Sitting or supine with arm moderately abducted with volar forearm facing toward ceiling

Technique

Forearm block technique:

- Visualize nerve target as “honeycomb” structure in the regions listed previously under nerve anatomy.
- Disinfect skin, apply sterile equipment (gloves, probe cover, etc).
- Place skin wheal at insertion site with 27-gauge needle and local anesthetic of choice.

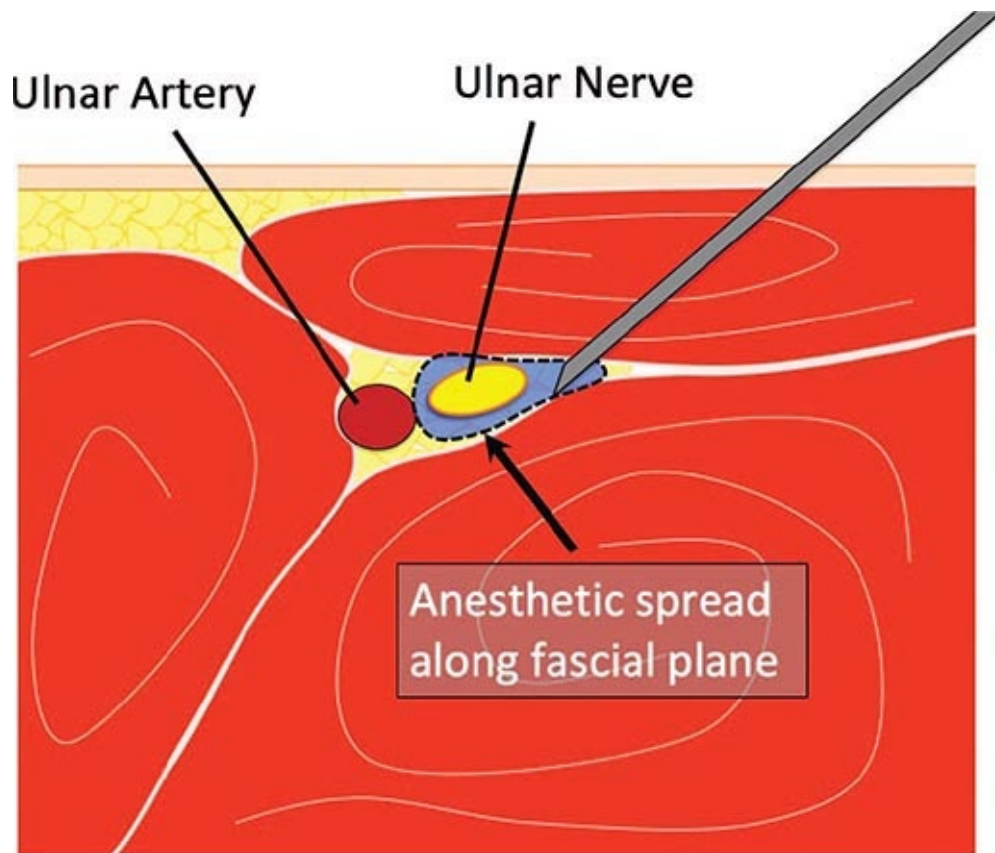


FIGURE 24.117 ■ **Ulnar Nerve Infiltration.** (Illustration contributor: Robinson M. Ferre, MD.)

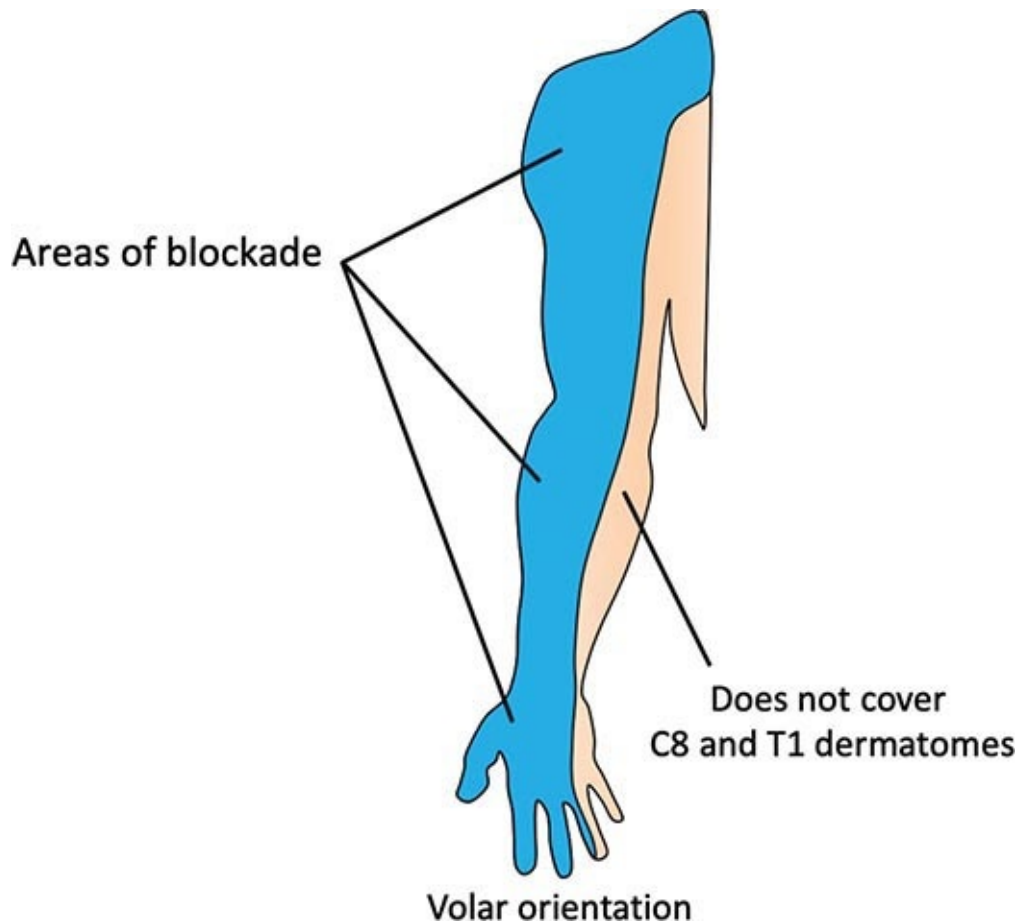


FIGURE 24.118 ■ Interscalene Sensory Distribution of Inter-scalene Brachial Plexus Nerve Block.

Note that the more proximal areas in this distribution will be most affected with smaller volumes of anesthetic. (Illustration contributor: Robinson M. Ferre, MD.)

- Under ultrasound guidance using long- or short-axis approach, target block needle tip toward nerve with goal of infiltrating fascial sheath surrounding nerve.
- Aspirate to ensure no vascular penetration.
- Inject 2 to 5 mL of anesthetic of choice.
- NOTE: It is not necessary to completely surround the nerve with anesthetic to obtain an effective block.



FIGURE 24.119 ■ Interscalene Nerve Block. Example of needle placement for interscalene nerve block with dynamic ultrasound guidance. (Photo contributor: Lawrence B. Stack, MD.)

Brachial Plexus

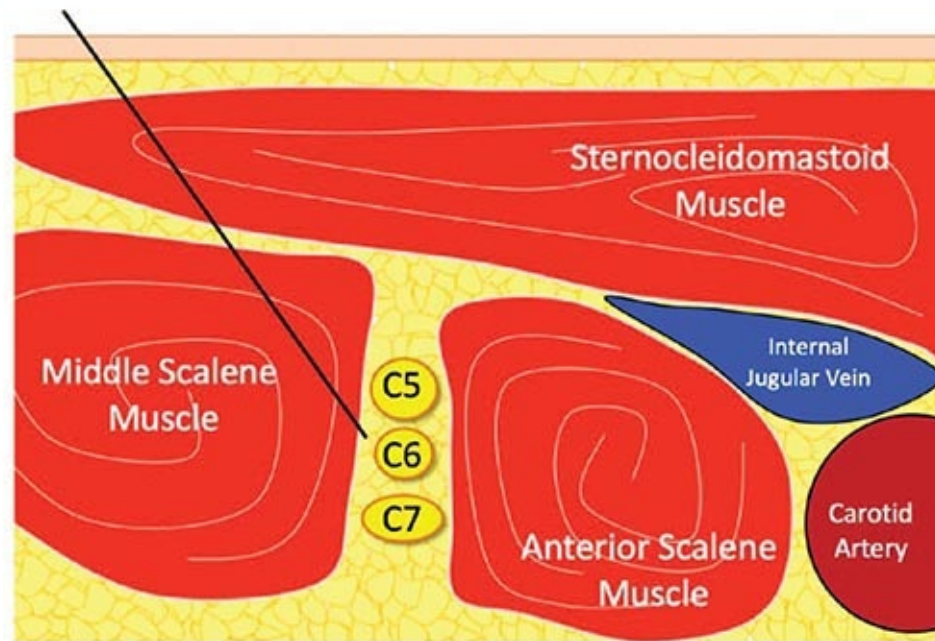


FIGURE 24.120 ■ Interscalene Nerve Anatomy. The brachial plexus is found between the scalene muscles and often has the appearance of a “stoplight” made by the three hypoechoic roots/trunks. (Illustration and ultrasound contributor: Robinson M. Ferre, MD.)

Interscalene Nerve Block

Nerve Anatomy

The brachial plexus lies between the anterior and middle scalene muscles at the level of the cricoid cartilage. Medially, the carotid artery and internal jugular veins are visible. Blocking the brachial plexus reliably provides anesthesia at C5-C7, which are the more superficial nerve roots. Some blocks will include C8-T1, especially with larger volumes of anesthetic. Some nearby nerves that should be avoided include the phrenic nerve, which usually runs superficial to the anterior scalene muscle, and the long thoracic nerve and dorsal scapular nerve, which run with the middle scalene muscle. Color Doppler should be used to identify any vascular structures in planned path of needle. The vertebral artery and other significant vascular structures can be avoided in this way.

Indications

- Analgesia for incision and drainage of shoulder or arm abscess within affected sensory distribution
- Analgesia for laceration closure on shoulder or arm within affected sensory distribution
- Shoulder dislocation
- Proximal humerus fracture

Equipment

- Skin disinfection agent and sterile gloves
- Sterile ultrasound probe cover
- Skin marker
- 27-gauge needle and 10-mL syringe for skin wheal of anesthetic of choice
- 22-gauge blunt tip block needle

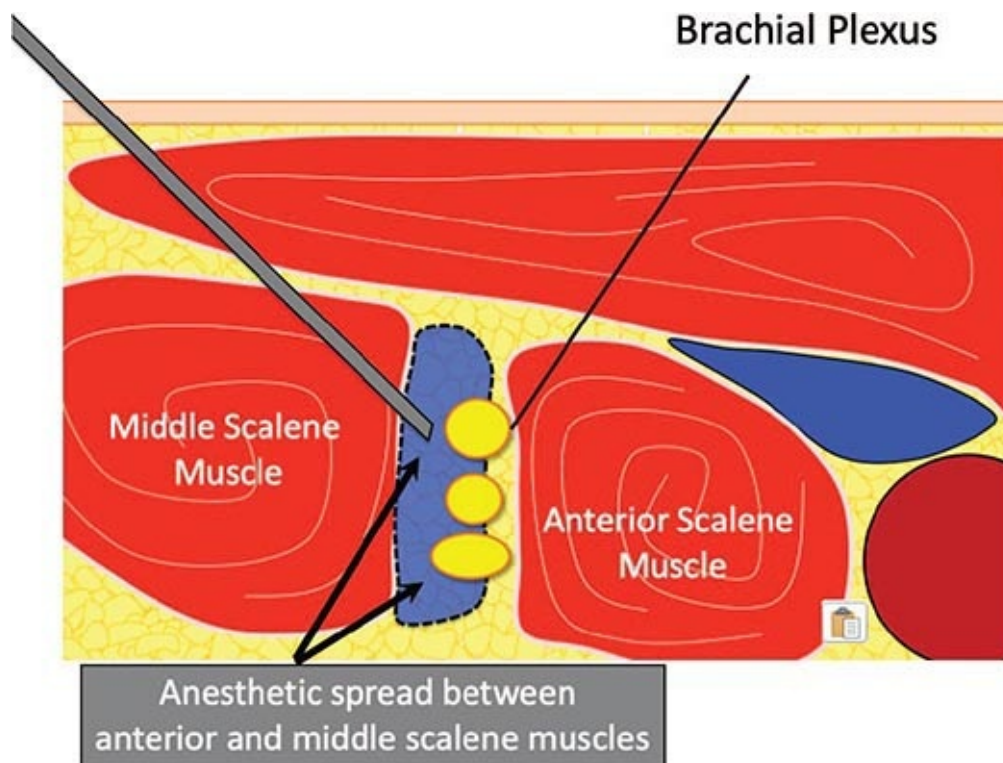


FIGURE 24.121 ■ Interscalene Nerve Infiltration. (Illustration contributor: Robinson M. Ferre, MD.)

- Empty syringe (10 mL) for injection of local anesthetic
- Local anesthetic of choice

Patient Preparation

- Sitting or supine with face turned away from side that is being blocked

Technique

- Visualize brachial plexus at level of cricoid cartilage between the anterior and middle scalene muscles.
- Use color Doppler to evaluate for any vascular structures in path of needle.
- Mark selected insertion site.
- Disinfect skin, and apply sterile equipment (gloves, probe cover, etc).
- Place skin wheal at insertion site with 27-gauge needle and local anesthetic of choice.
- Under ultrasound guidance in long axis, target block needle tip from lateral to medial toward the area between the nerve roots being certain not to injure the nerve roots.
- Aspirate to ensure no vascular penetration.

- Inject 1 to 2 mL of anesthetic to ensure proper location of block; anesthetic should spread around nerve roots; if high pressure is felt in syringe, stop and reposition (this indicates intrafascicular injection).
- If 1st aliquot is appropriately positioned, continue until 5 to 10 mL of anesthetic have been injected and then remove needle.
- NOTE: The phrenic nerve is often blocked as well during the procedure. As a result, this procedure must be avoided in any patient with poor respiratory function who could not tolerate blockade of unilateral phrenic nerve function.

PROCEDURAL GUIDANCE: THORACENTESIS/PARACENTESIS

Clinical Summary

Ultrasound is a source of valuable information that can improve the safety of a wide variety of procedures. The same principles of needle localization and guidance apply. Thoracentesis and paracentesis may be performed using these dynamic “real-time” needle imaging techniques. Alternatively, a static approach may be employed by identifying the location of pathologic fluid and a site for needle insertion. The procedure is then performed in the usual fashion without use of the transducer.

Indications

- Need for thoracentesis or paracentesis.
- Pleural effusion or ascites

Equipment

- Convex
- Linear

Patient Preparation

- For paracentesis, supine position or supine with the head of bed slightly elevated.

- For thoracentesis, the seated upright position is preferred. Lateral decubitus positioning or recumbent position with the ipsilateral arm raised behind the head may also be used.

Technique

Paracentesis

- After positioning patient, ultrasound the abdomen to identify the largest and safest pocket of ascites ([Fig. 24.122](#)). The patient may be repositioned to further improve the target.

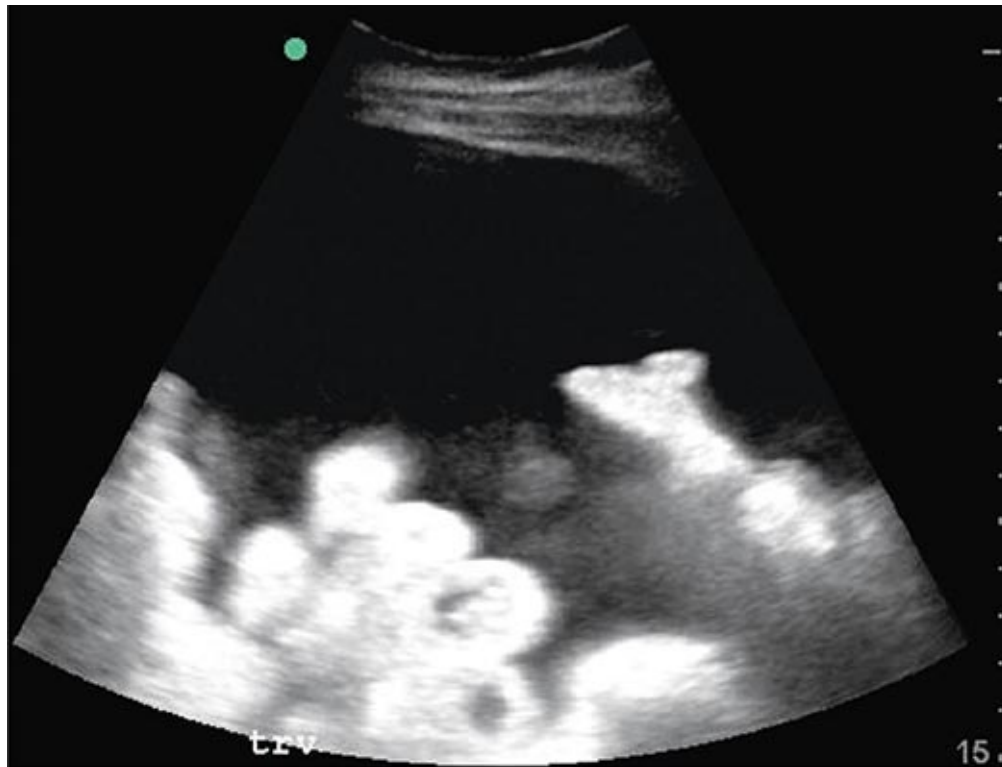


FIGURE 24.122 ■ Paracentesis: Abdomen, Transverse. Inferior abdomen with significant ascites. (Ultrasound contributor: Eric Wu, MD.)

- After optimizing the image and the patient's positioning, the proceduralist may ultrasound throughout the paracentesis (dynamic technique) or mark the ideal target on the patient and then move forward with the procedure in the usual fashion (static technique).
- The same sterile techniques should be applied with use of ultrasound as per common practice including a sterile probe cover and sterile gel if dynamic

imaging is used.

- Continue the procedure as per common practice.

Thoracentesis

- After positioning patient, ultrasound the pleural space posteriorly to identify the largest and safest pocket of pleural fluid ([Fig. 24.123](#)). The transducer should be held in the long or sagittal plane with the indicator aimed superiorly. The patient may be repositioned to further improve access.

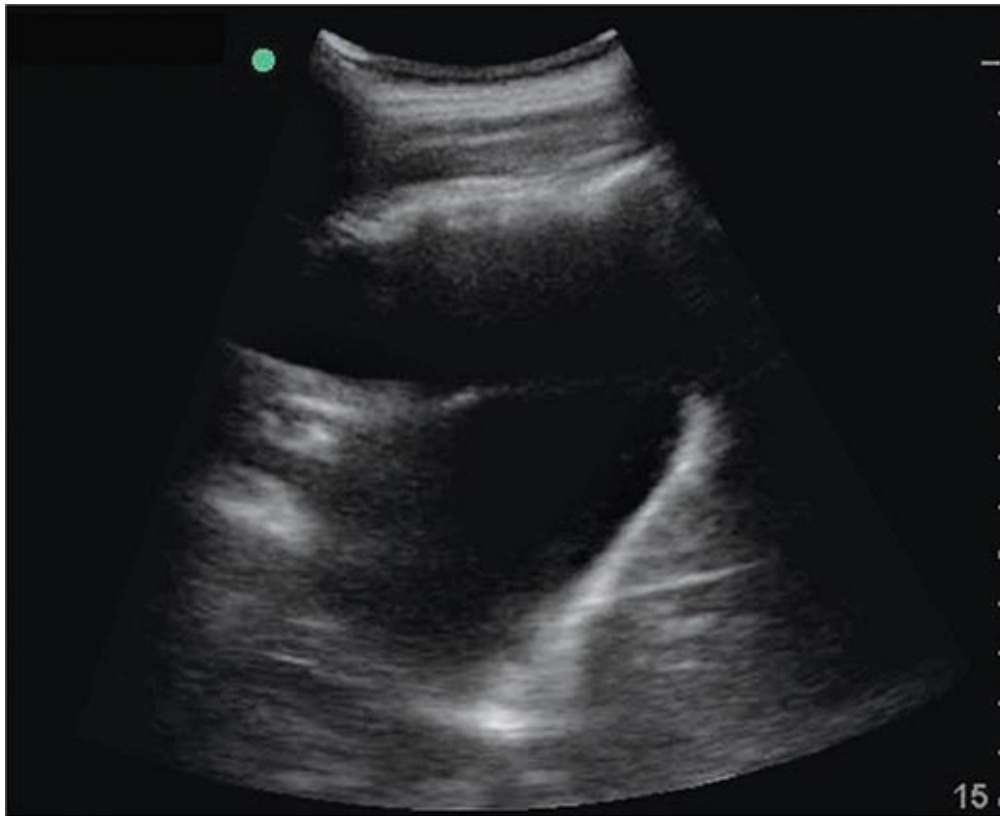


FIGURE 24.123 ■ Thoracentesis: Right Thorax, Sagittal View. Large pleural effusion identified prior to being drained via thoracentesis. (Ultrasound contributor: Craig Sheedy, MD.)

- After optimizing the image and the patient's positioning, the proceduralist may ultrasound throughout the paracentesis (dynamic technique) or mark the ideal target on the patient and then move forward with the procedure in the usual fashion (static technique).
- When choosing a procedure site using a static technique, locate a site that would be safe throughout the respiratory cycle and avoid diaphragmatic injury. The diaphragm and subdiaphragmatic contents can be easily visualized by ultra-sound during normal respiration.

- The same sterile techniques should be applied with use of ultrasound as per common practice including a sterile probe cover and sterile gel if dynamic imaging is used.
- Continue the procedure as per common practice.

Pearls

1. When performing procedures with dynamic ultrasound guidance, keeping the needle tip in view is crucial to procedure safety.
2. Ultrasound identifies the relevant anatomy, but care must be used to follow the typical procedures for paracentesis/thoracentesis (eg, inserting needle immediately superior to the rib to avoid the intercostal neurovascular bundle).
3. Color flow Doppler imaging should be used to identify abdominal or thoracic wall vessels and prevent vascular injury.

OCULAR ULTRASOUND

Clinical Summary

Emergency physician–performed ocular ultrasound is a rapid and reliable way to evaluate the anterior and posterior chambers of the eye for a variety of emergent conditions, including lens dislocation, intraocular foreign body, and retinal and posterior vitreous detachment. It is particularly useful in the setting of ocular trauma when a direct fundoscopic examination is needed, but difficult or impossible to obtain.

Indications

- Visual loss
- Ocular trauma

Equipment: Recommended Transducers for Ocular Ultrasound

- High-frequency linear array

- It is important to select the “ophthalmic exam setting” or confirm and then adjust the energy output levels of your ultrasound system that are approved for ocular ultrasound.

Patient Positioning

- With the patient lying supine, place a liberal amount of gel on top of the patient’s closed eyelid. The orientation marker should be directed toward the patient’s right for transverse images and cephalad for sagittal images.
- The probe should exert little to no pressure on the patient’s eye. This is accomplished by stabilizing your fingers on the bridge of the nose (when imaging the right eye) or the zygomatic arch (for the left eye) ([Fig. 24.124A,B](#)).

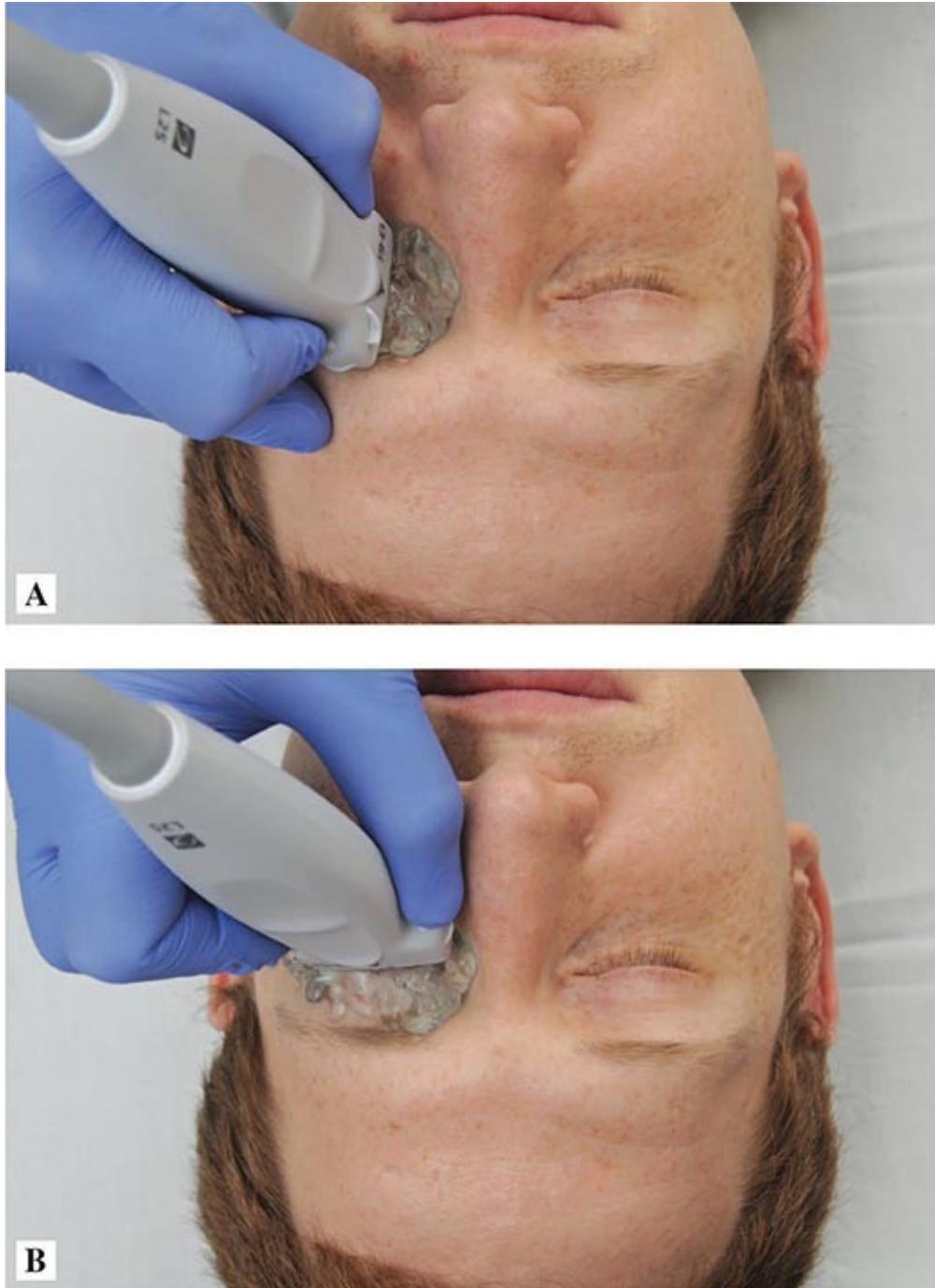


FIGURE 24.124A, B ■ Ocular Ultrasound. Place a liberal amount of gel on top of the patient's closed eyelid. The orientation marker should be directed toward the patient's right for transverse images and cephalad for sagittal images. The probe should exert little to no pressure on the patient's eye. (Photo contributor: Lawrence B. Stack, MD.)

Techniques

Transverse and Sagittal Views

- With the indicator pointing toward the patient's right, identify the anterior chamber, lens, posterior chamber, retina, optic nerve, and retrobulbar space ([Fig. 24.125](#)).

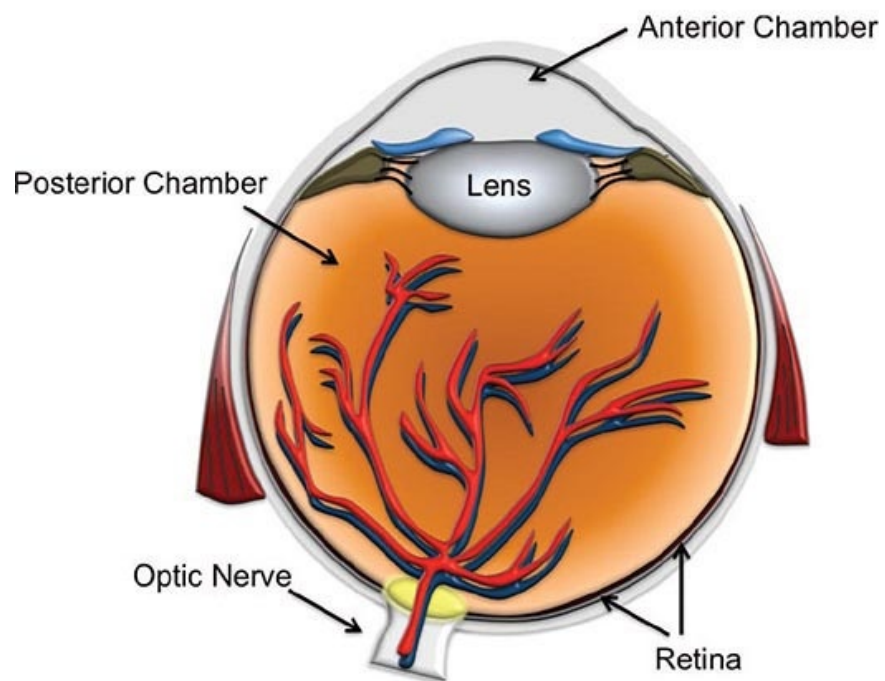


FIGURE 24.125 ■ Ocular Ultrasound. Normal eye anatomy. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

- The probe should be fanned back and forth to ensure that the entire retina is visualized.
- The overall gain should be increased to assess for evidence of hemorrhage or vitreous detachment in the posterior chamber.
- If assessing for elevated intracranial pressure, the optic nerve sheath diameter (ONSD) should be measured 3 mm from the retina ([Fig. 24.126](#)). If measuring the ONSD, it is essential to ensure that the plane of the ultrasound beam is not tangential to the optic nerve.

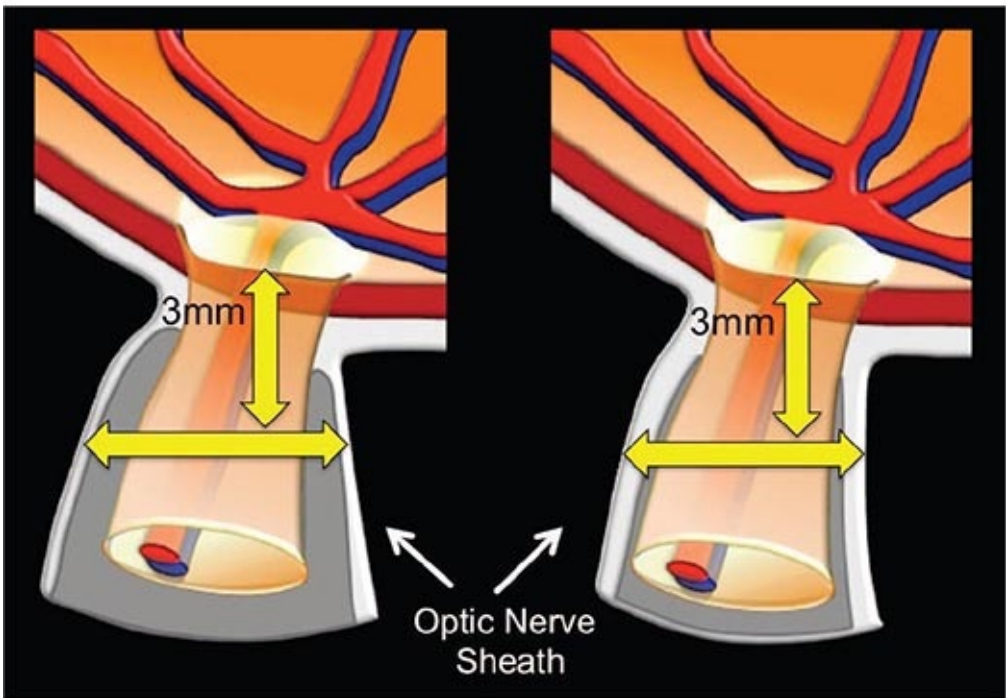
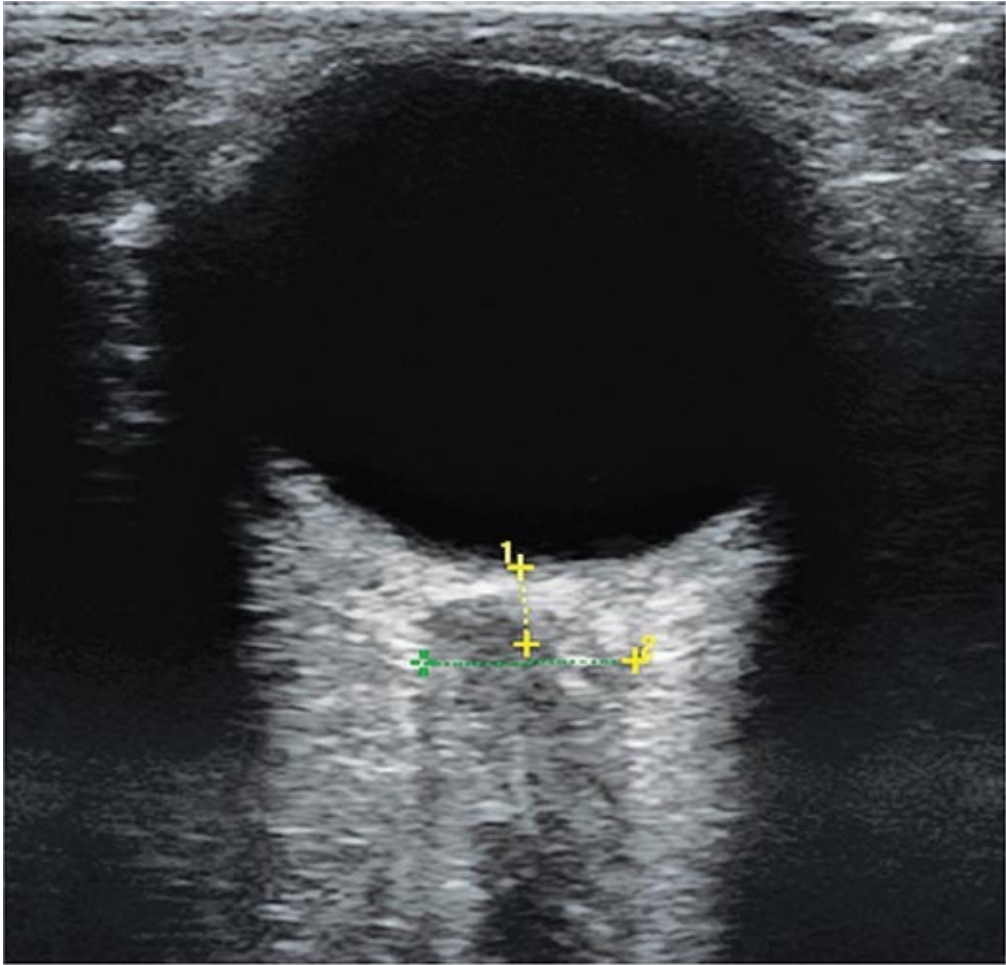


FIGURE 24.126 ■ Ocular Ultrasound. The ONSD is measured 3 mm from the retina. The diagram shows representations of a dilated ONSD (left) next to a normal one (right). (Ultrasound contributor Robinson M. Ferre, MD; illustration contributor: Robinson M. Ferre, MD.)

- The eye should be imaged in both the sagittal and transverse planes to increase the accuracy of detecting pathology, particularly retinal detachment.

Abnormal Findings

- Retinal detachment: A retinal detachment is the separation of the neurosensory retina from the retinal pigment epithelium. The neurosensory retina is a dense neurovascular structure that is highly reflective of ultrasound and will appear as a bright echogenic line on top of the remaining choroid and retinal pigment epithelium ([Fig. 24.127](#)). An acute retinal detachment will appear to “wave” if the patient is asked to move their eyes side to side. An older retinal detachment is more rigid and will often lack this mobility.

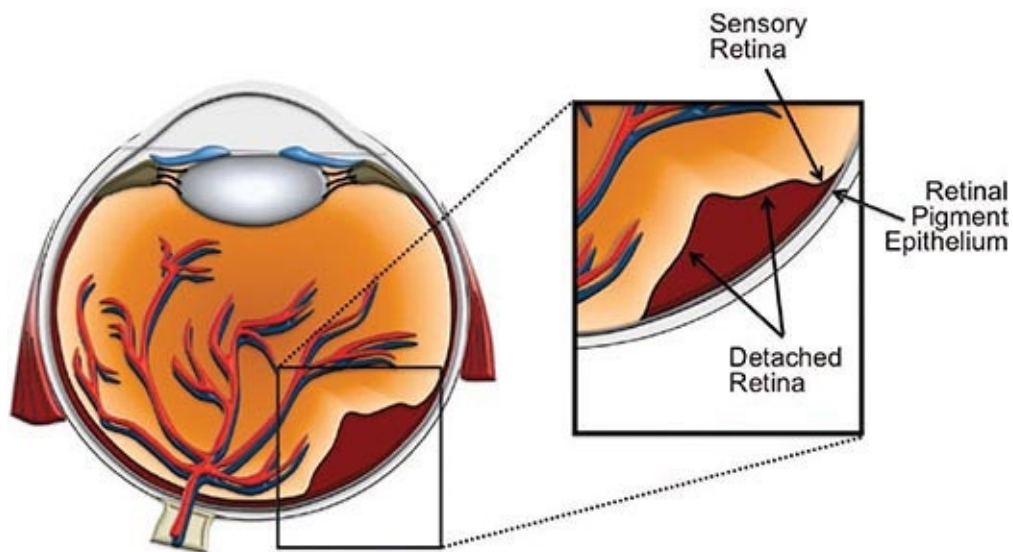


FIGURE 24.127 ■ Ocular Ultrasound: Retinal Detachment. A retinal detachment is the separation of the neurosensory retina from the retinal pigment epithelium. (Ultrasound contributor: Aaron Lacy, MD; illustration contributor: Robinson M. Ferre, MD.)

- **Vitreous detachment:** The normally anechoic vitreous will demonstrate various echoes that are usually only seen with increasing the gain. These small, filament-like threads of the detached vitreous body will “swirl” around with eye movements ([Fig. 24.128](#)).

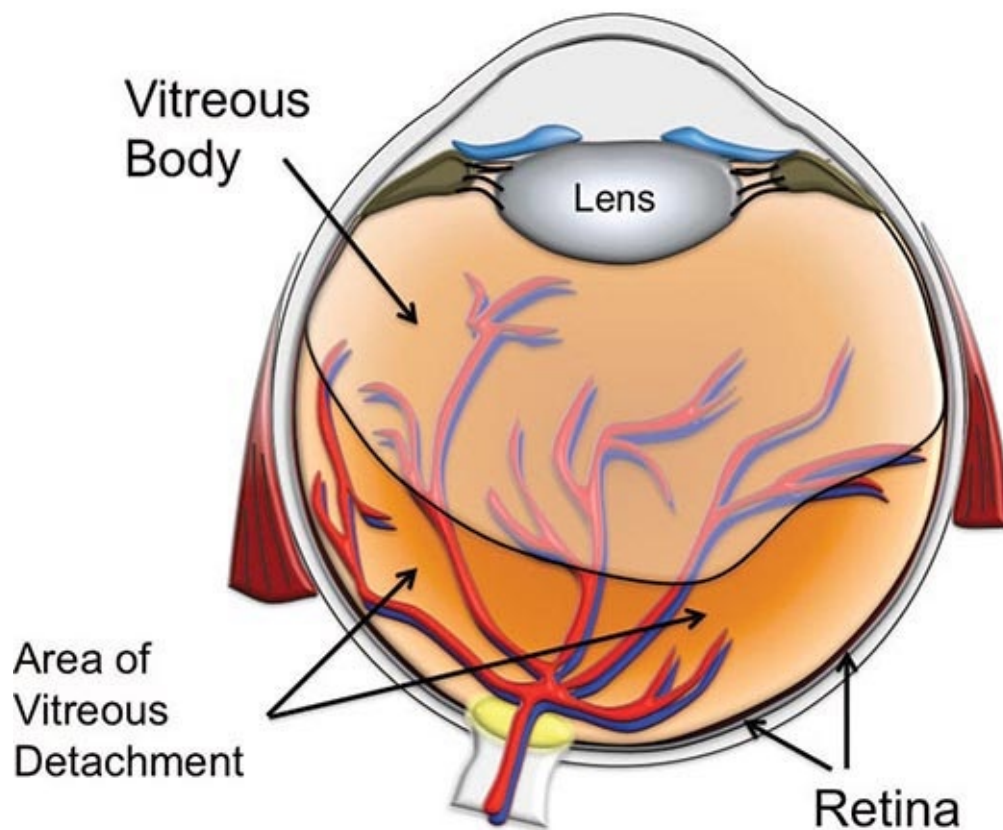
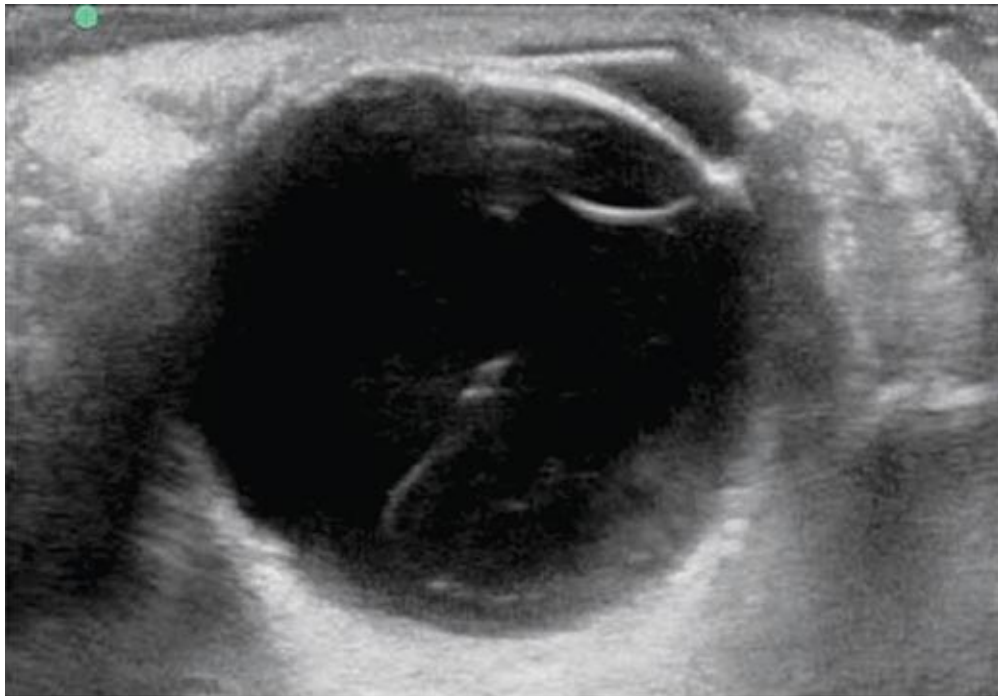


FIGURE 24.128 ■ Ocular Ultrasound: Vitreous Detachment. The normally anechoic vitreous will demonstrate various echoes that are usually only seen with increasing the gain. (Ultrasound contributor: Robinson M. Ferre, MD; illustration contributor: Robinson M. Ferre, MD.)

- Vitreous hemorrhage: Hyperechoic areas in the vitreous or anterior chamber may represent hemorrhage. It is usually necessary to increase the gain to see posterior vitreous hemorrhage ([Fig. 24.129](#)).

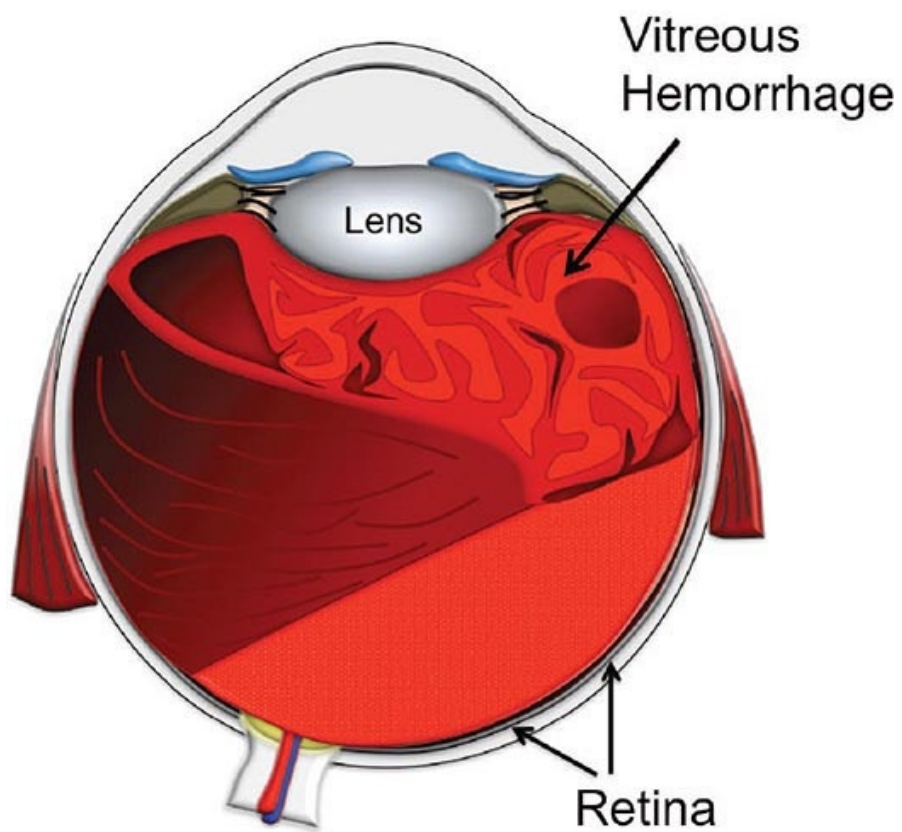


FIGURE 24.129 ■ Ocular Ultrasound: Vitreous Hemorrhage. Hyperechoic areas in the vitreous or anterior chamber may represent hemorrhage. It is usually necessary to increase the gain to see posterior vitreous hemorrhage. (Ultrasound contributor: Robinson M. Ferre, MD; illustration contributor: Robinson M. Ferre, MD.)

- **Dilated optic nerve sheath:** As intracranial pressure increases, this pressure is transmitted to spinal fluid and along the optic nerve sheath. An ONSD greater than 4.5 mm in children and 5 mm in adults is a sensitive, but not specific, finding for elevated intracranial pressure. The average of three repeated measurements increases accuracy.
- **Dislocated lens:** The lens is normally positioned in a central location, immediately under the iris. The anterior convex portion of the normal lens is not seen with ultrasound, but the posterior concave portion is easily seen. A dislocated lens will be absent from its normal position, and a diligent search will reveal its location somewhere in the posterior chamber (Fig. 24.130). A lens subluxation is much more subtle and is visualized as instability of the lens with eye movement.

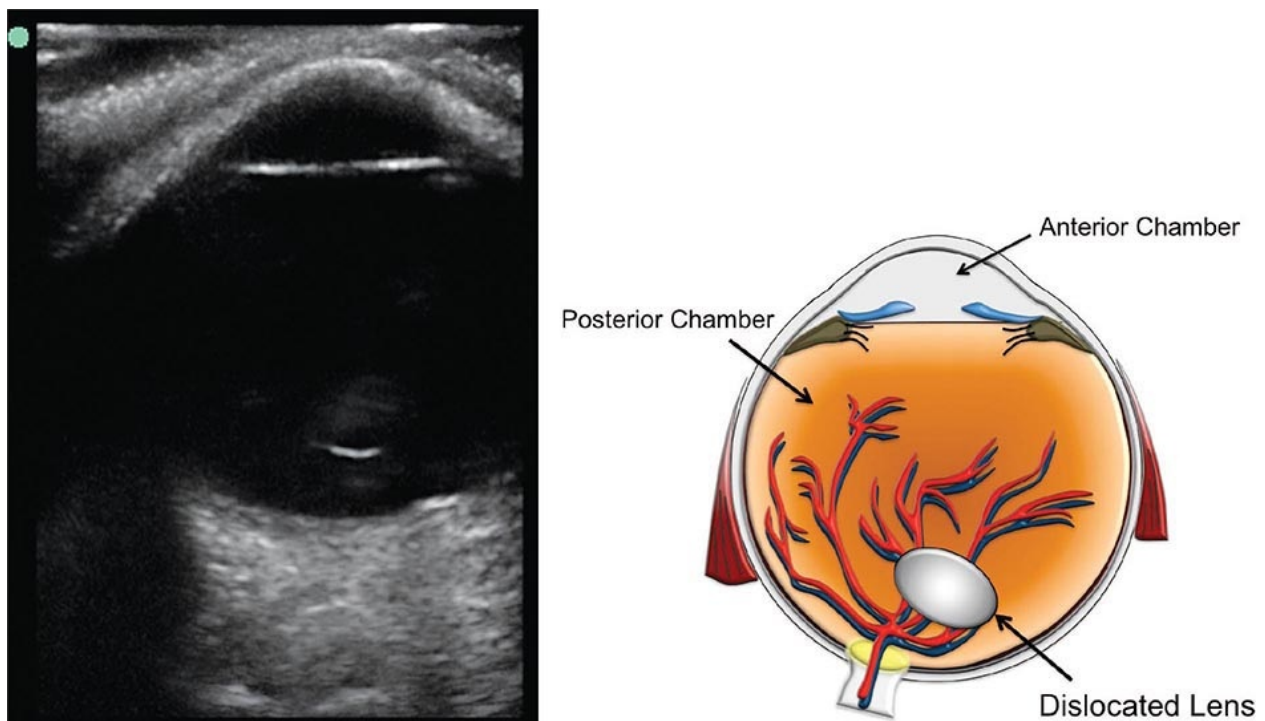


FIGURE 24.130 ■ Ocular Ultrasound: Lens Dislocation. A dislocated lens will be absent from its normal position and a diligent search will reveal its location somewhere in the posterior chamber. (Ultrasound contributor: Robinson M. Ferre, MD; illustration contributor: Robinson M. Ferre, MD.)

Pearls

1. Confirmation of an abnormality can often be made by using the unaffected eye as the control. Using multiple planes to image a structure will improve accuracy.
2. Ocular ultrasound often necessitates increasing gain settings to visualize pathologic findings, particularly in the posterior chamber. Overgain the image, then gradually decrease the gain. This will allow small areas of vitreous hemorrhage or the lacy appearance of a vitreous detachment to be readily identified.
3. Both retinal and vitreous detachments are tethered to their origin and can be seen moving when the eye is moved by the patient medially or laterally. Because the retinal is continuous with the optic nerve, a retinal detachment will never appear to cross over the optic nerve head.
4. If it is important to keep the eye free of ultrasound gel, a small Tegaderm can be placed over the closed eyelid and the gel can then be placed on top of the Tegaderm.

SOFT-TISSUE ULTRASOUND

Clinical Summary

Soft-tissue ultrasound has been shown to increase diagnostic accuracy, speed disposition, and reduce complications of procedures.

Indications

- Suspected fluid collection (abscess, hematoma, etc)
- Localization of subcutaneous foreign body
- Guidance for procedures

Equipment

- High-frequency linear array

Patient Positioning

- Place the patient in a position of comfort that allows maximum exposure of

the area of interest.

Technique

- Image the area of interest by gently sweeping the probe across the skin. This should be done in two perpendicular planes. If possible, orient the planes along conventional axes of the body (ie, transverse and longitudinal).
- Identify the dermis, subcutaneous tissue, tendon, muscle, ligament, bone, and relevant neurovascular structures.
- Waterbath evaluation technique (WET): Distal extremities can be placed in a tub of water covering the area of interest and the water can be used as a conductive media for the ultrasound (Fig. 24.131). Ultrasound waves travel well through water, permitting a standoff approach where the probe need not actually touch the skin. This is better tolerated by the patient as no contact with the painful area is necessary and this technique facilitates imaging of extremely superficial structures.

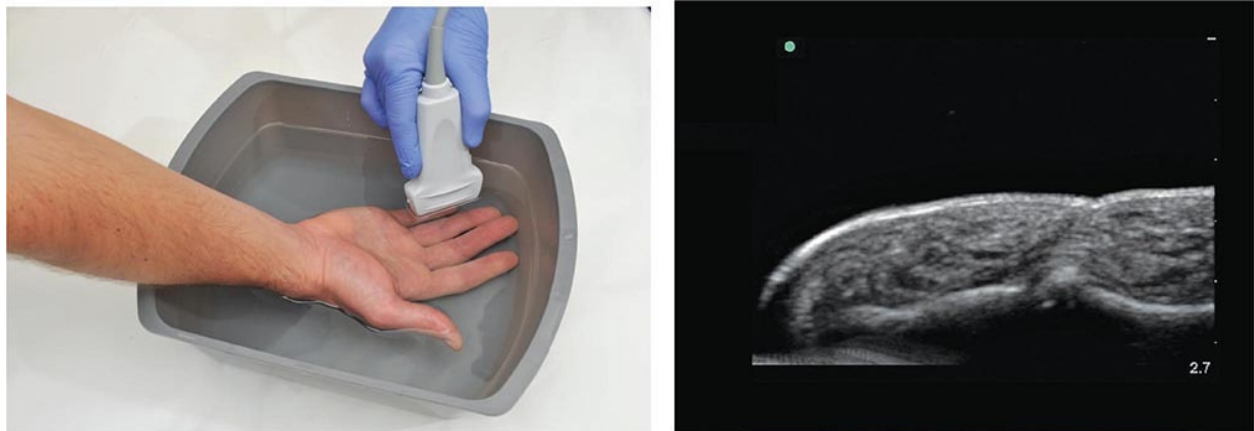


FIGURE 24.131 ■ Waterbath Evaluation of the Extremity. Distal extremities can be placed in a tub of water covering the area of interest, and the water can be used as a conductive media for the ultrasound. The ultrasound image shows the volar aspect of the distal phalanx of the middle finger obtained using the waterbath technique. (Photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Jordan Rupp, MD.)

- Apply superficial marking to overlying skin if necessary.

Abnormal Findings

- Edema/cellulitis: Increased interstitial fluid appears similar on ultrasound regardless of the cause (eg, cellulitis, lower extremity–dependent edema).

Initially, increased echo-genicity and loss of definition of the tissue are noted in the subcutaneous fat. This obscures deeper structures such as muscle and bone. As the condition progresses, fluid is noted to accumulate diffusely in a reticular pattern, creating a cobblestone appearance (Fig. 24.132).

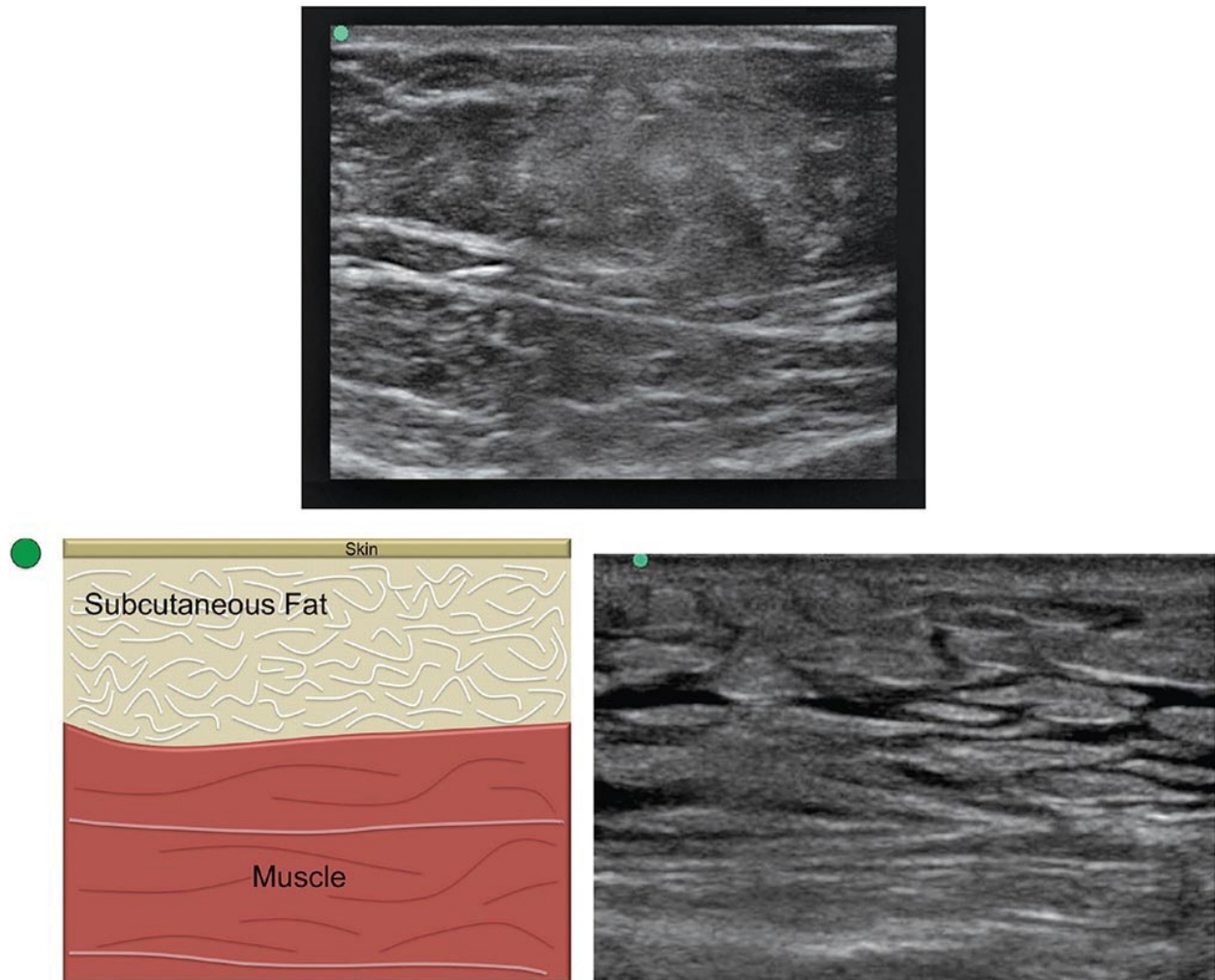


FIGURE 24.132 ■ Soft-Tissue Ultrasound: Cellulitis. Initially, increased echogenicity and loss of definition of the tissue are noted in the sub-cutaneous fat (top). As the condition progresses, fluid is noted to accumulate diffusely in a reticular pattern creating a cobblestone appearance (bottom). (Ultrasound contributor: Robinson M. Ferre, MD; illustration contributor: Robinson M. Ferre, MD.)

- **Abscess:** An abscess appears as a focal fluid accumulation, often with a significant amount of increased echogenicity in the surrounding tissues. The fluid may appear anechoic or have mixed complex echoes (Fig. 24.133). If drainage will be attempted, the depth and location of the abscess, any associated tracts, and other important and relevant adjacent structures should be noted and marked if appropriate (vessels, nerves, etc).

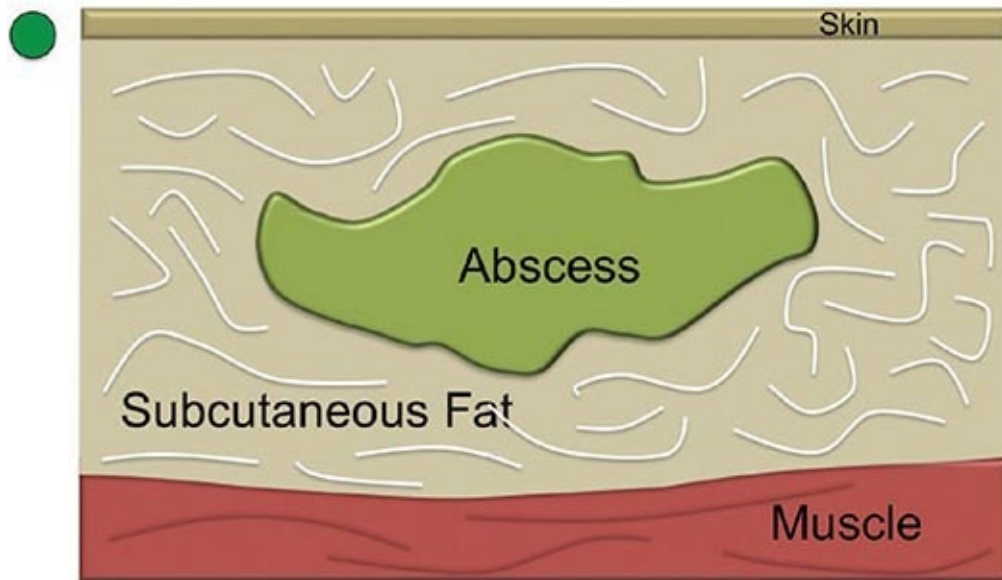
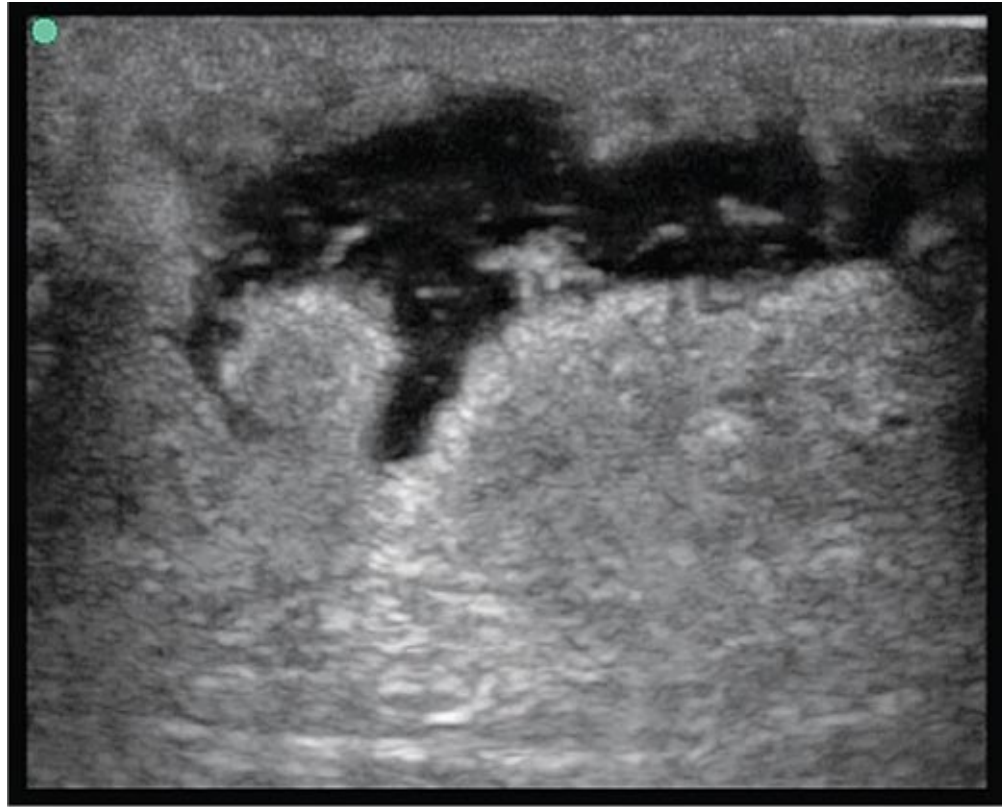


FIGURE 24.133 ■ Soft-Tissue Ultrasound: Abscess. An abscess appears as a focal fluid accumulation, often with a significant amount of increased echogenicity of the surrounding tissues. The fluid may appear anechoic or have mixed complex echoes. (Ultrasound contributor: Robinson M. Ferre, MD; illustration contributor: Robinson M. Ferre, MD.)

- Joint effusion: Ultrasound can readily identify fluid accumulation within joints. A distended joint capsule will be visualized with anechoic or

heterogeneous echogenic fluid ([Fig. 24.134](#)). Thickened synovial lining may be noted as well. Drainage can be assisted either statically, locating and marking area of maximal effusion, or dynamically, using ultrasound to guide the needle throughout the entire procedure.

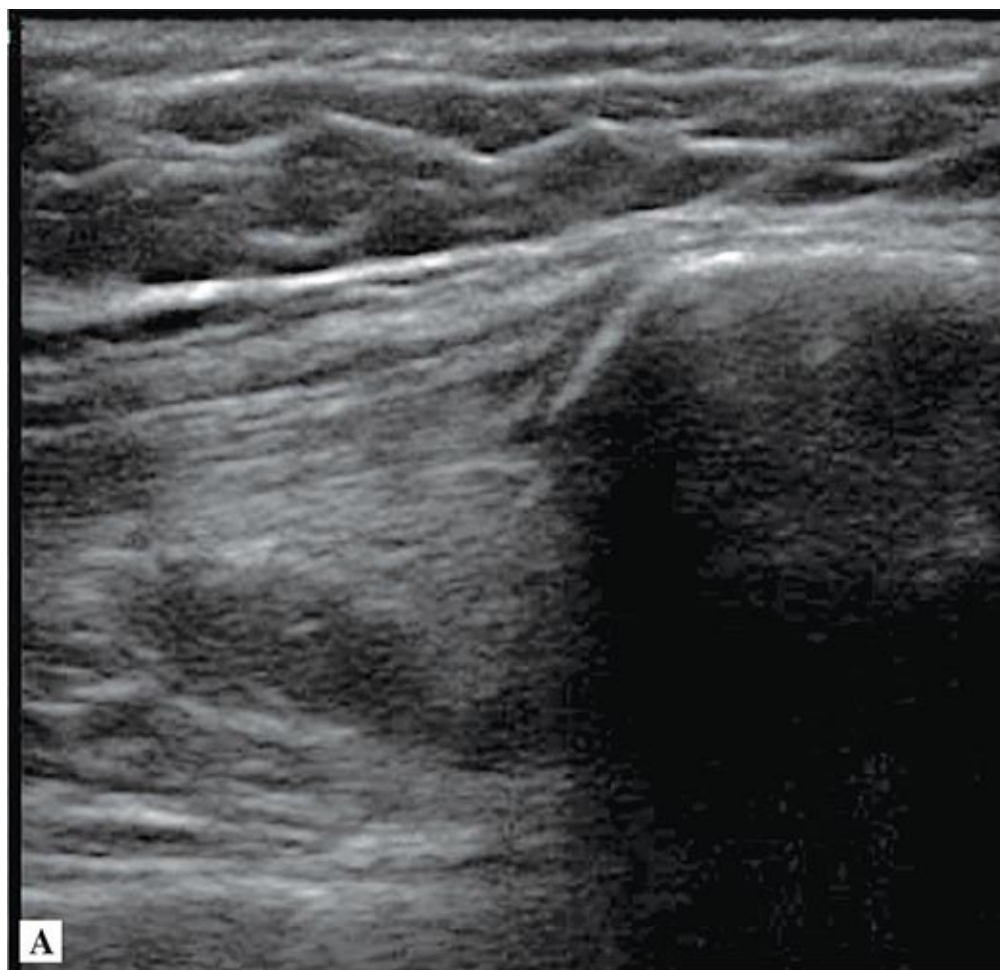


FIGURE 24.134 ■ Soft-Tissue Ultrasound: Joint Effusion. A knee effusion is seen in sagittal (A) and transverse (B) views. (Ultrasound contributor: Robinson M. Ferre, MD.)

- **Subcutaneous foreign body:** Most foreign bodies are visible using ultrasound. The foreign body's appearance depends on the shape, size, and composition of the object. Careful, slow passes should be made over the likely location, noting any abnormal object or defect in tissue ([Fig. 24.135](#)). EUS can assist in removal by either a static approach, marking the skin overlying the location, or dynamic technique guiding removal during real-time ultrasound. Care should be taken to identify any structures damaged by the foreign body or that may complicate removal.

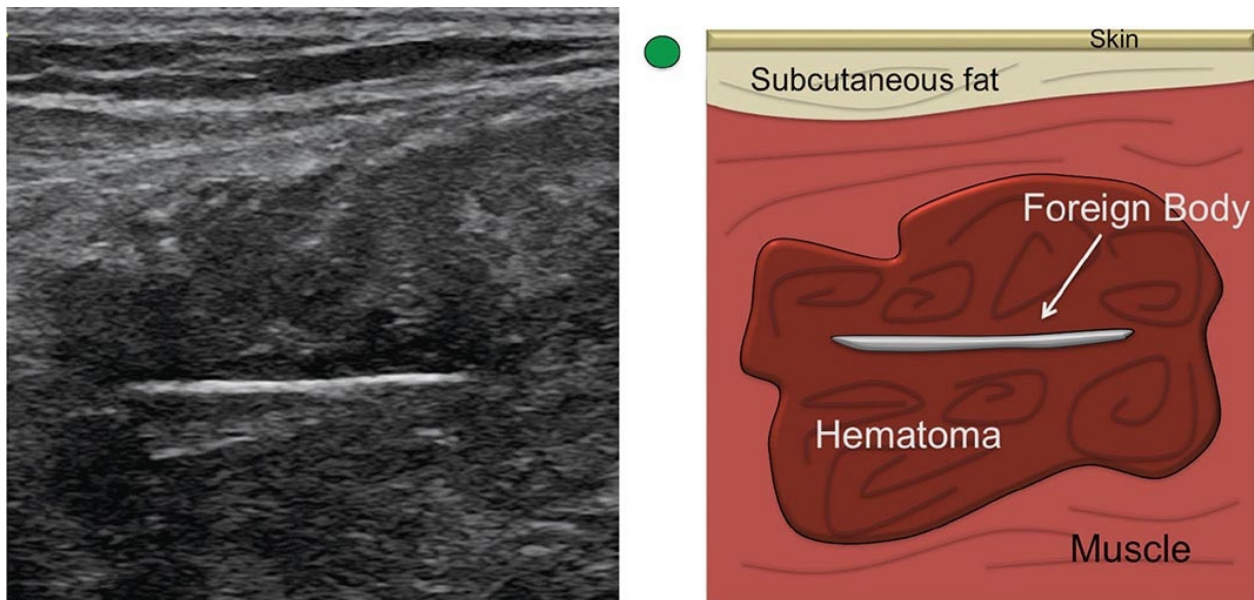


FIGURE 24.135 ■ Soft-Tissue Ultrasound: Foreign Body. An echogenic, linear foreign body is visualized in the superficial soft tissues with surrounding hematoma. (Illustration contributor: Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

Pearls

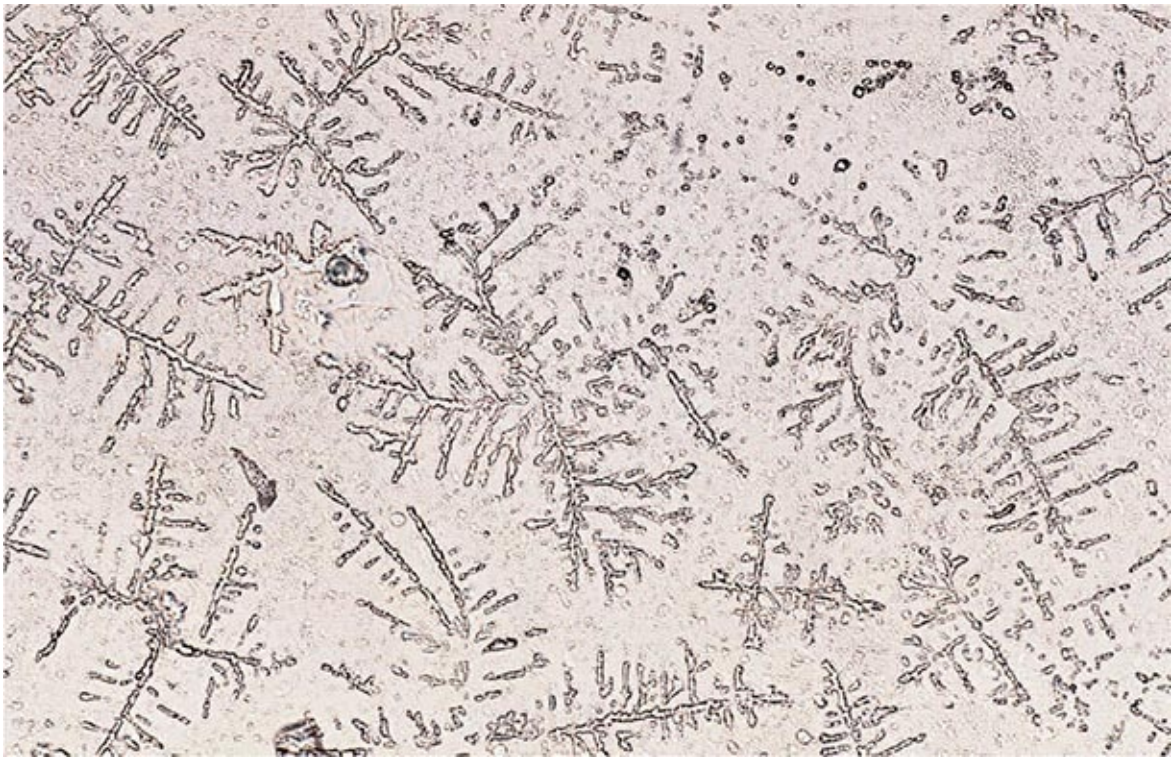
1. Always scan the area of interest in two planes. This will help to identify the exact location of the abnormality and exclude artifacts.
2. If needed, use the surrounding, unaffected tissue or other extremity as a control.
3. To optimize the image, reduce the depth to the appropriate level and increase the frequency of the probe. The dermis and other extremely superficial structures (< 0.5 cm) may not be well visualized due to near-field artifact. Consider the use of a standoff pad or water bath if feasible.

4. For procedures requiring sterile precautions, a sterile sheath should be used to cover the transducer for dynamically guided procedures. Many sterile ultrasound sheaths are commercially available, although, less preferably, a sterile glove or transparent adherent dressing (eg, Tegaderm) can be used. Sterile ultrasound gel or another sterile liquid (eg, Betadine or chlorhexidine) should be used as a conductive medium.

Chapter 25

MICROSCOPIC FINDINGS AND BODILY FLUIDS

Camiron L. Pfennig
B. Ethan Brown



Amniotic Fluid Ferning Pattern. Ferning is the arborization pattern found when a drop of amniotic fluid is allowed to air dry on a microscope slide and helps distinguish rupture of the amniotic sac in pregnancy from normal vaginal secretions. (Photo contributor: Robert Buckley, MD.)

Uses

To evaluate for the presence of cells, casts, and crystals.

Materials

Freshly collected urine specimen, centrifuge, graduated centrifuge tubes, glass microscope slide, and coverslip.

Method

1. Pour 10 mL of freshly collected urine into a graduated centrifuge tube.
2. Centrifuge at $\times 400$ to $\times 450$ gravity for 5 minutes.
3. Decant 9 mL of supernatant, leaving 1 mL in the tube.
4. Resuspend the centrifuged pellet in the remaining 1 mL of urine by stirring with a pipet.
5. Place one drop of resuspended urine on a glass microscope slide.
6. Overlay with a coverslip.
7. Examine initially using scanning $\times 10$ power, emphasizing the periphery of the coverslip, since urinary elements tend to gather at the edges.
8. Switch to $\times 40$ power to focus on specific urinary elements such as cells, casts, and crystals. Use $\times 100$ power as needed for specific identification.

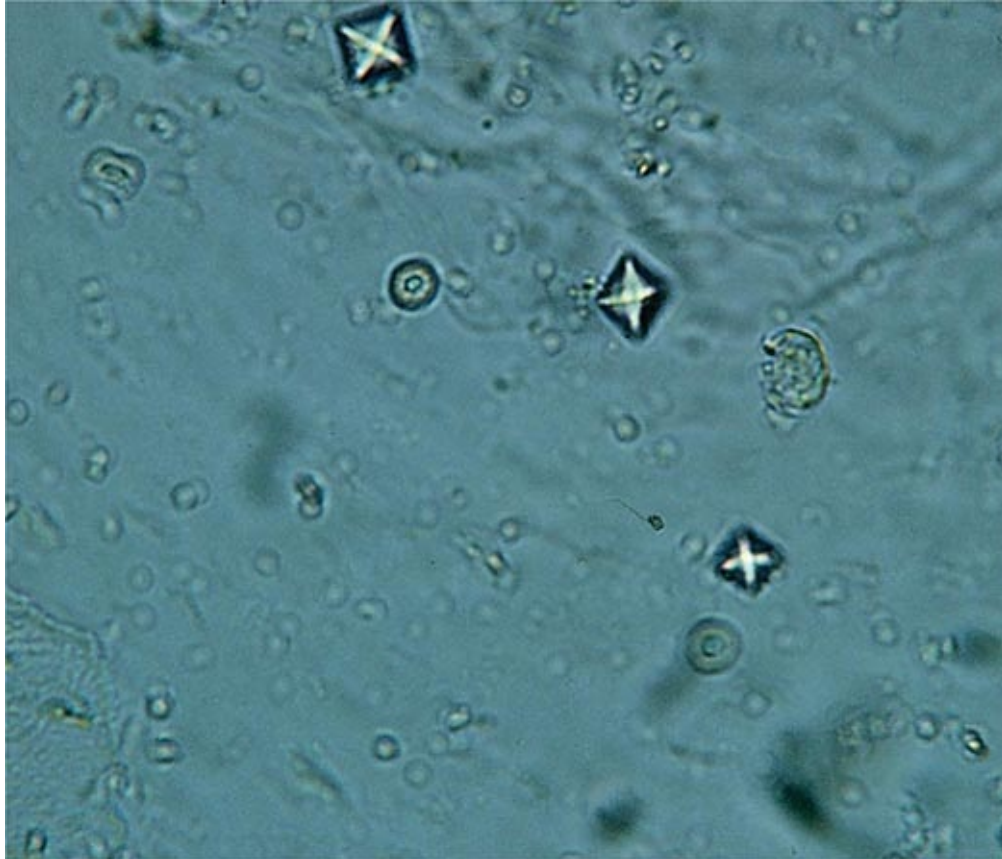


FIGURE 25.1 ■ Calcium Oxalate Crystals. Calcium oxalate crystals come in two shapes. The classically described octahedral, or envelope-shaped, crystals are made of calcium oxalate dihydrate. Calcium oxalate monohydrate crystals are needle-shaped. They are seen in acid or neutral urine. They may be found in the urine of patients with ethylene glycol ingestion. In addition, the urine of patients with ethylene glycol ingestion may also fluoresce under a Wood lamp. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. *Urinalysis and Body Fluids*. 4th ed. Philadelphia, PA: F.A. Davis Company; 2001, p. 97.)

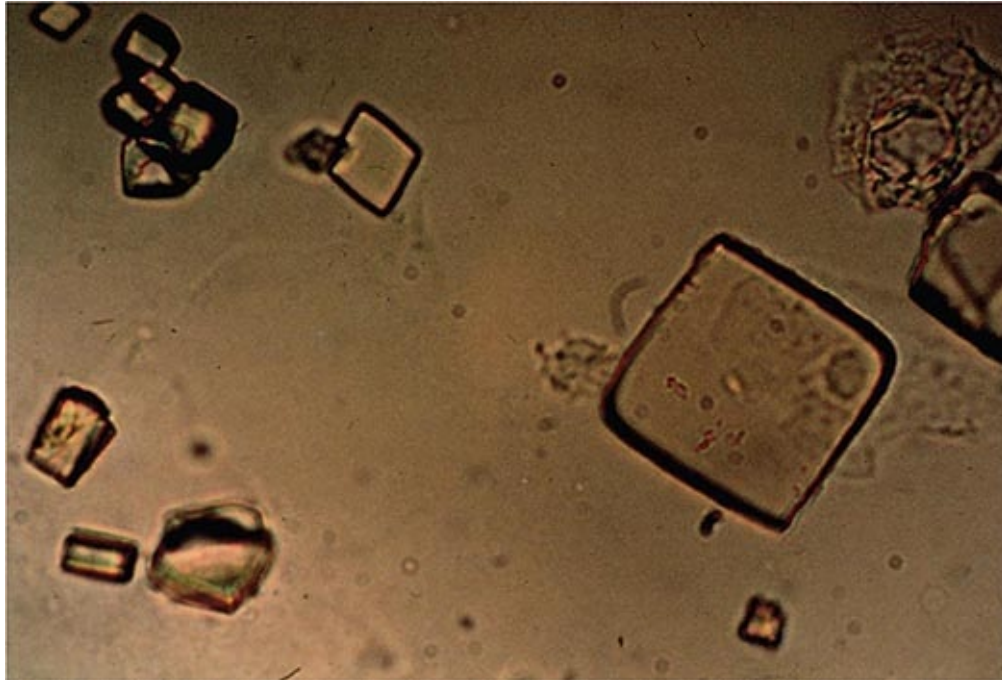


FIGURE 25.2 ■ Uric Acid Crystals. Uric acid crystals often have a yellow hue and a variety of sizes and shapes. They are found in acidic urine. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. *Urinalysis and Body Fluids*. 5th ed. Philadelphia, PA: F.A. Davis Company; 2008, p. 114.)

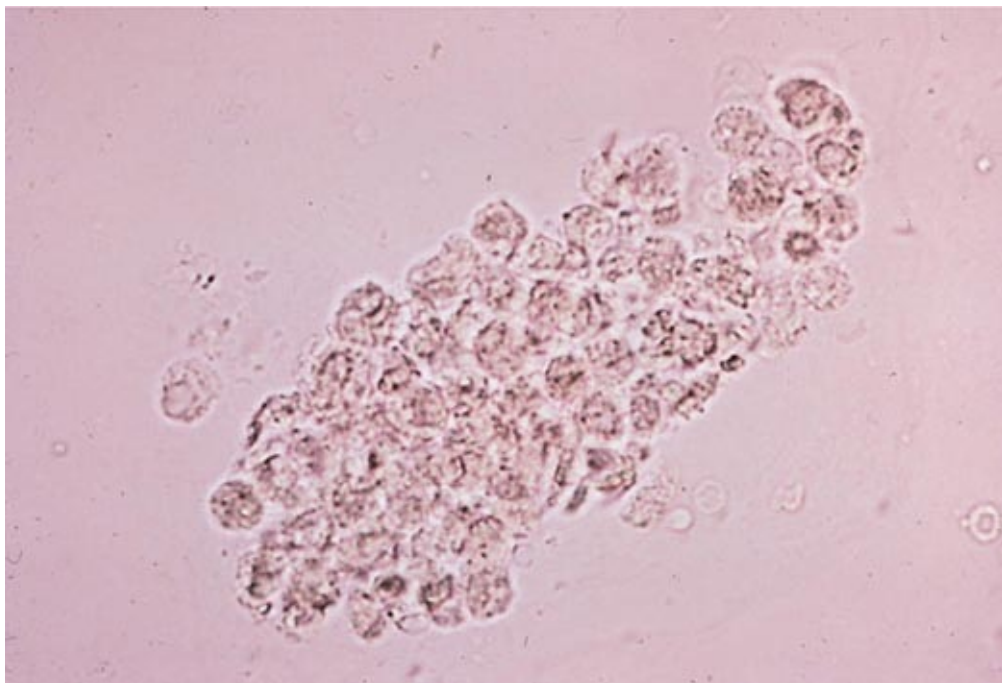


FIGURE 25.3 ■ White Blood Cell Casts. Usually two to three cells in width, white blood cell casts are indicative of upper urinary tract infection such as pyelonephritis. (Photo contributor: American Society for Clinical Pathology.)

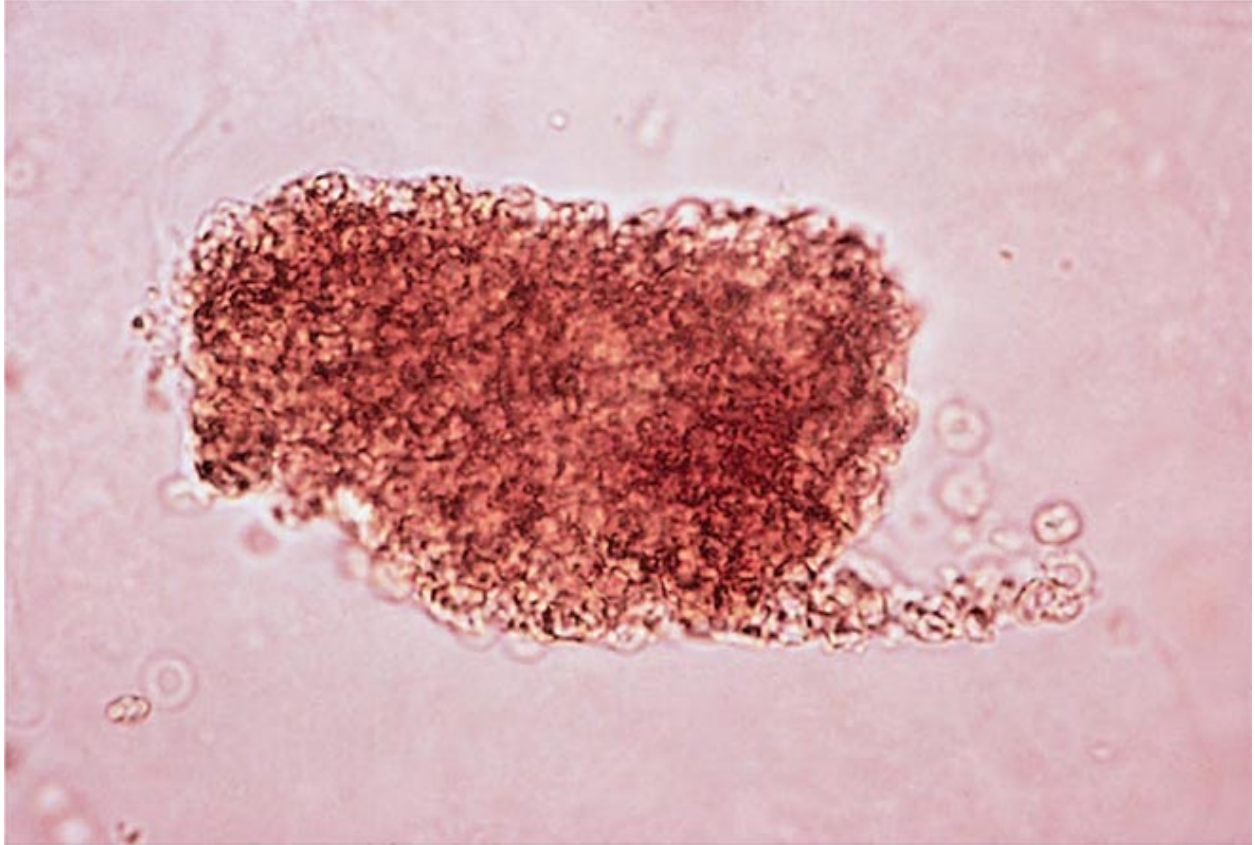


FIGURE 25.4 ■ Red Blood Cell Casts. Red blood cells casts range from 3 to 10 cells in width and are seen in glomerulonephritis. (Photo contributor: American Society for Clinical Pathology.)

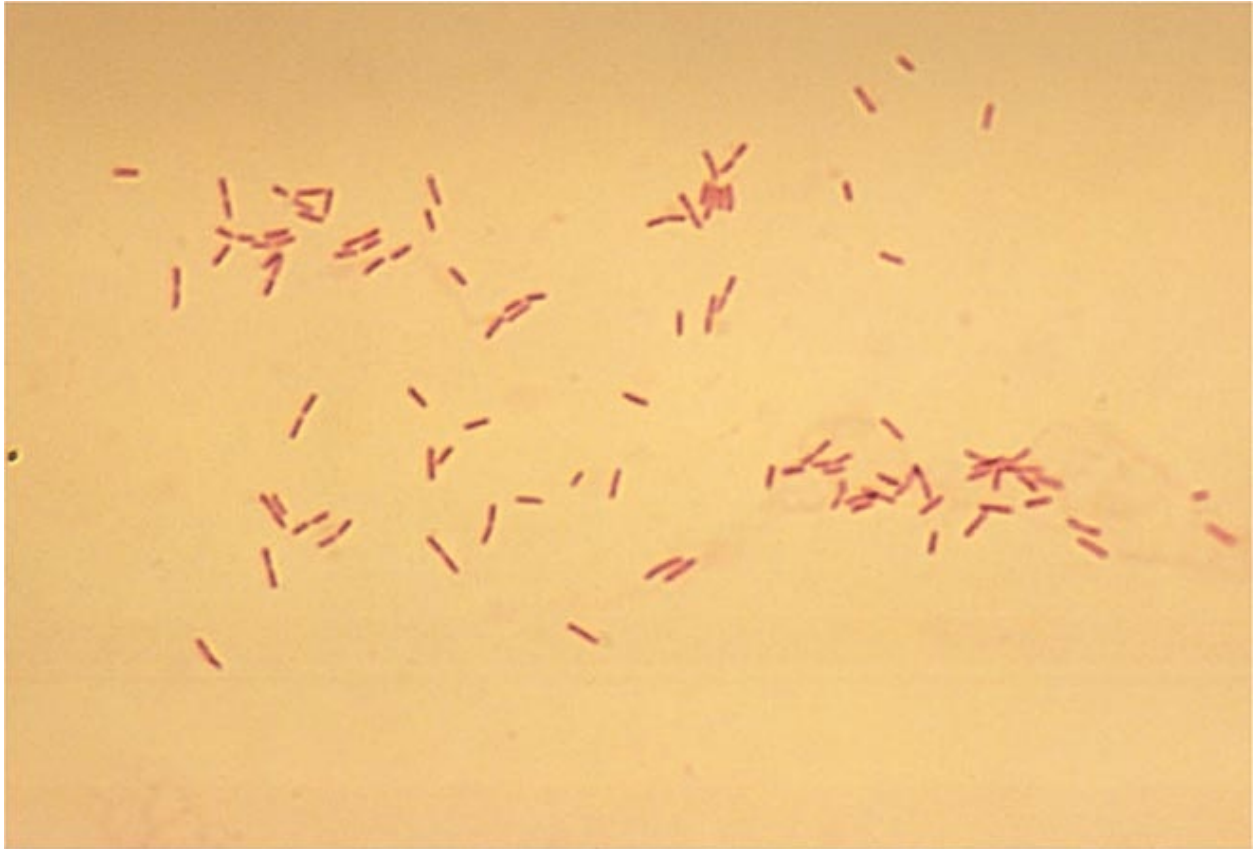


FIGURE 25.5 ■ Bacteria. Bacteria are often seen in urine specimens and either can be consistent with infection or may result from local contamination from surrounding skin during specimen collection. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc., Nutley, NJ.)

SYNOVIAL FLUID ANALYSIS

Uses

To determine the presence of uric acid crystals (in patients with gout) or calcium pyrophosphate crystals (in patients with pseudogout) in joint fluid.

Materials

Freshly collected joint fluid, glass microscope slide, coverslip, and polarizer.

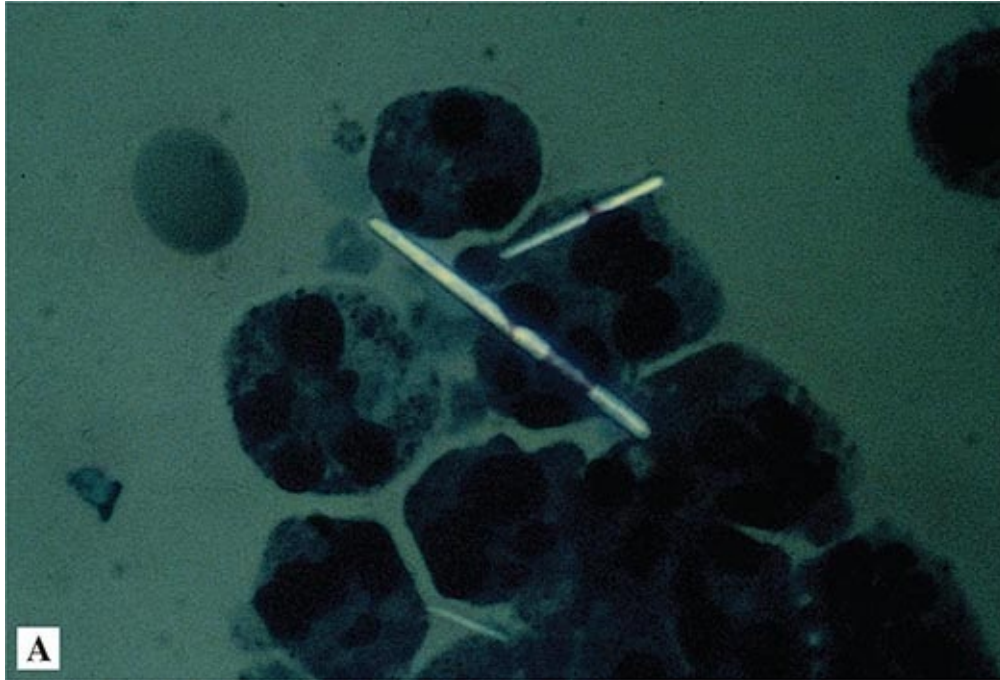


FIGURE 25.6A ■ Polarized Uric Acid Crystals (×500). Intracellular needle-like uric acid crystals are seen within the polymorpho-nuclear cells from the joint fluid in a patient with gout, using a direct polarizing light. (Reproduced with permission, from Strasinger SK, Di Lorenzo MS. *Urinalysis and Body Fluids*. 5th ed. Philadelphia, PA: F.A. Davis Company; 2008, p. 216.)

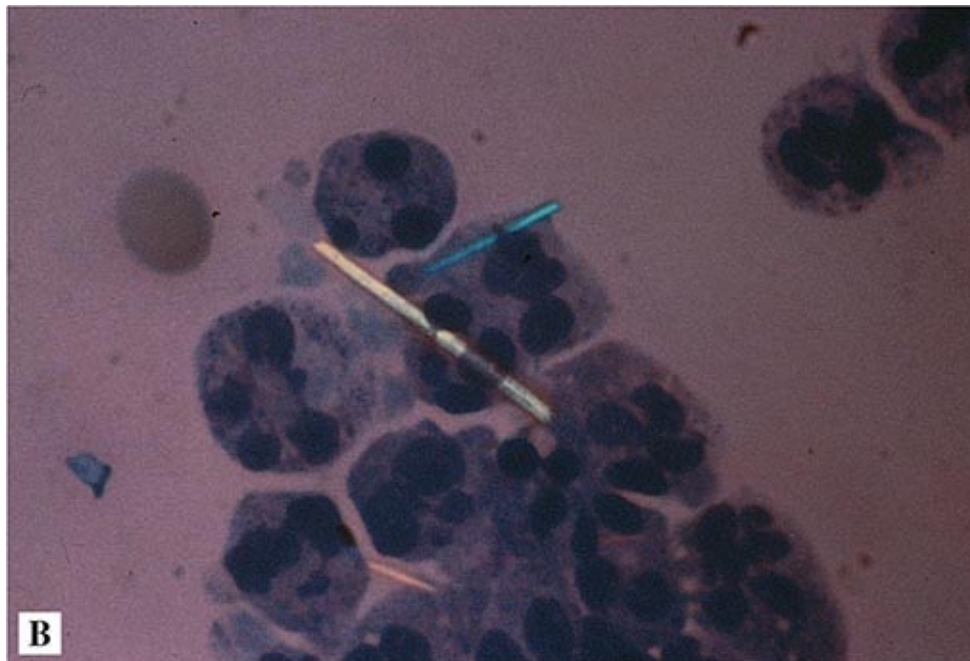


FIGURE 25.6B ■ Compensated Polarized Uric Acid Crystals (×500). Once crystals are found with a direct polarizing light, identification is made by using a compensated polarized light. The yellow crystal is aligned parallel to the slow vibration component of the compensator (negatively birefringent). The blue crystal is perpendicular (crossed urate blue). (Reproduced with permission, from Strasinger SK, Di Lorenzo MS. *Urinalysis and Body Fluids*. 5th ed. Philadelphia, PA: F.A. Davis Company; 2008, p. 217.)

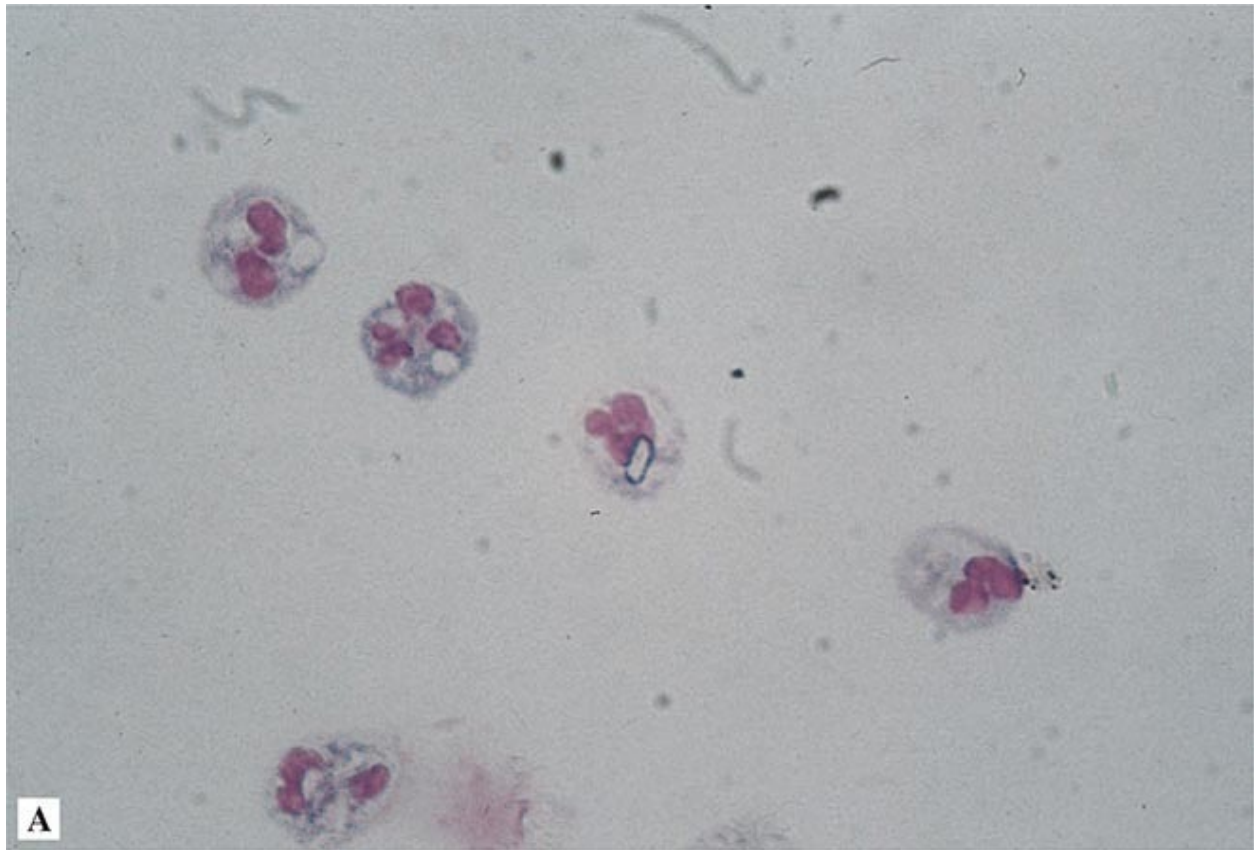


FIGURE 25.7A ■ Polarized Calcium Pyrophosphate Crystals ($\times 1000$). Intracellular rhomboid crystals in the joint of a patient with pseudogout. They may also appear as rods. (Reproduced with permission, from Strasinger SK, Di Lorenzo MS. *Urinalysis and Body Fluids*. 5th ed. Philadelphia, PA: F.A. Davis Company; 2008, p. 216.)

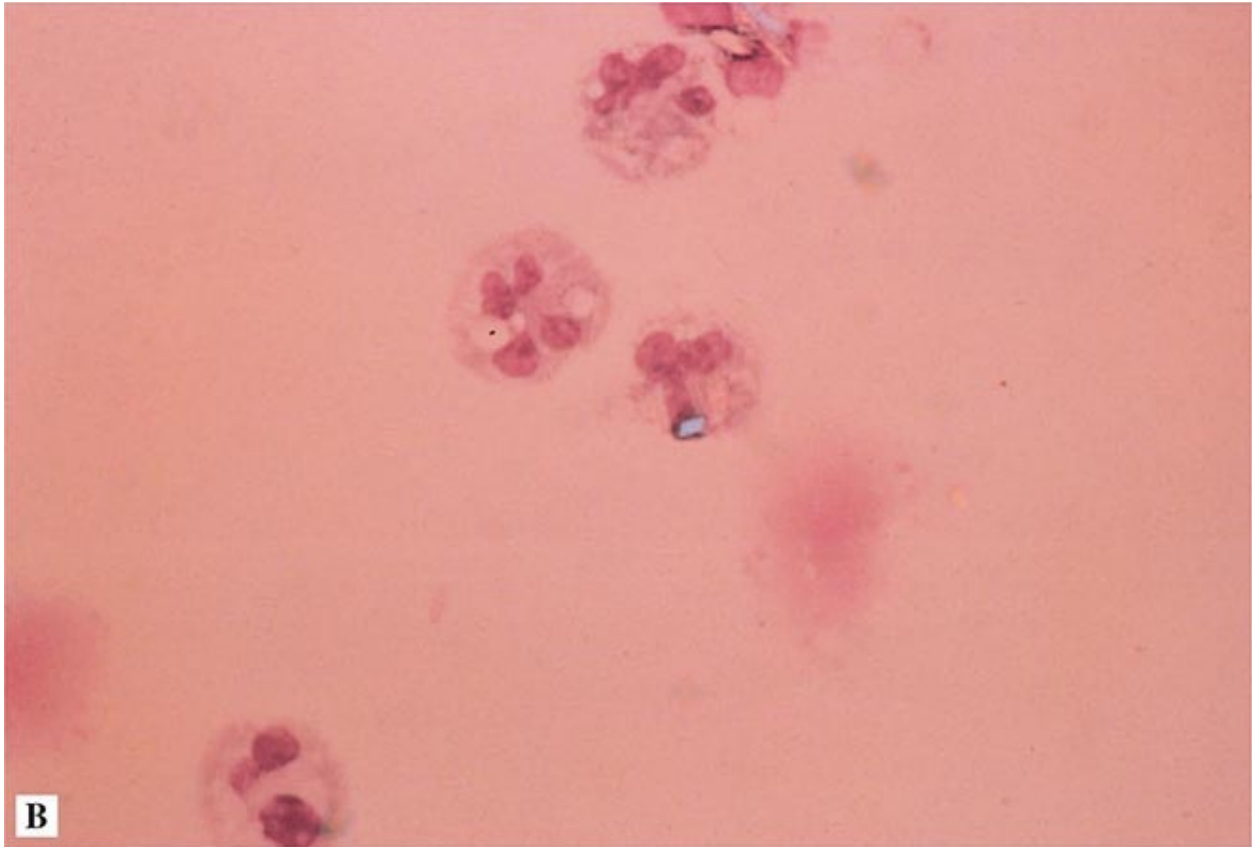


FIGURE 25.7B ■ Compensated Polarized Calcium Pyrophosphate Crystals ($\times 1000$). The blue calcium pyrophosphate crystal is aligned parallel to the slow vibration component of the compensator (positively birefringent). (Reproduced with permission, from Strasinger SK, Di Lorenzo MS. *Urinalysis and Body Fluids*. 5th ed. Philadelphia, PA: F.A. Davis Company; 2008, p. 217.)

Method

1. To prevent interference from polarizing artifacts, clean the slide and coverslip with alcohol prior to using them.
2. Using freshly collected unspun joint fluid, place a drop of joint fluid on the glass microscope slide.
3. Overlay coverslip.
4. View the slide using the polarizer.
5. Scan at $\times 10$ power; $\times 100$ power is needed to see intracellular crystals.

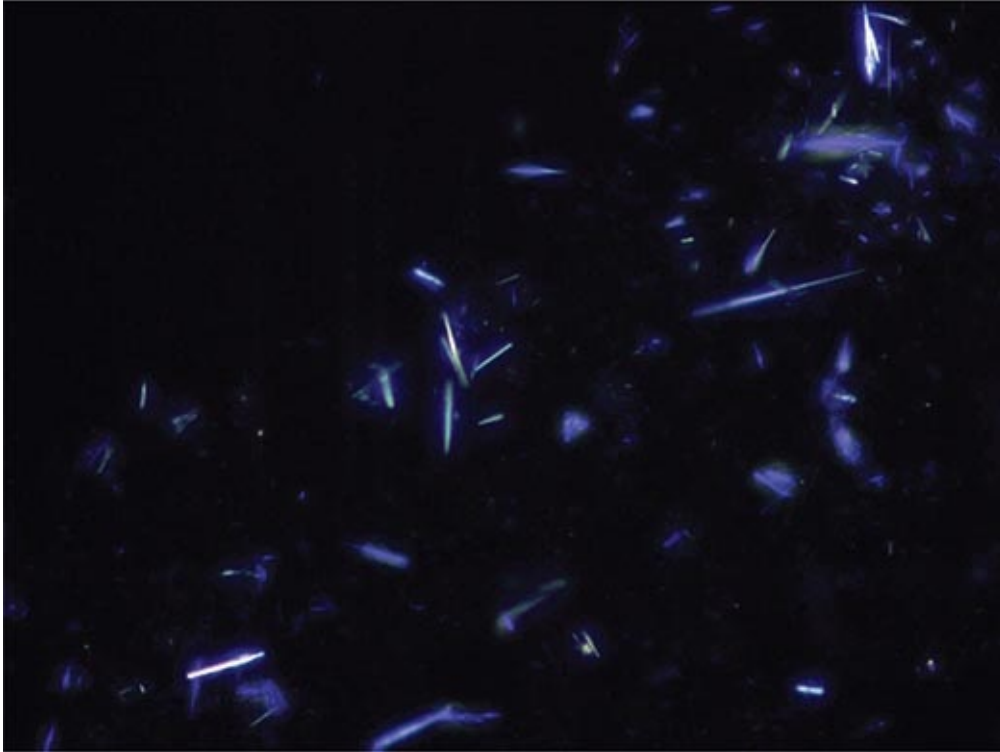


FIGURE 25.8 ■ Uncompensated Polarized Uric Acid Crystals (×400). The needle-like crystals can sometimes appear similar to those of calcium pyrophosphate dihydrate (CPPD) in pseudogout if not viewed under compensated polarized light. (Photo contributor: Jennifer Knight, MD.)

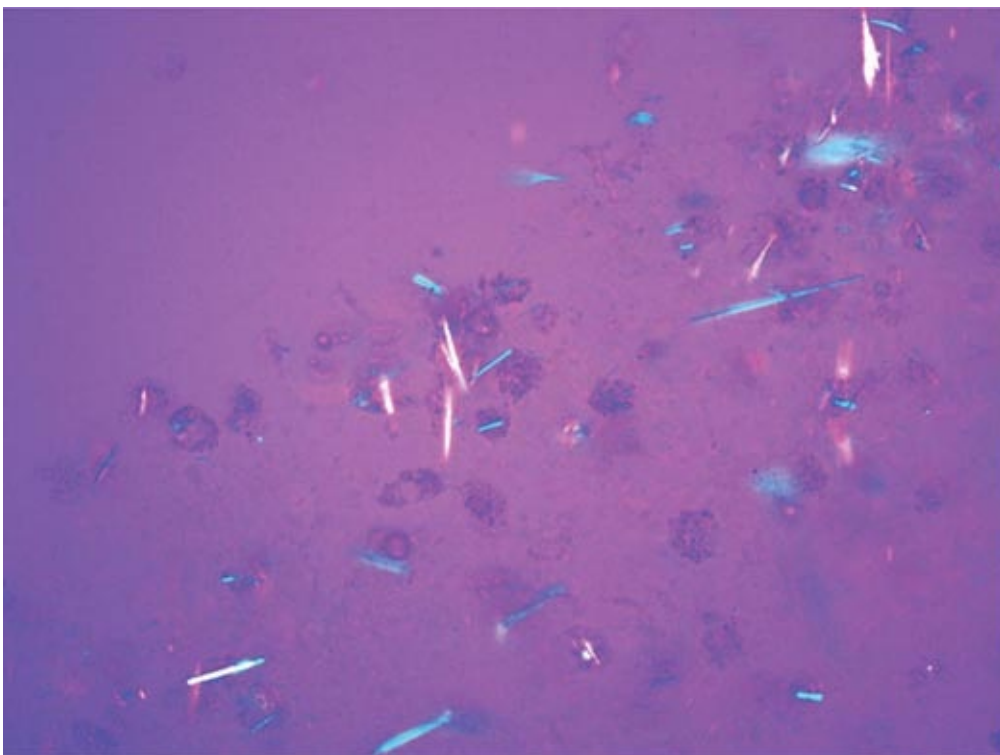


FIGURE 25.9 ■ Compensated Polarized Uric Acid Crystals (×400). The needle-like crystals are

negatively birefringent (yellow when parallel to the axis of the slow ray and blue when perpendicular), unlike CPPD, which is positively birefringent. (Photo contributor: Jennifer Knight, MD.)

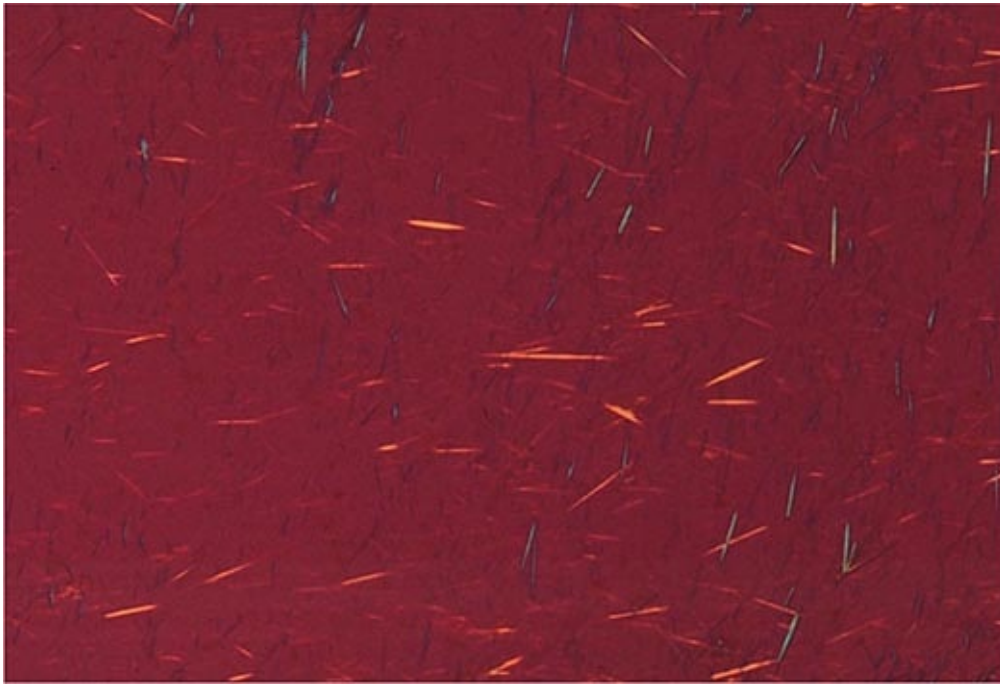


FIGURE 25.10 ■ Extracellular Uric Acid Crystals (×100). Extracellular uric acid crystals are seen under compensated polarized light. Notice the change of color with crystal alignment. (Reproduced with permission, from Strasinger SK, Di Lorenzo MS. *Urinalysis and Body Fluids*. 5th ed. Philadelphia, PA: F.A. Davis Company; 2008, p. 216.)

GRAM STAIN

Uses

The 1st step in the identification of a predominant bacterial organism in a specimen. Classifies an organism by its cell wall's ability to retain crystal violet dye during solvent treatment. Morphology of the organism is also identified.

Materials

Freshly collected specimen to be examined, glass microscope slide, crystal violet, Gram iodine, acetone-alcohol (acetone, 30 mL, and 95% alcohol, 70 mL), safranin, and Bunsen burner.

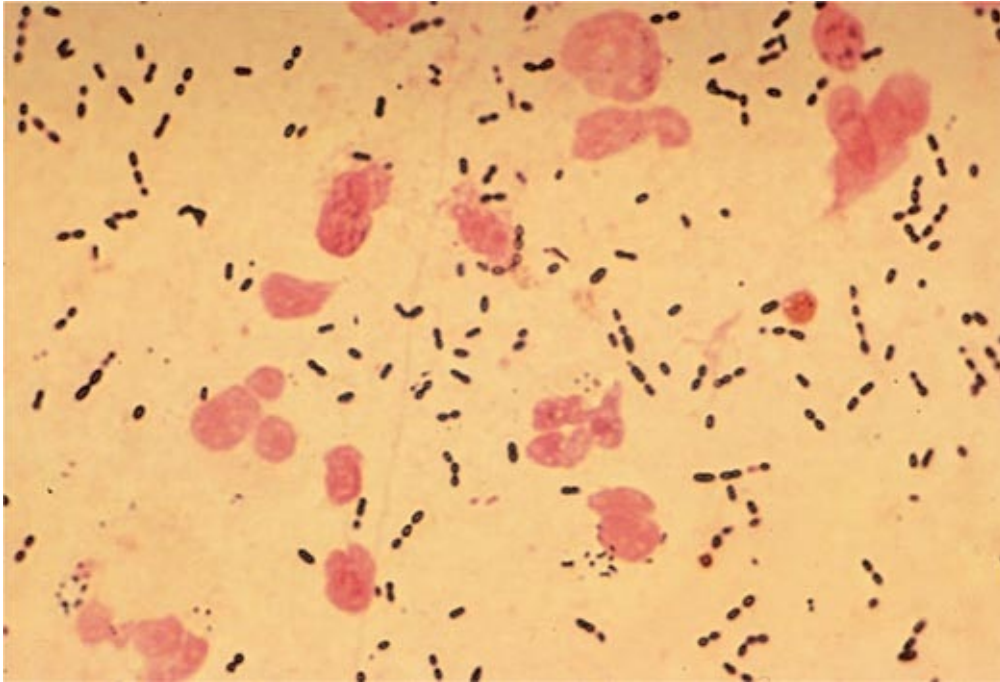


FIGURE 25.11 ■ **Gram Stain—*Streptococcus pneumoniae*.** Gram-positive, kidney-shaped diplococci of *S pneumoniae*. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc. Nutley, NJ.)

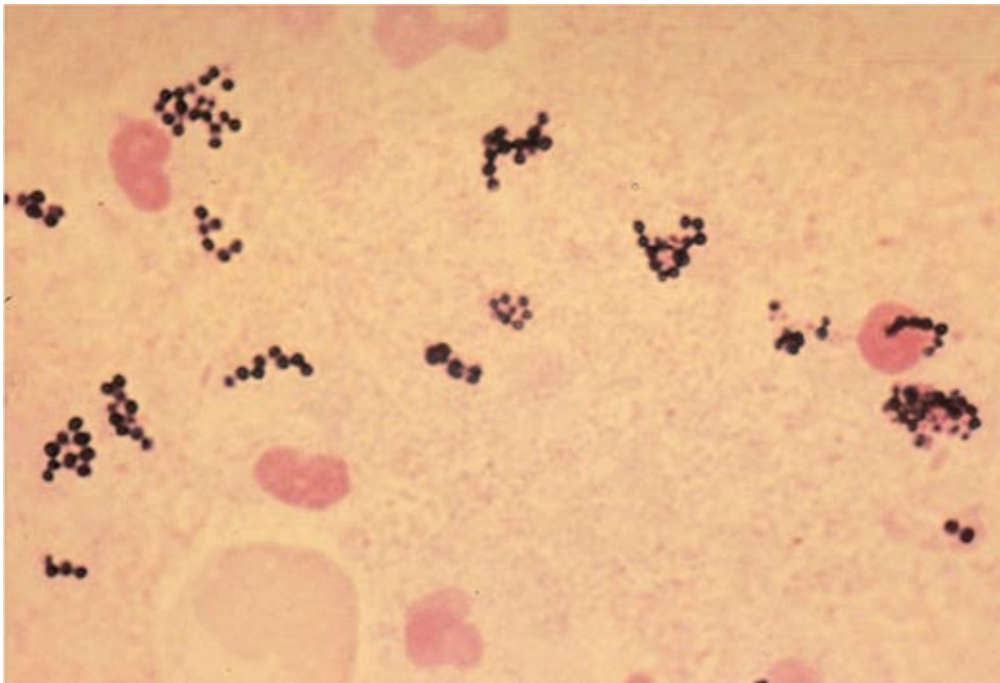


FIGURE 25.12 ■ **Gram Stain—*Staphylococcus aureus*.** Small clusters of gram-positive cocci seen in *S aureus* infection. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc. Nutley, NJ.)

Method

1. Put specimen on dry, clean glass microscope slide and allow to air dry.
2. Heat-fix specimen by gently passing over flame.
3. Cover specimen with crystal violet for 1 minute.
4. Rinse off completely with water; do not blot.
5. Cover specimen with Gram iodine for 1 minute.
6. Rinse off completely with water; do not blot.
7. Decolorize for 30 seconds with gentle agitation in acetone-alcohol.
8. Rinse off completely with water; do not blot.
9. Cover with safranin for 10 to 20 seconds.
10. Rinse off completely with water and let air dry.

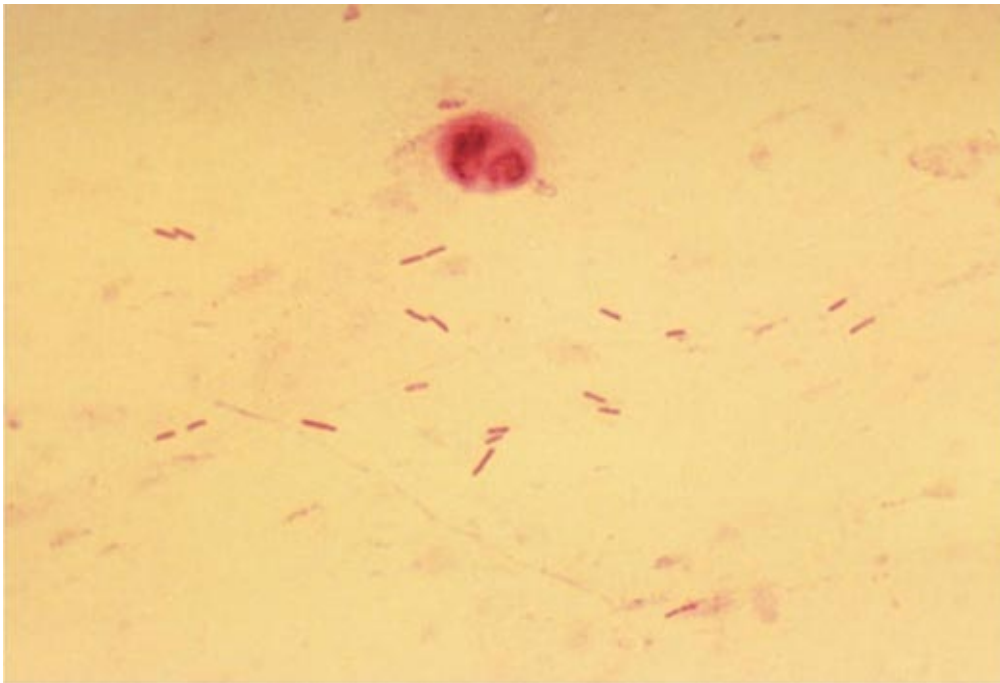


FIGURE 25.13 ■ Gram Stain—Gram-Negative Rods. Gram-negative rods of *Pseudomonas aeruginosa*. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc. Nutley, NJ.)

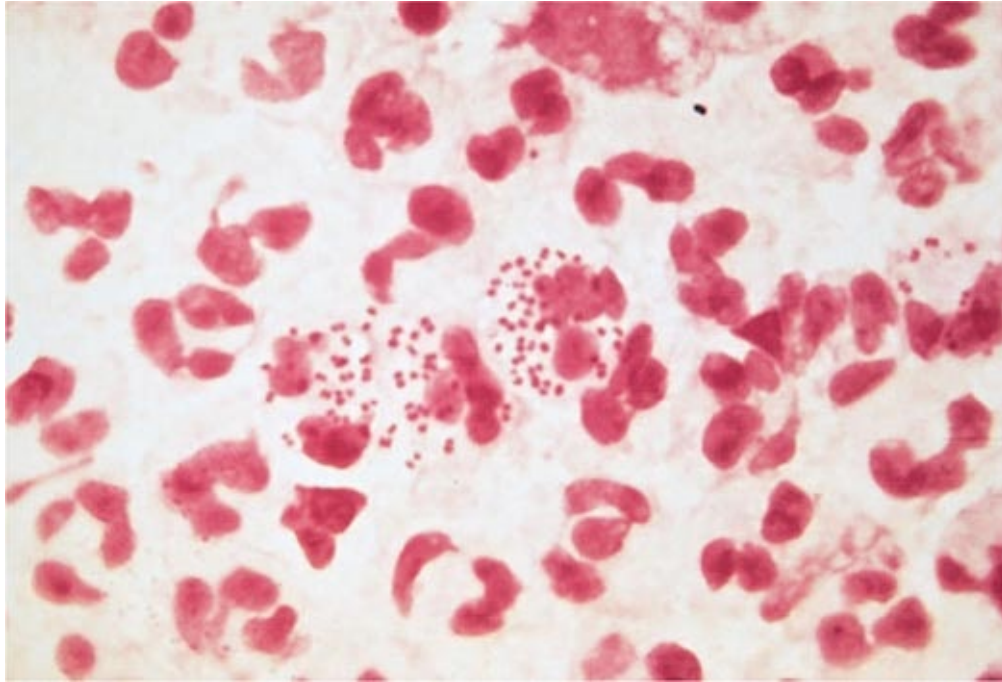


FIGURE 25.14 ■ Gram Stain—*Neisseria gonorrhoeae*. Multiple gram-negative, intracellular diplococci from a patient with *N gonorrhoeae*. (Reproduced with permission, from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). *Atlas of Sexually Transmitted Diseases*. London, United Kingdom: Mosby-Wolfe; 1990.)

DARK-FIELD EXAMINATION FOR *TREPONEMA PALLIDUM*

Uses

To evaluate a lesion (chancre, mucous patch, condyloma lata, skin rash) for the presence of *Treponema pallidum*.

Materials

Compound microscope with dark-field condenser (dark-field microscope), glass microscope slide, coverslip, and physiologic saline.

Method

1. From chancre or condyloma lata:

- a. Gently abrade the lesion with a dry gauze.
 - b. Dab away any bleeding.
 - c. Touch slide to exudative fluid in base of lesion.
 - d. Overlay coverslip and view immediately under dark-field microscope using $\times 40$ and $\times 100$ objectives.
2. From mucous patch:
- a. Touch slide to mucous patch.
 - b. Overlay coverslip and view immediately under dark-field microscope using $\times 40$ and $\times 100$ objectives.
3. From skin lesion:
- a. Gently scrape surface of skin lesion with edge of a number 15 scalpel blade.
 - b. Dab away any bleeding.
 - c. Touch slide to exudative fluid rising from skin lesion.
 - d. Overlay coverslip and view immediately under dark-field microscope using $\times 40$ and $\times 100$ objectives.



FIGURE 25.15 ■ Dark-Field Microscopy. Examined under a dark-field microscope at $\times 40$ or $\times 100$ power,

spirochetes appear as motile, bright corkscrews against a black background. (Reproduced with permission, from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). *Atlas of Sexually Transmitted Diseases and AIDS*. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)

VAGINAL FLUID WET MOUNT

Uses

To examine for clue cells, *Trichomonas*, and sperm.

Materials

Aqueous sodium chloride, glass microscope slide, and coverslip.

Method

1. Place a drop of saline onto the middle of the glass slide. (Alternative method: Place several drops of saline in a small glass test tube and place the swab in the tube. The swab can then be wiped onto a slide at a later time.)
2. Mix a small amount of vaginal fluid to be examined into the saline drop.
3. Overlay a coverslip.
4. Examine directly through microscope at $\times 40$ and $\times 100$ (oil immersion).

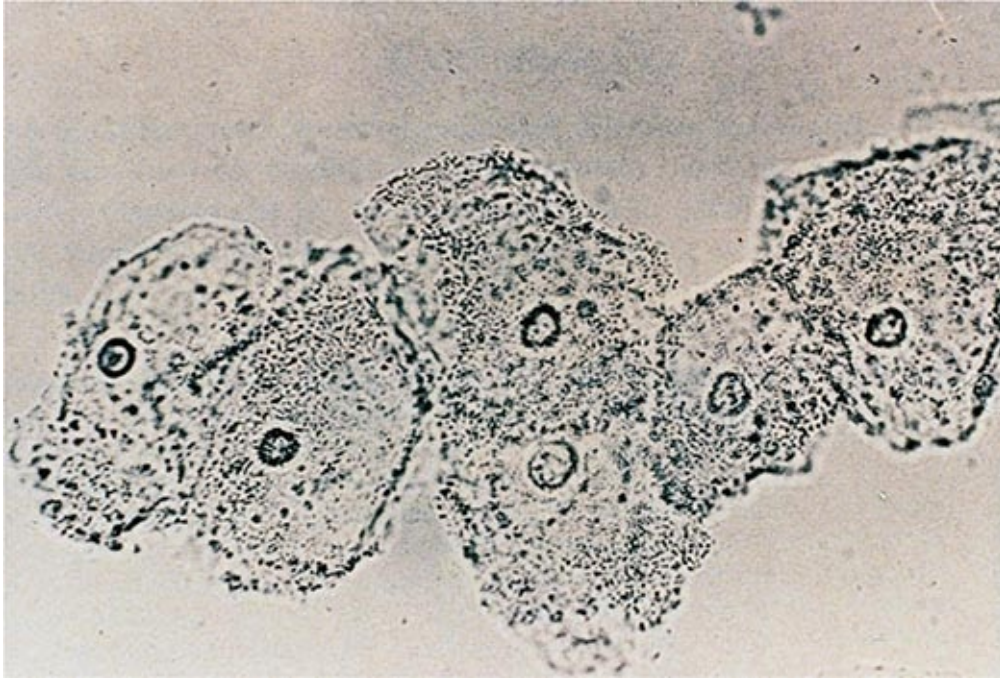


FIGURE 25.16 ■ Clue Cells. “Glitter cell” or “clue cell”: Epithelial cell covered with adherent bacteria in a wet mount of a vaginal specimen from a patient with *Gardnerella vaginalis* (also known as nonspecific vaginitis or bacterial vaginosis). Note the refractile appearance, indistinct borders, and ragged edges of the epithelial clue cell. (Photo contributor: Curatek Pharmaceuticals.)

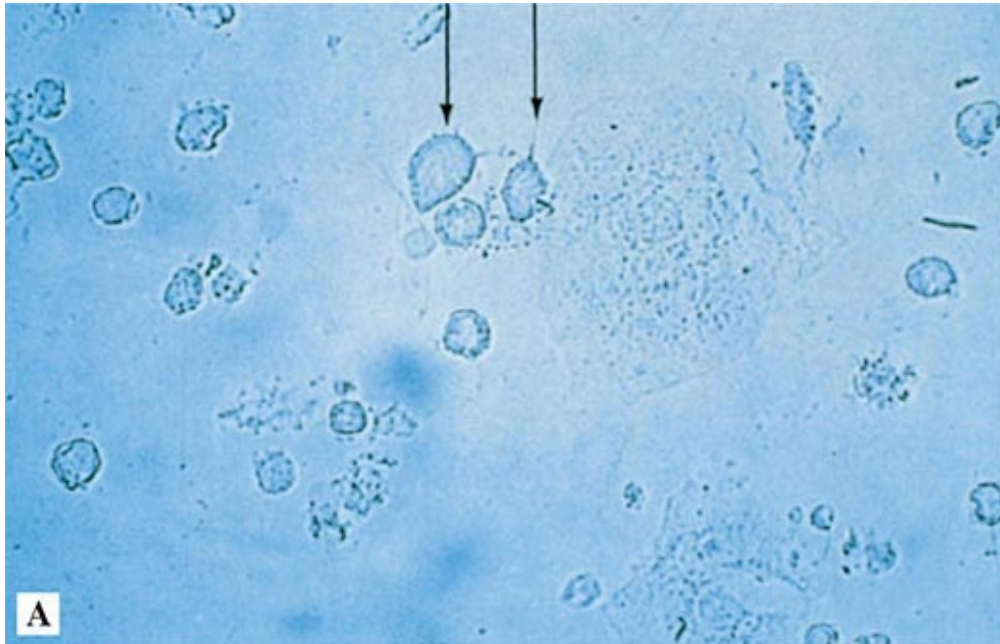


FIGURE 25.17A ■ Trichomonas. Saline wet mount demonstrating oval-bodied, flagellated trichomonads. They are similar in size to leukocytes and can be distinguished from them by their motility and presence of flagella. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed). *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)

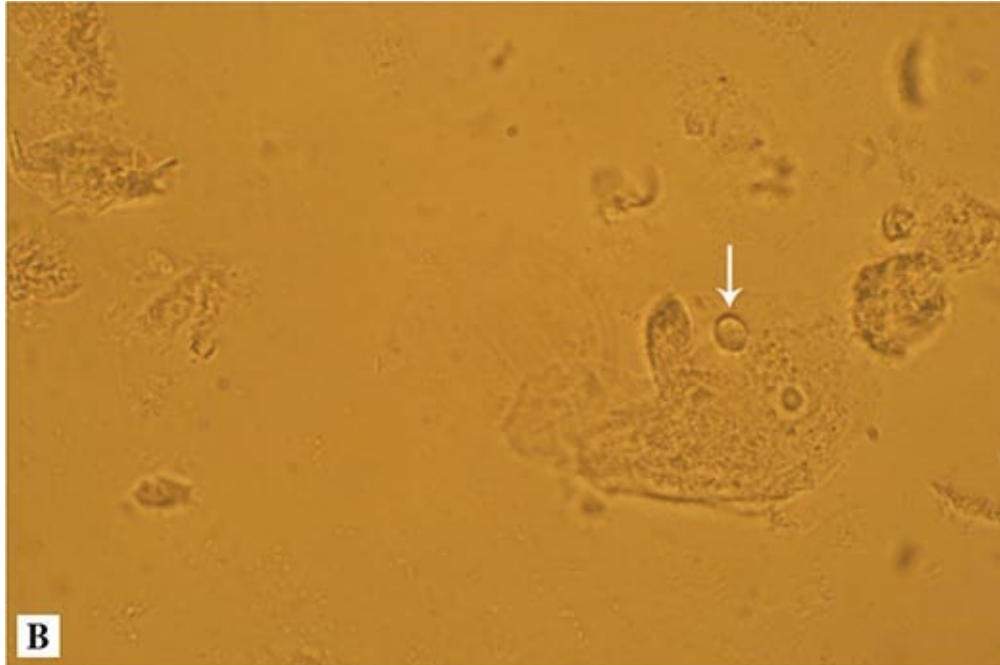


FIGURE 25.17B ■ **Trichomonas.** Saline wet mount demonstrating a single trichomonad. Its flagella are not easily seen in this photograph. See Video 25.1 to see flagellating movement. (Photo contributor: Lawrence B. Stack, MD.)

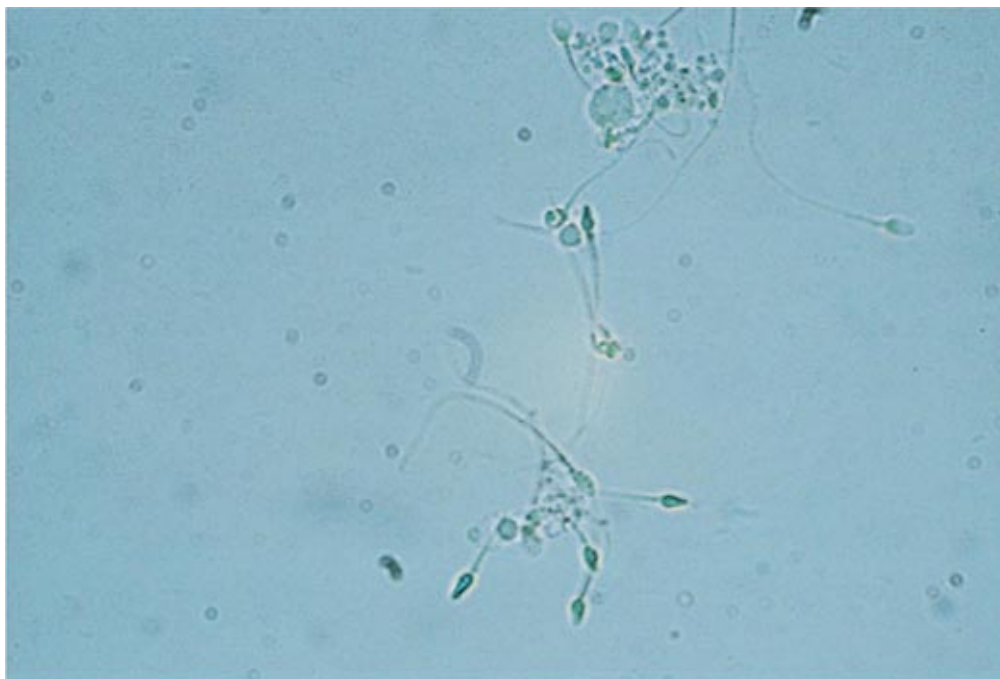


FIGURE 25.18 ■ **Spermatozoa.** Spermatozoa may be motile or immotile. (Reproduced with permission, from Strasinger SK, Di Lorenzo MS. *Urinalysis and Body Fluids*. 5th ed. Philadelphia, PA: F.A. Davis Company; 2008, p. 102.)

POTASSIUM HYDROXIDE MOUNT FOR *CANDIDA ALBICANS*

Uses

To examine for yeast.

Materials

Aqueous potassium hydroxide (KOH) 10%, glass microscope slide, and coverslip.

Method

1. Place a drop of KOH onto the middle of the glass slide.
2. Suspend a small amount of vaginal fluid into the drop of KOH.
3. Overlay a coverslip.
4. Let sit at room temperature for 30 minutes; as an alternative, gently heat the slide over a Bunsen burner but do not boil.
5. Examine under microscope for hyphae and spores.



FIGURE 25.19 ■ *Candida albicans*. Potassium hydroxide preparation of vaginal secretions from a patient with vaginal candidiasis due to *C. albicans*. Note the pseudohyphae characteristic of this organism. (Photo contributor: David A. Eschenbach, MD. From Handsfield HH (ed). *Atlas of Sexually Transmitted Diseases*, 3rd ed. New York, NY: McGraw Hill; 2011.)

STOOL EXAMINATION FOR FECAL LEUKOCYTES

Uses

To evaluate a patient's stool sample for the presence of fecal leukocytes.

Materials

Freshly collected liquid stool specimen, glass microscope slide, coverslip, and methylene blue.

Method

1. Place a drop of liquid stool onto the glass slide.
2. Add two drops of methylene blue to the stool specimen.
3. Mix thoroughly.
4. Overlay with a coverslip.
5. Place the edge of a piece of filter paper adjacent to the cover-slip to absorb any excess methylene blue.
6. Examine using $\times 10$ objective to scan specimen and $\times 40$ and $\times 100$ to identify specific leukocytes.

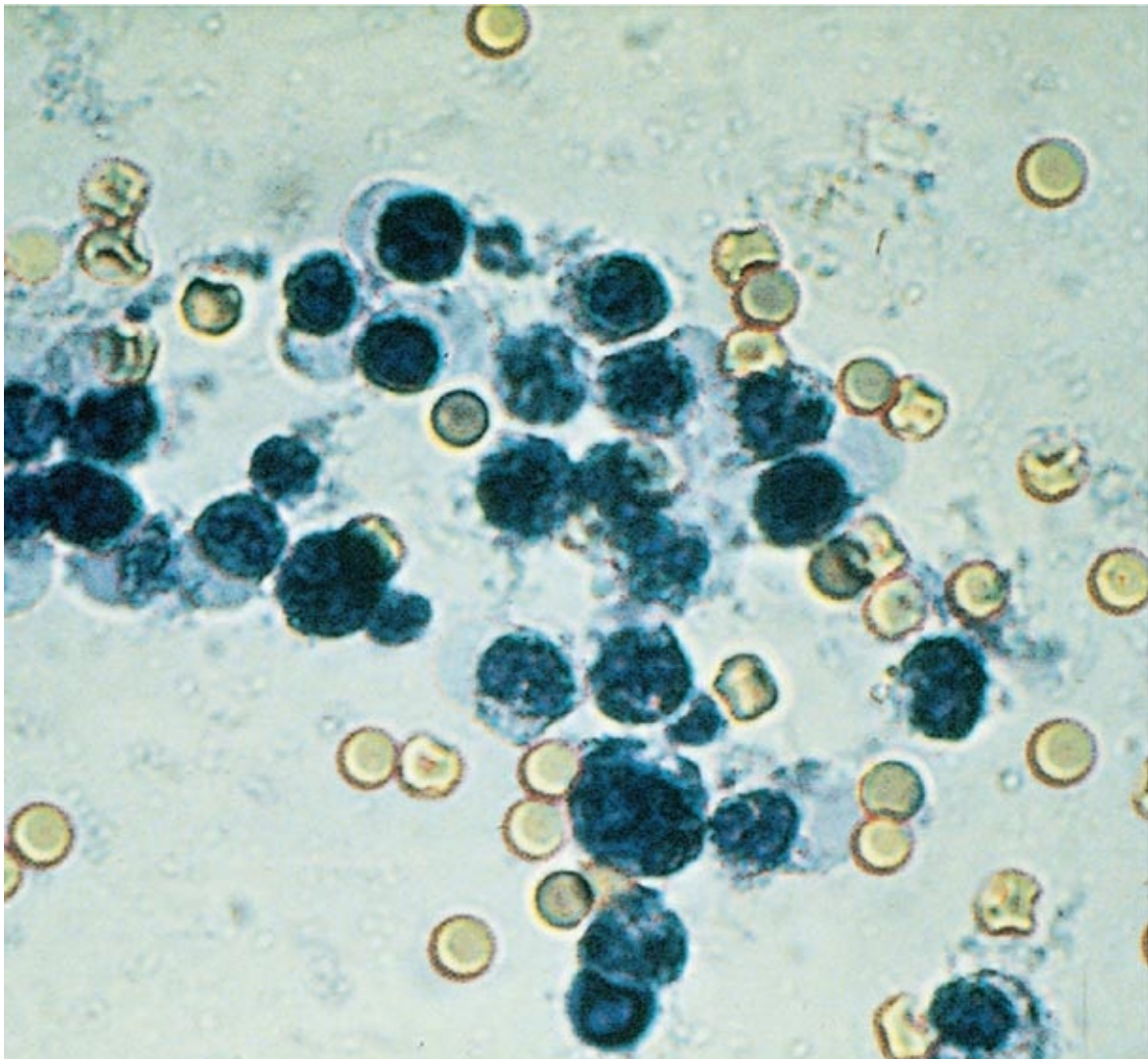


FIGURE 25.20 ■ Fecal Leukocytes. Multiple white cells in the stool specimen from a patient with bacterial diarrhea. (Photo contributor: Herbert L. DuPont, MD.)

SKIN SCRAPING FOR DERMATOSES AND INFESTATIONS

Uses

To determine fungal dermatoses or skin infestations.

Materials

Fresh skin scraping, glass microscope slide, coverslip, 10% potassium hydroxide or mineral oil.

Method

1. Specimen collection:
 - a. Gently scrape skin lesion with edge of a number 15 scalpel.
2. Slide preparation:
 - a. Pediculosis may be seen grossly clinging to individual hairs or under low power. Live nits may fluoresce with a Wood lamp.
 - b. For scabies, place a drop of KOH or mineral oil onto the slide.
 - c. Suspend a small amount of the scraping onto the drop.
 - d. Overlay a coverslip.
 - e. Let sit at room temperature for 30 minutes; as an alternative, gently heat the slide over a Bunsen burner but do not boil.
 - f. Examine under microscope for hyphae, spores, or infestations.

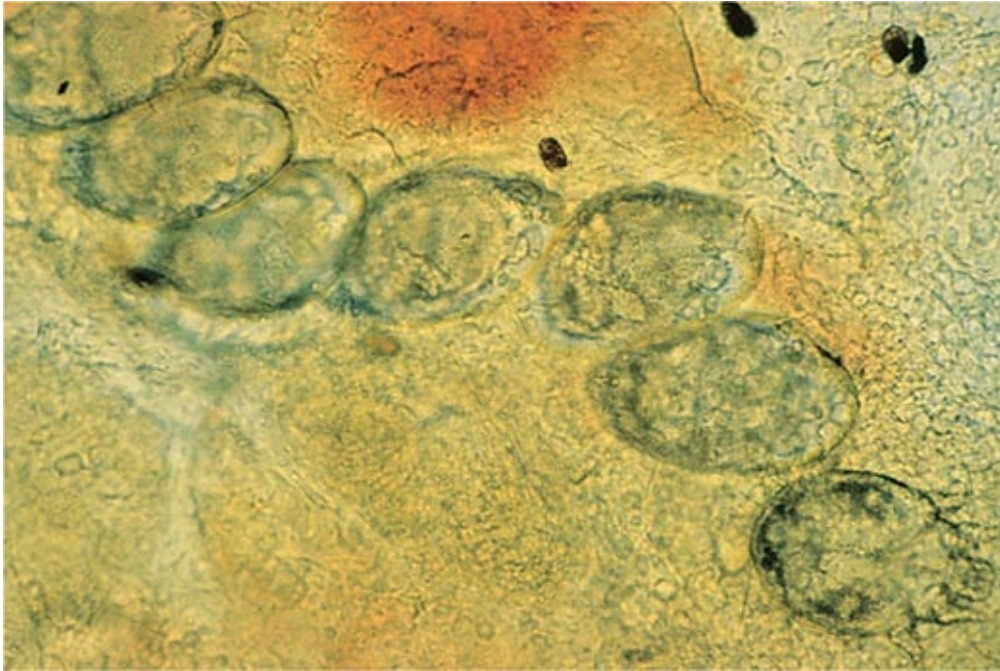


FIGURE 25.21 ■ Scabies. Skin scraping from a patient with scabies. Note the intact mite at the lower right of the photograph, and the ova and fecal pellets. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)

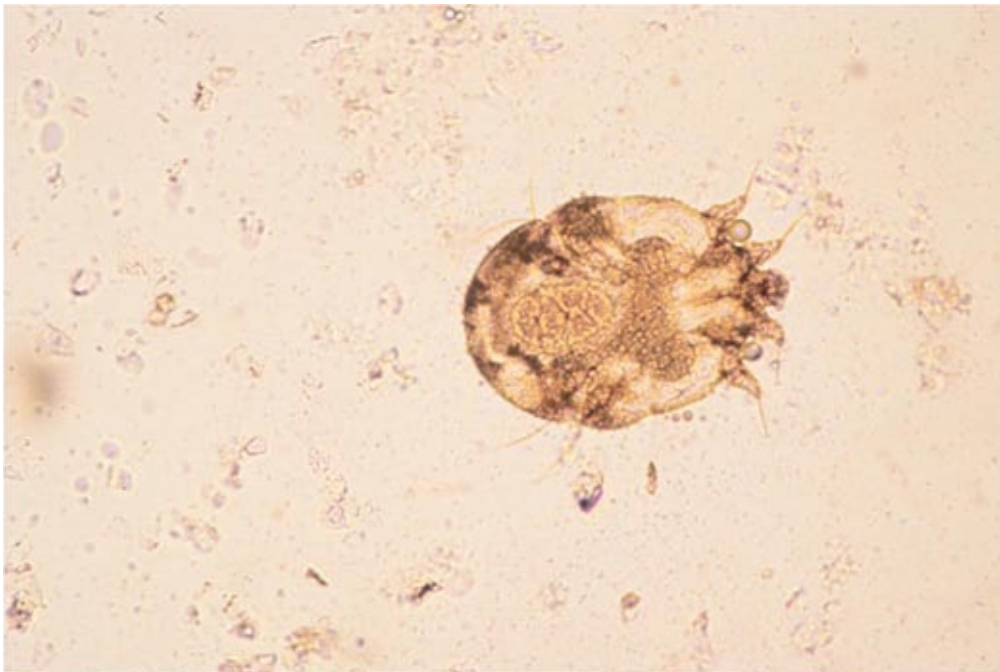


FIGURE 25.22 ■ Scabies. Adult female scabies mite. (Reproduced, with permission, from Morse SA, Ballard RC, Holmes KK, Moreland AA (eds). *Atlas of Sexually Transmitted Diseases and AIDS*. 3rd ed. Philadelphia, PA: Mosby; 2003. Copyright © 2003 Elsevier.)



FIGURE 25.23 ■ Pediculosis. *Phthirus pubis*, the crab louse. Note the short body and claw-like legs, which are ideally suited for clinging to the hair shaft. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)



FIGURE 25.24 ■ Pediculosis. *Phthirus corporis*, the body louse. Note the elongated body. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)

CEREBROSPINAL FLUID EXAMINATION—INDIA INK PREPARATION FOR *CRYPTOCOCCUS NEOFORMANS*

Uses

To examine cerebrospinal fluid for organisms with capsules, particularly *Cryptococcus neoformans*.

Materials

India Ink, glass microscope slide, and coverslip.

Method

1. Lightly centrifuge cerebrospinal fluid to concentrate cells at bottom of tube (1-2 minutes).
2. Pour off excess fluid (retain if further testing may be necessary).
3. Take a drop from the bottom of the centrifuge tube and place it in the middle of a glass microscope slide.
4. Place a drop of India Ink into the specimen drop; gently mix.
5. Overlay a coverslip.
6. Examine at $\times 10$ to screen specimen; use $\times 40$ objective to confirm findings.

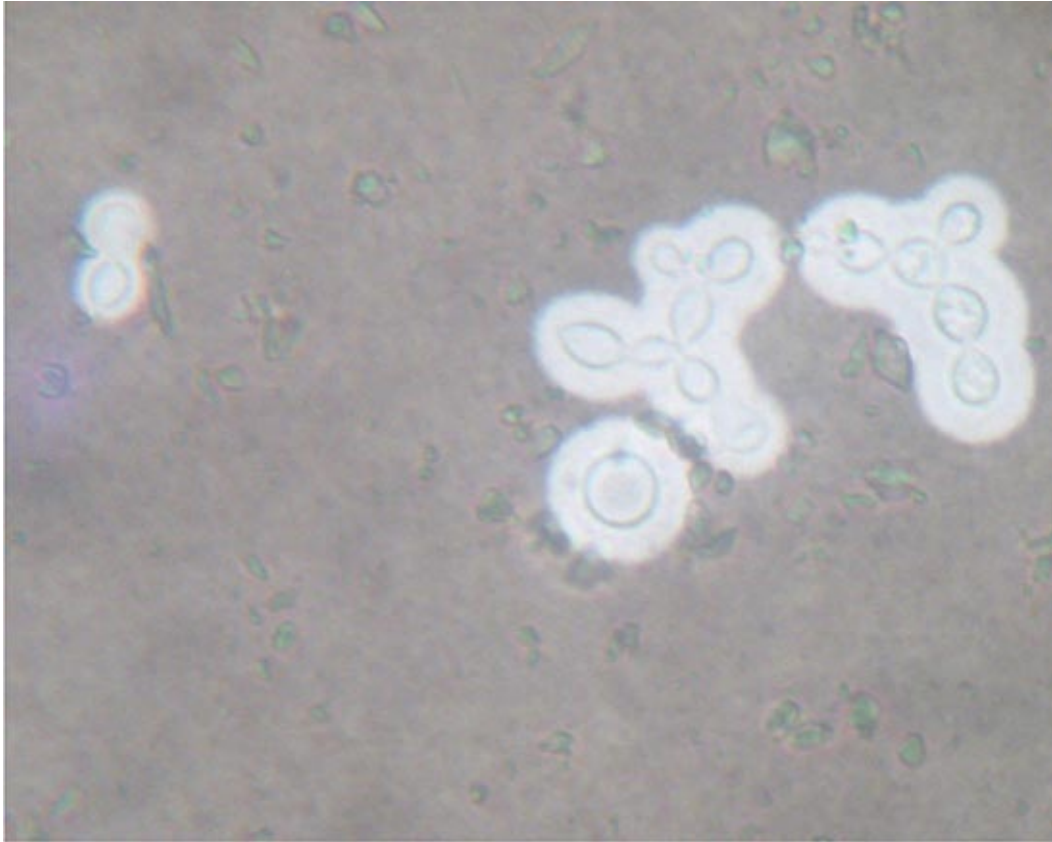


FIGURE 25.25 ■ India Ink Preparation. Budding yeast with prominent capsule on India Ink preparation from a patient with *C neoformans* meningitis. (Photo contributor: Seth Wright, MD.)

WRIGHT STAIN—THIN SMEAR FOR MALARIA

Uses

To evaluate for the presence of ring trophozoites.

Materials

Air-dried blood smear, Coplin jar of Wright stain, slide rack, pH 7.2 buffer, and blotting paper.

Method

1. Place a drop of blood on the middle of a slide.

2. Hold another slide evenly on top of the slide at a 45-degree angle and drag the slide over the drop of blood to the opposite edge to spread the blood evenly.
3. Allow the blood to dry for 5 to 10 minutes.
4. Stain air-dried smears in a closed Coplin jar of Wright stain for 5 minutes.
5. Place the slide on a rack.
6. Rinse and treat with pH 7.2 buffer primed with 1 mL Wright stain per 400 mL for 3 minutes.
7. Rinse in pH 7.2 buffer for 20 seconds.
8. Blot dry and mount on microscope at $\times 100$ (oil immersion).

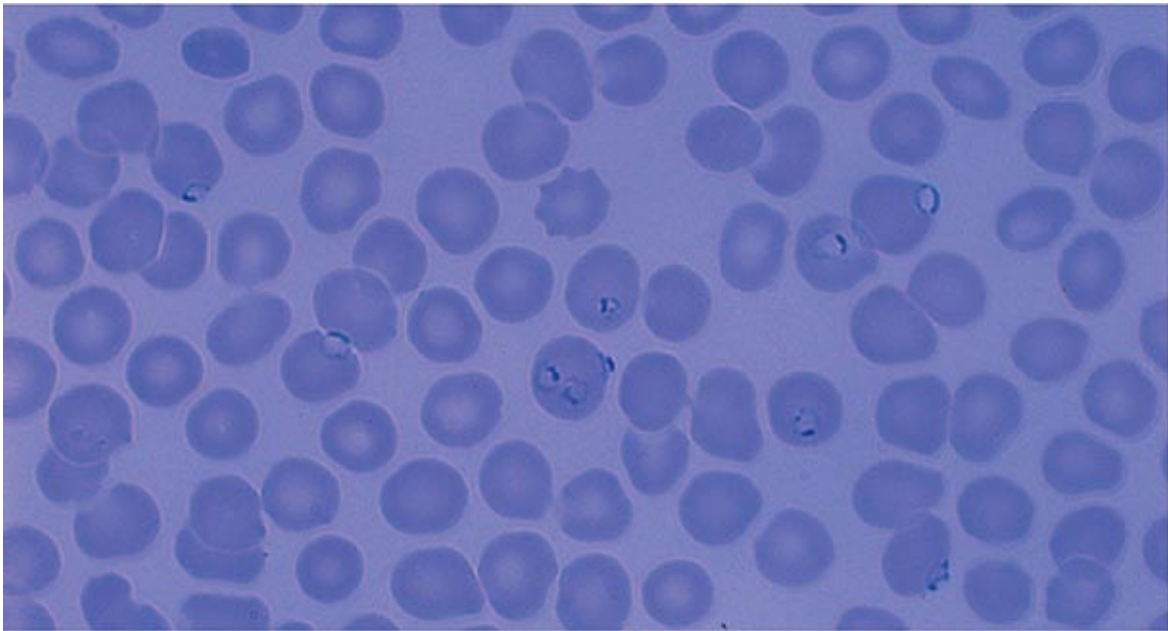


FIGURE 25.26 ■ *Plasmodium falciparum* Thin Film. Ring forms (trophozoites) of *P falciparum* are seen on the Wright stain thin film in a patient with intermittent fever who had recently traveled to Africa. (Photo contributor: James P. Elrod, MD, PhD.)

FERNING PATTERN FOR AMNIOTIC FLUID

Uses

To distinguish between amniotic fluid due to membrane rupture and normal vaginal secretions in patients beyond the 20th week of pregnancy who present with spontaneous vaginal fluid passage. Characteristic arborization, or typical ferning pattern, confirms amniotic fluid and spontaneous membrane rupture.

Materials

Sterile speculum, vaginal fluid, microscope slide, microscope, and sterile swab or pipette.

Method

1. Place patient in dorsal lithotomy position.
2. Do not use lubricants or cleaning agents.
3. Place sterile speculum into vaginal vault.
4. Obtain sample of vaginal secretions from posterior vaginal pool using a pipette or sterile swab.
5. Place a drop of vaginal fluid on a microscope slide.
6. Spread the specimen evenly so that a thin smear is formed.
7. Allow the fluid to air dry for 5 to 10 minutes. Do not apply heat.
8. Examine slide under low power (10×) for ferning pattern.

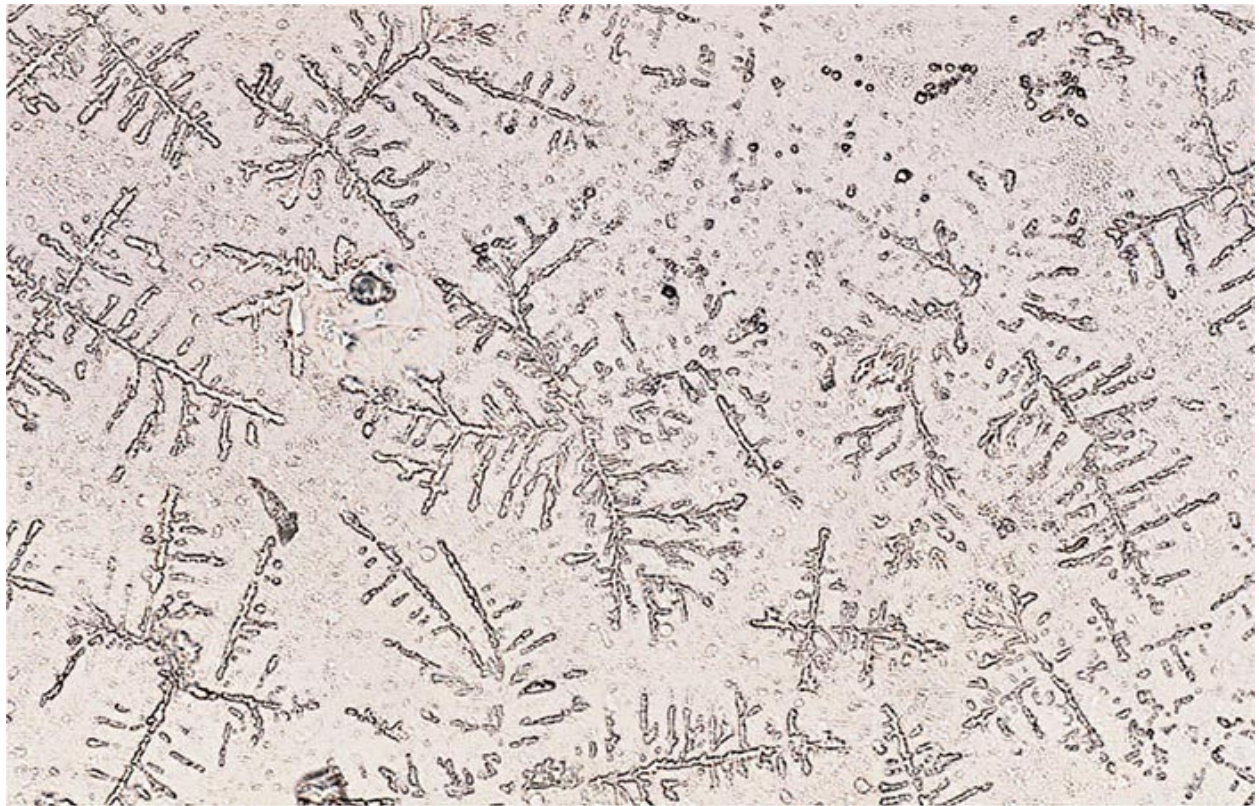


FIGURE 25.27 ■ Ferning Pattern. The arborization pattern found when a drop of amniotic fluid is allowed to air dry on a microscope slide, known as ferning. (Photo contributor: Robert Buckley, MD.)

PERIPHERAL BLOOD SMEAR

Uses

To detect the presence of abnormal hematologic cells such as schistocytes or blast cells in a peripheral blood smear as well as to observe the morphology of such cell lines and any abnormalities associated with them such as in the case of sickle cell disease.

Schistocytes are fragmented red blood cells due to shearing forces in microarterioles lined or meshed with fibrin strands. They are found in patients with disseminated intravascular coagulation, thrombotic thrombocytopenic purpura/hemolytic uremic syndrome, microangiopathic hemolytic anemia, uremia, and carcinoma. Turbulent blood flow due to congestive heart failure, artificial heart valves, or valvular stenosis may cause schistocyte formation. Greater than 1% of forms or greater than two schistocytes per high-powered field suggests schistocytosis.

Peripheral blood smear blasts are circulating immature white blood cells. When elevated white blood cell count and circulating blasts are seen in addition to anemia and thrombocytopenia, acute leukemia is suspected. As leukemia blast cells build up in the bone marrow, they allow less room for production of healthy white blood cells, red blood cells, and platelets. Although the diagnosis of leukemia can usually be made from the peripheral smear, bone marrow examination (aspiration or needle biopsy) is routinely done for definitive diagnosis.

Materials

Two glass microscope slides, drop of blood, pipette, Wright stain, Giemsa stain, immersion oil, and microscope.

Method

Smear Preparation

1. Agitate sample well, by inversion of tube or mechanical rocker.
2. Place a 2- to 3-mm drop of whole blood $\frac{1}{4}$ inch from the right edge of a 1×3 inch slide using a wooden applicator stick.

3. Place the slide on a flat surface and hold securely.

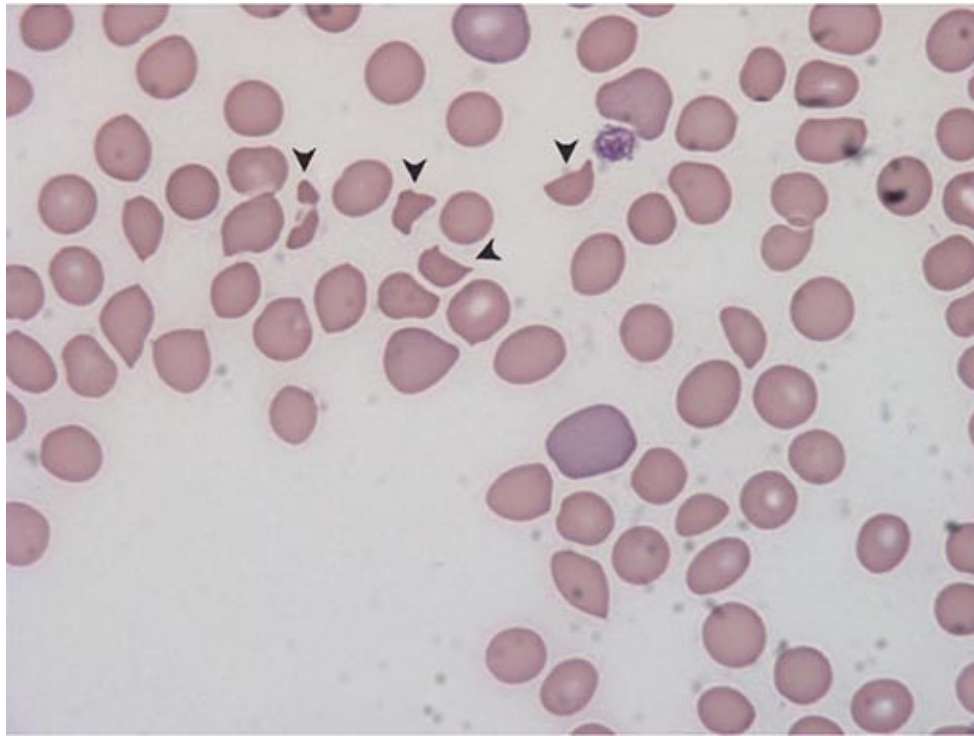


FIGURE 25.28 ■ Schistocytes. Multiple fragmented red blood cells (black arrowheads) seen on the peripheral smear at $\times 100$ in a patient with microangiopathic hemolytic anemia. (Photo contributor: James P. Elrod, MD, PhD.)

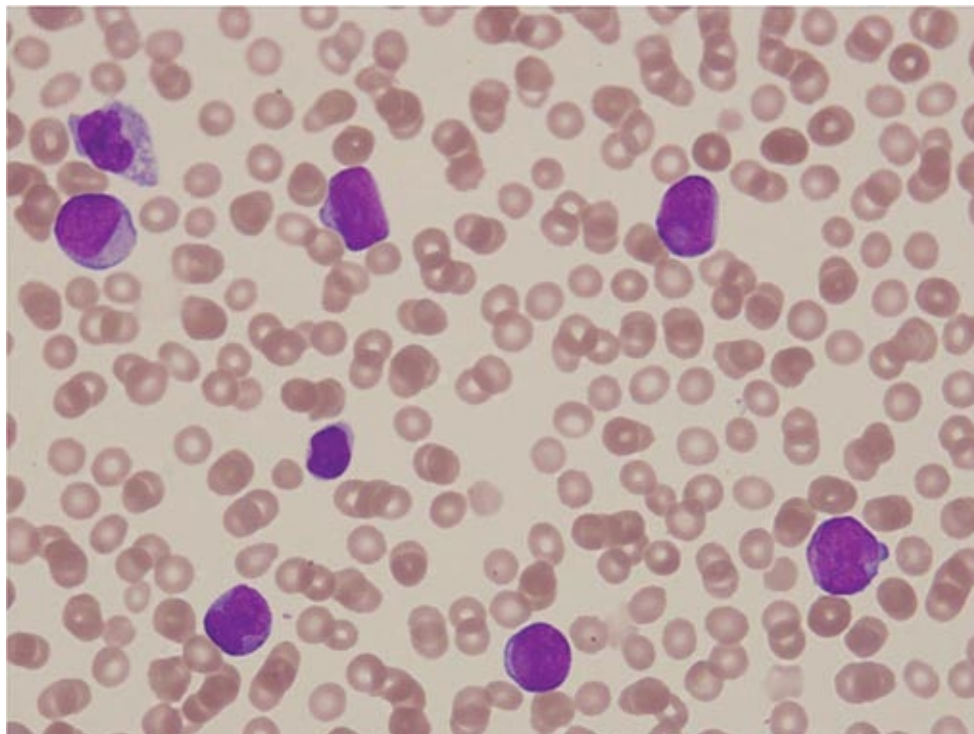


FIGURE 25.29 ■ Acute Myeloid Leukemia (AML) Blast Cells. Wright stained peripheral blood smear, $\times 1000$. Multiple blast cells seen in this smear from a patient with shortness of breath and a white blood cell count of 105 K/ μ L, 80% blasts, hematocrit 20%, and platelet count of 75 K/ μ L. Flow cytometry confirms these are myeloid blasts, confirming AML. (Photo contributor: Mary Ann Thompson Arildsen, MD, PhD.)

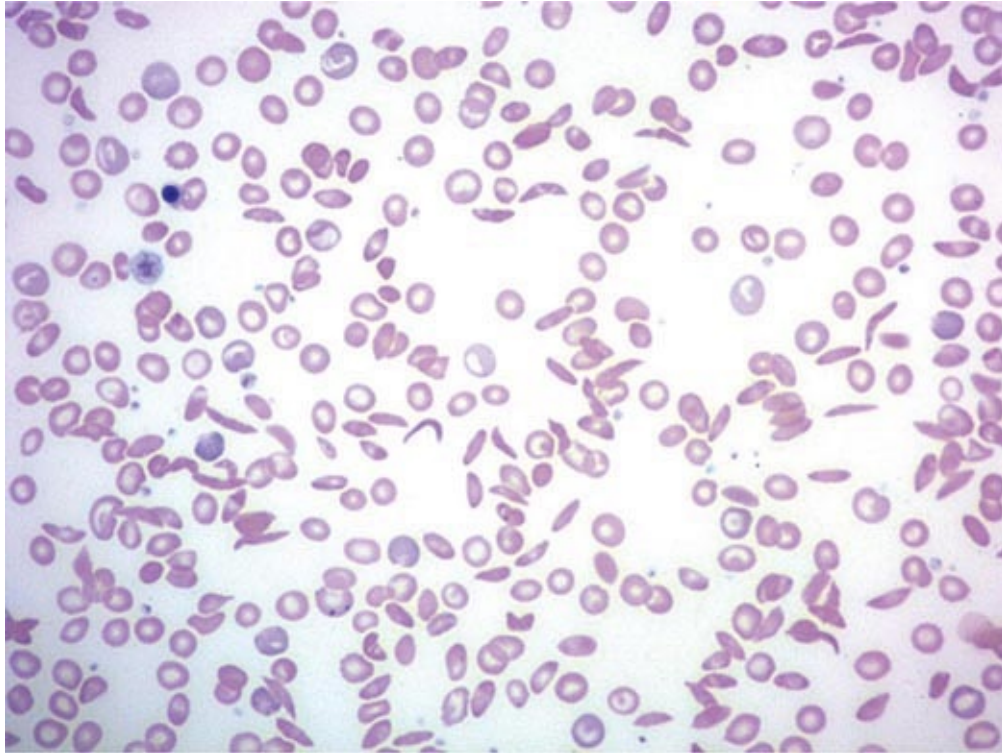


FIGURE 25.30 ■ Sick Cell ($\times 400$, Wright Stain). Sick cells, also called drepanocytes, are pointed at both ends and do not show central pallor. Notice also target cells, increased reticulocytes (the cells that are larger and slightly bluer in color), and the nucleated red blood cell in the top left. (Photo contributor: Jennifer Knight, MD.)

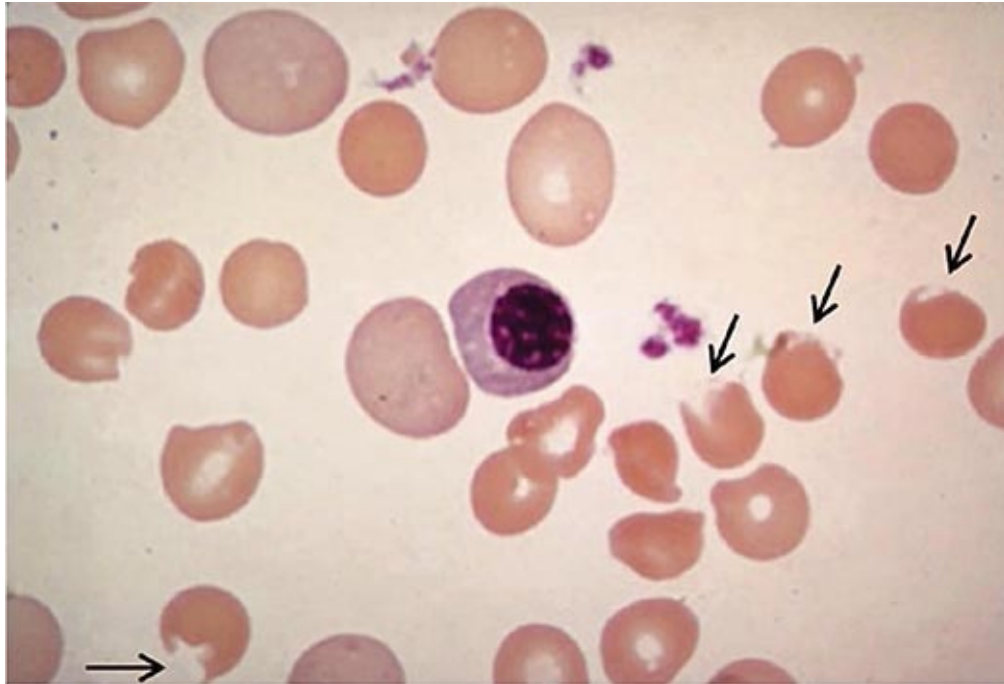


FIGURE 25.31 ■ Glucose-6-phosphate dehydrogenase (G6PD) deficiency. (Peripheral blood smear, 100x.) Several features of G6PD deficiency in the setting of an oxidative challenge are shown. The polychromatophils (large, bluish young red blood cells) and nucleated red blood cells indicate the current hemolytic state. Also shown are “bite” cells (arrows), which are red blood cell morphologic changes that are the consequence of macrophage action on the Heinz bodies, which have precipitated in the inner leaflet of the red blood cell membranes. (Photo contributor: Lloyd E. Damon, MD. From Papadakis MA, McPhee SJ, Rabow MW. *Current Medical Diagnosis & Treatment* 2021. New York, NY: McGraw Hill; 2021.)

4. Grasp a 2nd slide (spreader slide) in the right hand between thumb and forefinger.
5. Place the spreader slide onto the lower slide in front of the blood drop, and pull the slide back until it touches the drop.
6. Allow the blood to spread by capillary action almost to the edges of the lower slide.
7. Push the spreader slide forward at a 30-degree angle, using a rapid even motion. The weight of the spreader slide should be the only weight applied. The drop of blood must be spread within seconds or the cell distribution will be uneven.
8. Allow to air dry.

Wright Stain of Peripheral Smear

1. Completely cover peripheral smear with Wright stain using a pipette. The stain layer should be $\frac{1}{8}$ inch thick.
2. Wait for 2 minutes.

3. Cover with equal amount of Giemsa solution.
4. Blow gently on the slide to mix solutions.
5. Allow to stand for 4 minutes.
6. Wash slide for 30 seconds with distilled water.
7. Allow to air dry.
8. View smear with oil emersion at high ($\times 100$) power.

TAPE TEST FOR *ENTEROBIUS VERMICULARIS* EGGS

Uses

To detect the presence of eggs of *Enterobius vermicularis* in patients who present with nocturnal perianal pruritis or concern for pinworm infestation.

Materials

Microscope slide, clear transparent tape, tongue blade, microscope, or a cellulose-tape slide preparation.

Method

1. Affix $\frac{1}{2}$ inch of a 4-inch piece of tape to the underside of a microscope slide (see [Fig. 25.33A](#)).
2. Hold the slide against the tongue depressor 1 inch from the end and lift the long end of the tape from the slide (see [Fig. 25.33B](#)).
3. Loop tape over end of the depressor to expose gummed surface (see [Fig. 25.33C](#)).
4. Hold tape and slide firmly against the tongue depressor (see [Fig. 25.33D](#)).
5. Press gummed surfaces of the tape against several areas of the perianal area (see [Fig. 25.33E](#)).
6. Affix long portion of the tape to the slide (see [Fig. 25.33F](#)).
7. Smooth tape with cotton gauze (see [Fig. 25.33G](#)).
8. View specimen under low ($\times 10$) power.

Note: Specimens are best obtained several hours after going to sleep or upon waking before a bowel movement or bath.



FIGURE 25.32 ■ *Enterobius vermicularis*. Characteristic appearance of the *E. vermicularis* egg with contained larval form.

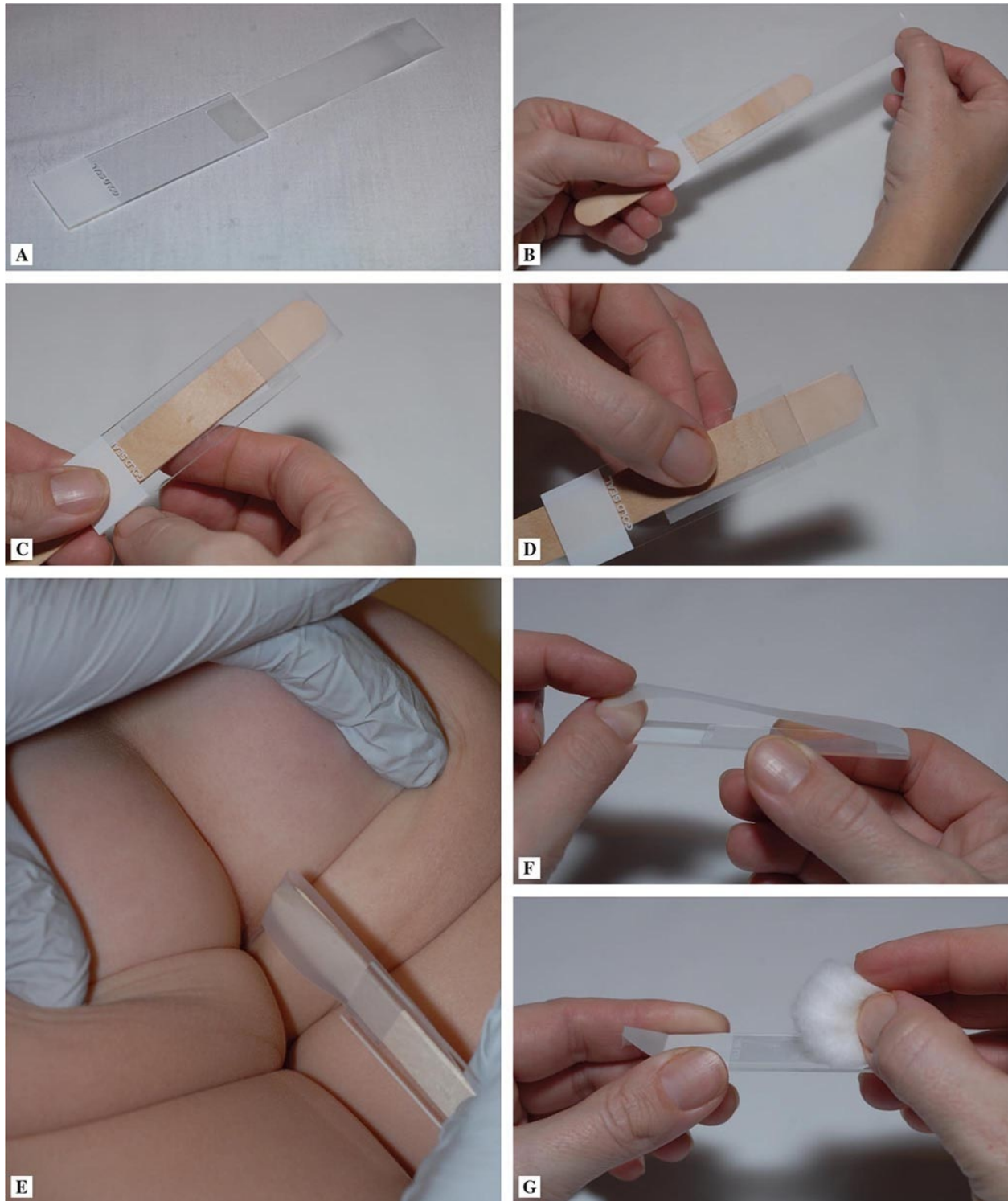


FIGURE 25.33 ■ Cellophane Tape Preparation. Steps for obtaining the eggs of *E. vermicularis*. See text for details. (Photo contributor: Lawrence B. Stack, MD.)

TZANCK PREPARATION FOR HERPES INFECTION

Uses

To detect the multinucleated giant cells confirming the presence of a herpes infection.

Materials

Microscope, Bunsen burner, glass microscope slide, 5% methylene blue or Wright stain or Giemsa stain, immersion oil, and sterile scalpel or hypodermic needle.

Method

1. Unroof a fresh, uncrusted vesicle with a sterile hypodermic needle or scalpel.
2. Scrape the floor of the vesicle with the scalpel and smear scrapings of the lesion onto a glass microscope slide.
3. Let air dry.
4. Fix specimen with absolute alcohol or gentle heat.
5. Stain with blue stain (5% methylene blue, Wright stain, or Giemsa stain) for 5 seconds, rinse, and air dry.
6. View preparation through immersion oil at high power ($\times 40-50$).

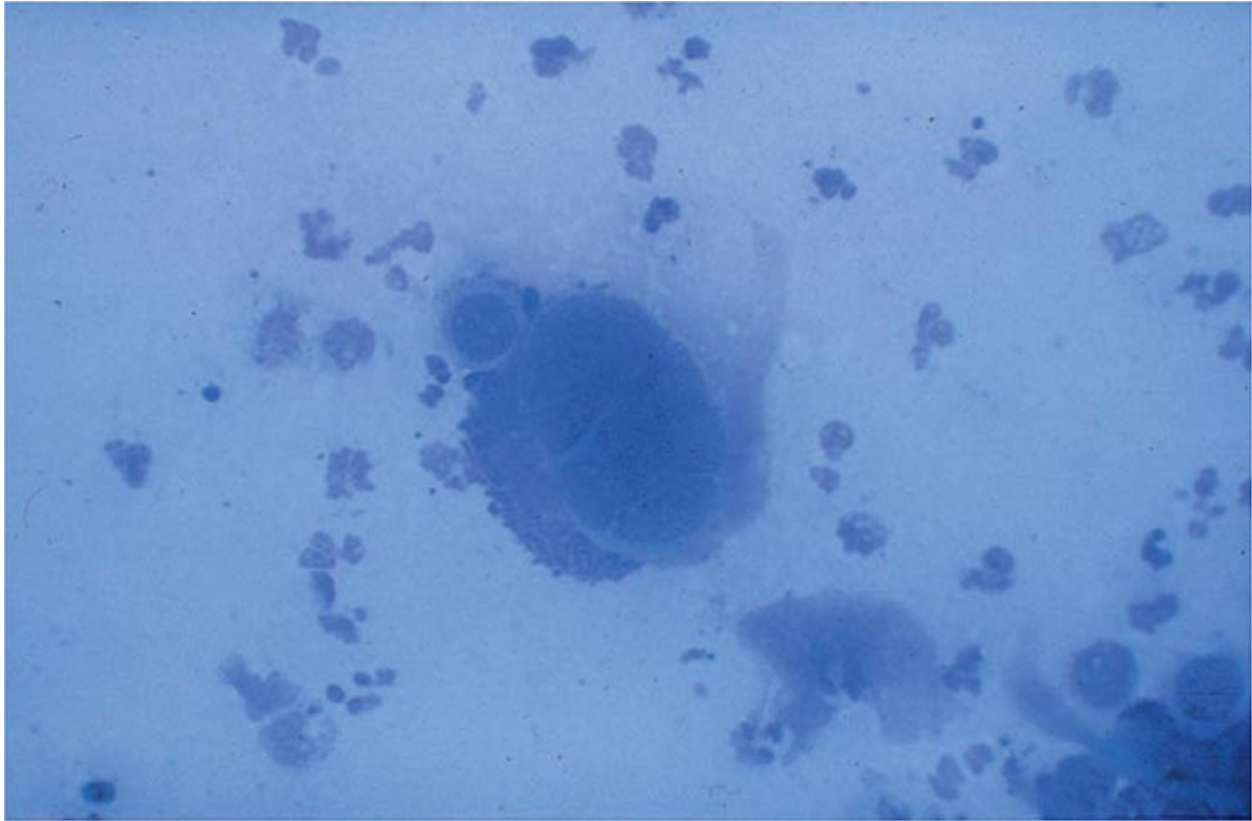


FIGURE 25.34 ■ Tzanck Preparation. A Tzanck preparation of both the roof and floor of a herpetic vesicle demonstrating a multinucleated giant cell. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brook Army Medical Center, San Antonio, TX.)

ACID-FAST STAIN FOR *MYCOBACTERIUM*

Uses

The purpose of this staining method is to differentiate between acid-fast and non-acid-fast bacteria. This method is used for organisms not stained by the Gram stain method, particularly organisms of the genus *Mycobacterium*. This is called the Ziehl-Neelsen staining technique.

Materials

Glass slide, carbol fuchsin stain, 3% acid alcohol, and malachite green stain.

Method

1. Prepare bacterial smear on a clean slide using sterile technique.
2. Allow smear to air dry and then heat fix.
3. Cover the smear with carbol fuchsin stain.
4. Heat the stain until vapor just begins to rise (ie, about 60°C). Do not overheat. Allow the heated stain to remain on the slide for 5 minutes.
5. Wash off the stain with water.
6. Cover the smear with 3% acid alcohol for 5 minutes or until the smear is sufficiently decolorized (ie, pale pink).
7. Wash well with water.
8. Cover the smear with malachite green stain for 1 to 2 minutes, using the longer time when the smear is thin.
9. Wash off the stain with clean water.
10. Wipe the back of the slide clean, and place it in a draining rack for the smear to air dry (do not blot dry).
11. Examine the smear microscopically, using the $\times 100$ oil immersion objective.

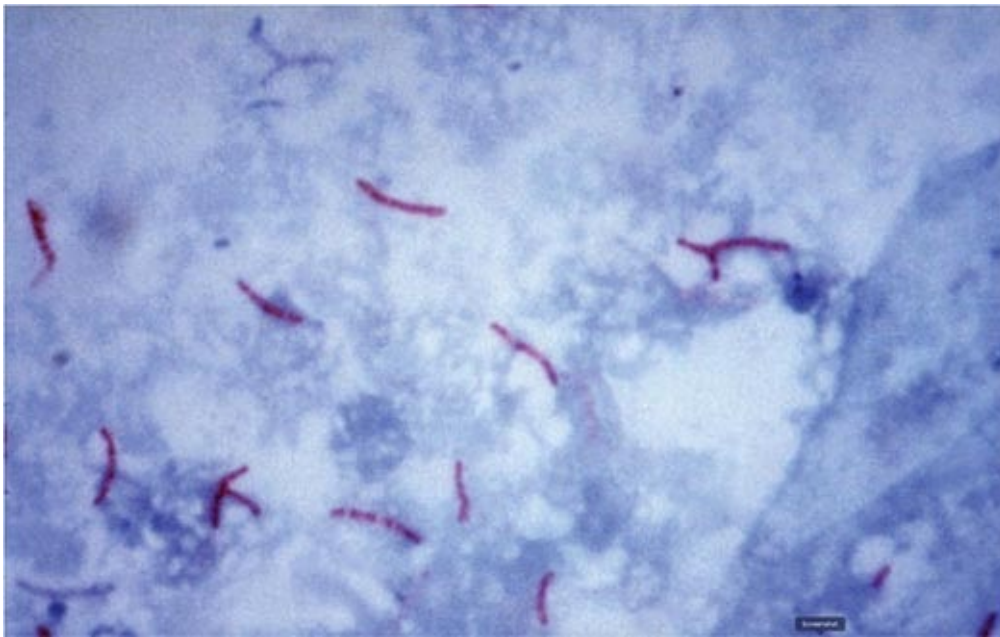


FIGURE 25.35 ■ Acid-Fast Bacilli. *Mycobacterium tuberculosis* seen on Ziehl-Neelsen staining technique for acid-fast bacilli. (Photo contributor: George P. Kubica, PhD. CDC Public Health Library. PHIL ID# 5789.)

Bodily Fluids

STOOL

Clinical Summary

Stool characteristics that cause angst in patients and parents prompting emergency department visits include changes in color (black, red, white, green, blue), consistency (hard, loose, watery), or frequency (too often, too infrequent) and associated pain.

Melena is a dark maroon (see [Fig. 25.36](#)) to black tar-like (see [Fig. 25.37](#) and [Fig 25.38](#)) stool typically due to upper gastrointestinal (GI) bleeding of 100 to 200 mL of blood. Melena can occur from lower GI bleeds with delayed transit, swallowed blood from maternal mammary bleeding, epistaxis, or hemoptysis. Substances that cause black stool that are negative for occult blood include iron supplements, bismuth preparations, activated charcoal, and blueberries.



FIGURE 25.36 ■ Melena and Maroon Stool. The maroon watery stool with solid melena suggests a brisk bleeding upper gastrointestinal bleed. Esophagogastroduodenoscopy confirmed a briskly bleeding gastric ulcer. (Photo contributor: Maria Ladino, MD.)



FIGURE 25.37 ■ Melena. The black tarry appearance of melena in a patient with a duodenal ulcer. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 25.38 ■ Melena. Guaiac-positive stool specimen in a patient with melena due to a duodenal ulcer. The fecal occult blood test card contains white paper impregnated with guaiac resin, which turns blue in the presence of hemoglobin after adding hydrogen peroxide (Photo contributor: Lawrence B. Stack, MD.)

Bright red bloody stool (hematochezia) suggests lower GI bleeding and can be caused by colonic polyps, colon cancer, diverticulosis, anal fissures,

hemorrhoids, inflammatory bowel disease, aortoenteric fistula, arteriovenous malformations, infectious diarrhea, and coagulopathy. Causes in children include necrotizing enterocolitis, malrotation with midgut volvulus, Hirschsprung disease, milk-protein allergy, intussusception (see [Fig. 25.39](#)), Meckel diverticulum, GI duplication cysts, vasculitis, hemolytic uremic syndrome, rectal ulcers, and juvenile polyps (see [Fig. 25.40](#)). Causes of red but not bloody stool include cefdinir (see [Fig. 25.41](#)), spicy food dyes, red licorice, beets, and red-colored food dyes.



FIGURE 25.39 ■ Currant Jelly Stool. Stool mixed with blood and mucus, referred to as “currant jelly” stool, seen in this patient with intussusception. Bloody stools are a late finding in intussusception and occur from bowel ischemia and injury of the entrapped bowel segment. (Photo contributor: Donald H. Arnold, MD.)



FIGURE 25.40 ■ Juvenile Polyp. Note blood mixed with normal stool in a 3-year-old patient who passed this juvenile polyp. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 25.41 ■ Cefdinir Stool. Cefdinir combines with iron to form a precipitate that gives stool a characteristic discoloration that is guaiac negative. (Photo contributor: Lawrence B. Stack, MD.)

Acholic (white or pale colored) stools (see [Fig. 25.42](#)) are most commonly caused by biliary system disease. Normally the liver releases bile salts into the gut, causing a brown color. Diseases include hepatitis, cirrhosis, liver tumors, bile duct cysts, gallstones, sclerosing cholangitis, and biliary atresia.



FIGURE 25.42 ■ Acholic Stool. Clay-colored stools lacking bile pigment seen in biliary atresia. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Unstable (orthostatic, acute anemia, hypotensive, symptomatic) patients with upper or lower GI bleeding should be admitted to the intensive care unit (ICU) for resuscitation and endoscopy. Stable patients may be admitted to the floor. Patients with black or red stools not caused by GI bleeding should be reassured and discharged with instructions to avoid the offending agent.

Healthy-appearing and asymptomatic patients with one to two acholic stools can be observed for recurrence. Otherwise, laboratory and imaging studies focusing on biliary disease should be performed in the emergency department.

Pearls

1. Ingestion of rare red meat may cause a false-positive fecal occult blood test.
2. Melena can be caused by as little as 60 mL of upper GI bleeding.
3. Biliary atresia is the most common cause of conjugated hyperbilirubinemia in resource-rich countries.
4. Stercobilin is the bile pigment that gives stool its normal brown appearance.
5. “Melenic” pertains to melena or black stool. “Melanotic” pertains to melanin or skin pigment.

Clinical Summary

Emesis characteristics that prompt evaluation for life-threatening causes include bilious emesis, hematemesis, projectile vomiting, feculent emesis, prolonged emesis (> 12 hours in a neonate, > 24 hours in children < 2 years old, or > 48 hour in older children), associated mental status change, bradycardia, hypertension, trauma, bulging fontanelle, seizures, abdominal distention, and diminished urine output.

Hematemesis is bloody (see [Fig. 24.43](#)) or coffee ground– appearing emesis. Life-threatening causes include esophageal variceal bleeding, swallowed blood from posttonsillectomy bleeding, esophageal rupture, aortoenteric fistulas, epistaxis, arteriovenous malformations, peptic ulcer disease, and upper GI tumors. Volume of blood loss from hematemesis is often overestimated by patients and healthcare workers.



FIGURE 25.43 ■ Hematemesis. Vomiting of clotted blood 4 days after a tonsillectomy and adenoidectomy. (Photo contributor: Lawrence B. Stack, MD.)

Bilious (green) vomiting (see [Fig. 25.44](#)) in newborns suggests malrotation with midgut volvulus, a life-threatening surgical emergency. Malrotation occurs because the normal embryological sequence of bowel development, rotation, and fixation within the abdomen is not completed. Malrotation creates an environment where the intestines of the mid-gut can twist on themselves, causing an immediate bowel obstruction and intestinal ischemia.

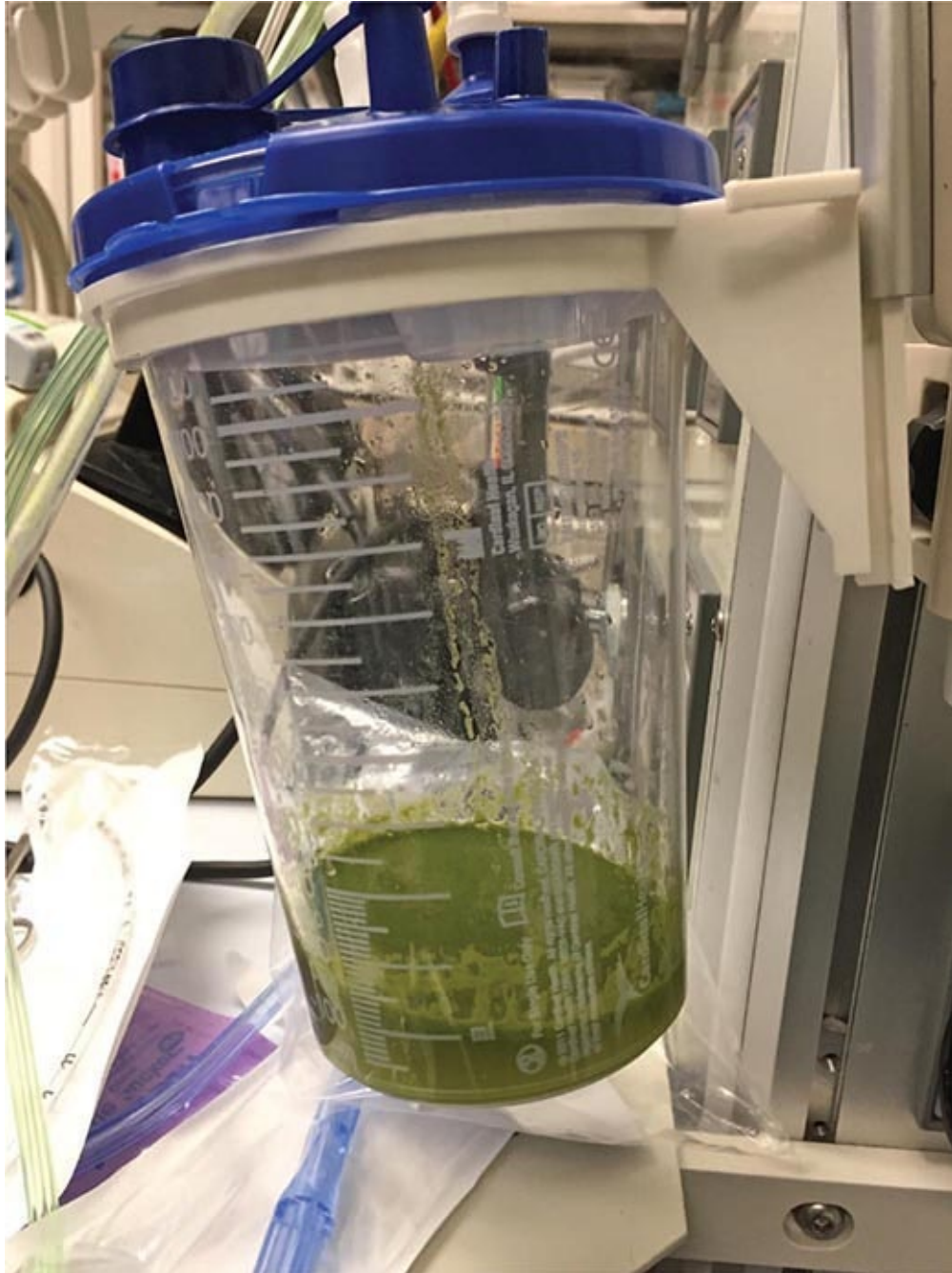


FIGURE 25.44 ■ Bilious Emesis. Emesis with green color indicating larger amounts of bile in the stomach often associated with intestinal obstruction. (Photo contributor: Jaime Kaye Otillo, MD.)

Feculent vomiting (see [Fig. 25.45](#)) is an unusual symptom typically caused by a mechanical intestinal obstruction, but can be due to an adynamic ileus. Other causes include gastrocolic fistula, coprophagy, and violent reverse peristalsis.



FIGURE 25.45 ■ Feculent Emesis. Vomit of fecal origin associated with a gastrocolonic fistula or ileal obstruction. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Unstable (orthostatic, acute anemia, hypotensive, symptomatic) patients with upper GI bleeding should be admitted to the ICU for continued resuscitation and endoscopy. Stable patients may be admitted to the floor. Consider octreotide,

ceftriaxone, pantoprazole, and blood products in unstable patients with variceal bleeds. Linton, Blakemore, and Minnesota tubes are temporary balloon tamponade devices that buy time until patients are stable for endoscopy. Intubation for airway protection is frequently needed in severe variceal bleeds. Patients with Mallory-Weiss tear bleeding may be discharged from the emergency department after a period of observation without continued hematemesis.

Bilious emesis in the infant should prompt immediate pediatric surgery consultation while the evaluation and fluid resuscitation are ongoing. Plain films of the abdomen should be initially obtained, and if there is no free air, an upper GI series should be obtained. Fluid resuscitation should be ongoing.

Pearls

1. Emesis of gastric fluid is yellow (see [Fig. 25.46](#)) and may be mistaken for bilious emesis.



FIGURE 25.46 ■ Gastric Content Emesis. Gastric content without food will typically have a yellow appearance and should not be confused with bilious emesis, which is green. (Photo contributor: Lawrence B. Stack, MD.)

2. Simultaneously obtain pediatric surgery consultation and workup for true bilious emesis.
3. Feculent emesis can be caused by coprophagy (ie, ingestion of dung).

4. Five neurotransmitter receptor sites are involved in the vomiting reflex: M1–muscarinic; D2–dopamine; H1–histamine; 5-hydroxytryptamine–serotonin; and neurokinin 1–substance P.

SPUTUM

Clinical Summary

Sputum is expectorant from the lungs and respiratory passages that contains mucus and pus and is a clue to the severity of respiratory illness. While microscopic examination provides the most accurate clues to illness, gross examination provides valuable information. Color, consistency, and volume are features that provide insight to the cause and severity of illness. Bloody sputum or hemoptysis (see [Fig. 25.47](#)) may occur in streaks or in life-threatening amounts. Acute bronchitis, pneumonia, lung cancer, tuberculosis, bronchiectasis, lung abscess, pulmonary embolism, and pulmonary infarction are causes of hemoptysis. Pink or white frothy sputum suggests pulmonary edema.



FIGURE 25.47 ■ Hemoptysis. Coughing up of blood or bloodstained mucus. (Photo contributor: Lawrence B. Stack, MD.)

Green sputum suggests chronic respiratory infection, as in chronic bronchitis, cystic fibrosis, and ruptured lung abscess. Rust-colored sputum suggests pneumococcal pneumonia, pulmonary embolism, lung cancer, or tuberculosis. Yellow sputum (see [Fig. 25.48](#)) in a healthy patient suggests viral bronchitis. However, in chronic obstructive pulmonary disease patients, yellow sputum may represent a bacterial infection where antibiotics are likely to be effective. Black

sputum (see [Fig. 25.49](#)) suggests pneumoconiosis and acute or chronic smoke exposure (as in seen fire fighters).



FIGURE 25.48 ■ Sputum—Viral Bronchitis. Yellowish-gray sputum seen in patients with viral bronchitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 25.49 ■ Carbonaceous Sputum. Smoke inhalation in a patient removed from a house fire. (Photo contributor: R. Jason Thurman, MD.)

Management and Disposition

Life-threatening hemoptysis, defined as greater than 150 mL in 24 hours should prompt computed tomography (CT) angiography of the chest and hospital admission. Causes of non-life-threatening hemoptysis should be treated accordingly.

Colored sputum will prompt a search for the cause of the finding. A chest x-ray or chest CT is likely to be helpful. A complete blood count, blood culture, and inflammatory markers may provide clues to the cause and guide treatment.

Pearls

1. Hypoxic patients with life-threatening hemoptysis from uni-lateral disease may benefit from a “bad lung down” position to keep bleeding confined to a single lung.
2. Neutrophil myeloperoxidase has a heme pigment that causes a green color in mucus and pus with a high neutrophil count.
3. Green or yellow sputum is a weak diagnostic marker for determining the presence of an acute pulmonary bacterial illness.

Clinical Summary

Although a urinalysis gives definitive microscopic clues to the presence of disease, gross appearance can provide useful clues prior to formal urinalysis results. Color, clarity, odor, and associated pain give insight to the presence of disease. Normal urine appearance ranges from transparent yellow to dark amber, which is due to urobilin, a bile pigment of hemoglobin. Dark urine (see [Fig. 25.50](#)) may be due to dehydration or high concentrations of bilirubin in the blood. Deep red or brown urine may suggest porphyria. Other causes of red urine include myoglobinuria due to hemolysis from brown recluse envenomation (see [Fig. 25.51](#)), rhabdomyolysis (see [Fig. 25.52](#)), hematuria from cancer (see [Fig. 25.53](#)), ureteral stones, clotting disorders, recent genitourinary surgery, urinary tract infection, sexually transmitted infection, or falsely present in females during menses. Blackberries, beets, rhubarb, rifampin, phenazopyridine, and senna can turn urine a red-orange color. Food dyes, methylene blue, indomethacin, and propofol may turn urine blue-green.

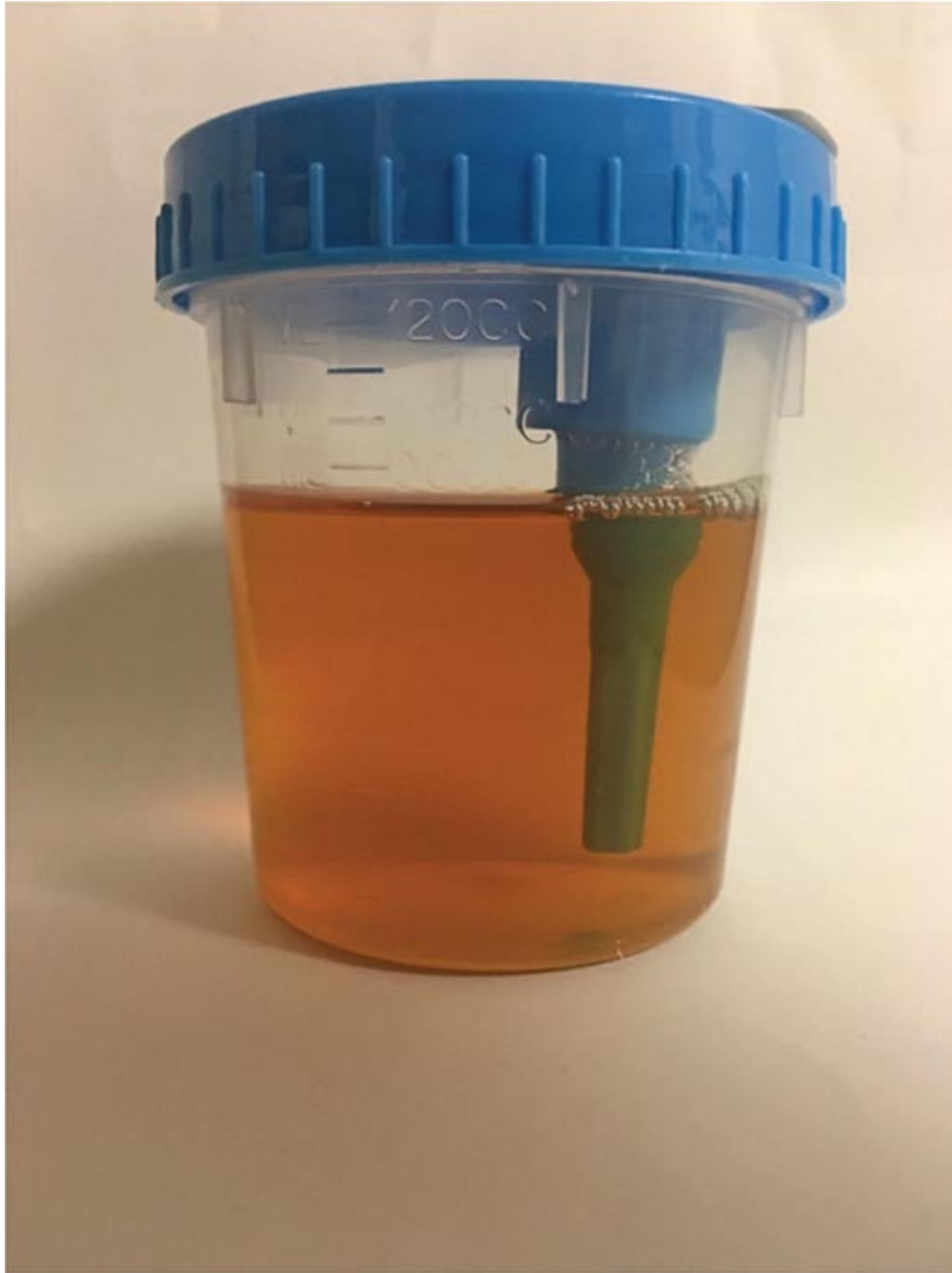


FIGURE 25.50 ■ Urinalysis—Dehydration. Dark transparent urine suggests dehydration in the correct clinical context. Dehydration was confirmed with formal urinalysis. (Photo contributor: Lawrence B. Stack, MD.)

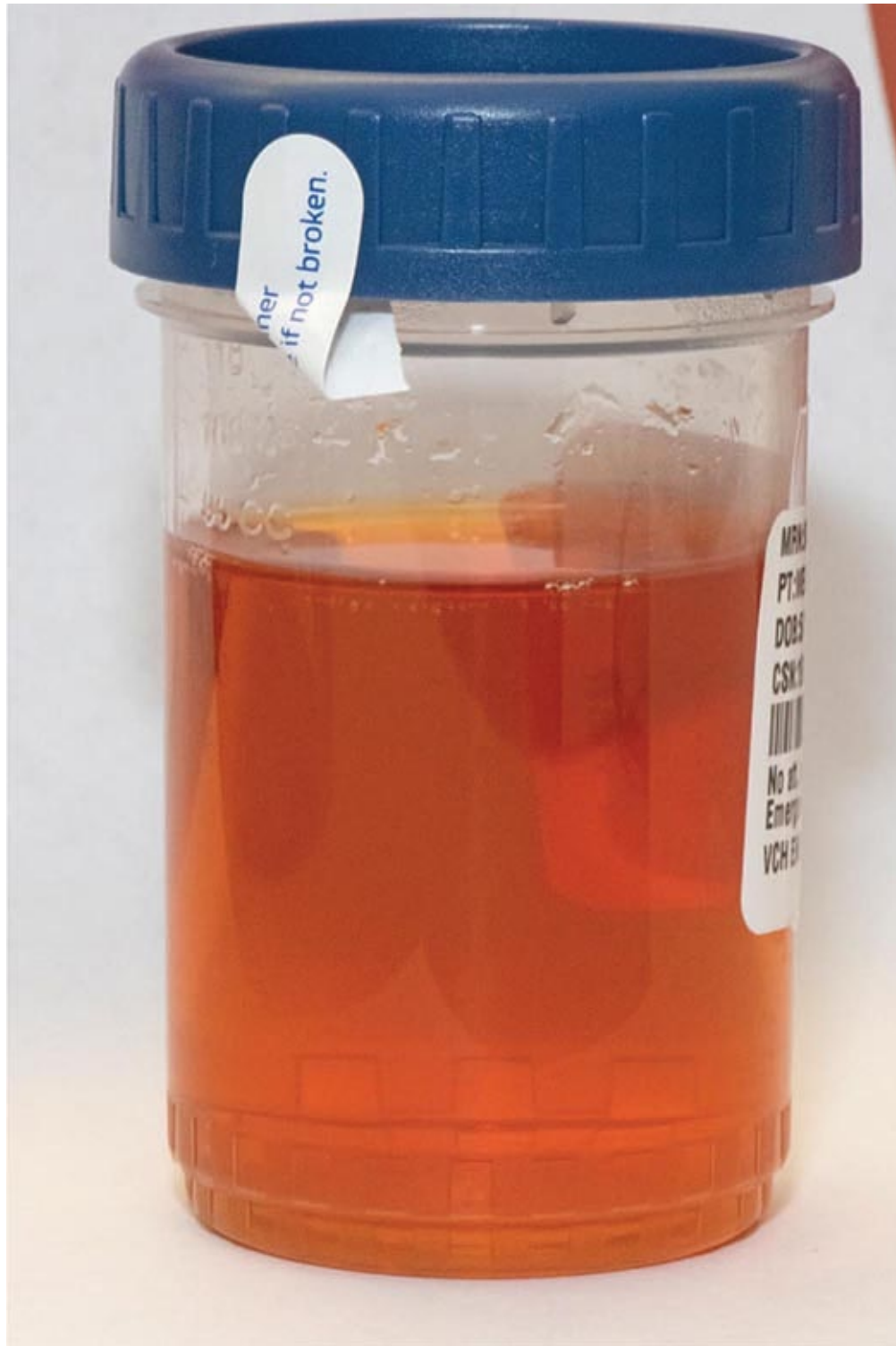


FIGURE 25.51 ■ Urine—Hemolysis due to Systemic Loxoscelism. Gross sample of hemoglobinuria as a result of an acute hemolysis seen in systemic loxoscelism after brown recluse spider bite. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 25.52 ■ Urine—Rhabdomyolysis. Gross sample of cola-colored urine in a patient during resuscitation after rhabdomyolysis due to a crush injury of the leg.



FIGURE 25.53 ■ Urine—Gross Hematuria. Spontaneous gross hematuria after initiation of apixaban. Patient was later found to have bladder cancer. (Photo contributor: Lawrence B. Stack, MD.)

Cloudy or turbid-appearing urine (see [Fig. 25.54](#)) is likely due to urinary tract

infections or ureteral stones.



FIGURE 25.54 ■ Urine—Urinary Tract Infection. Turbid or cloudy urine suggests infection, dehydration, or hematuria. Microscopic examination of this urine revealed too numerous to count white blood cells suggestive of a urinary tract infection. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Management varies depending on the cause of the urine discoloration.

Intravenous fluids for dehydration, forced diuresis for rhabdomyolysis, ICU admission to observe for ongoing hemolysis in brown recluse envenomations, CT scan to search for ureteral stones or cancer, and cystoscopy may be needed. Antibiotics are needed for urinary tract infections and sexually transmitted infections. An irrigation Foley catheter should be placed if blood clots are present and causing bladder outlet obstruction.

Pearls

1. Urobilin is the bile pigment that gives urine its yellow color.
2. Green urine can be caused by *Pseudomonas* infections.
3. Male sex, age greater than 50 years, family history of urinary tract disease, and strenuous exercise are risk factors for conditions that can cause change in urine color.

SYNOVIAL FLUID

Clinical Summary

Synovial fluid normally appears colorless or straw colored (see [Fig. 25.55](#)), viscous, and stringy. Arthrocentesis is performed in patients with a joint effusion or suspicion for joint inflammation or infection. Fluid can appear transparent (normal), translucent (mild inflammation), opaque (purulent inflammation), or bloody (traumatic tap, trauma, or bleeding disorders). Synovial fluid is normally viscous due to high levels of hyaluronic acid. These characteristics make a long “string” when a drop of normal fluid is expressed from a syringe.



FIGURE 25.55 ■ Synovial Fluid—Normal. Normal synovial fluid should be straw-colored fluid that is easy to see through. (Photo contributor: Amy C. Ramsay, MD.)

Management and Disposition

Patients with a septic joint (see [Fig. 25.56](#)) require orthopedic consultation and hospitalization. Joint washout is frequently necessary. Fat globules in bloody synovial fluid suggest an intraarticular fracture requiring orthopedics consult.

Gout (see [Fig. 25.57](#)) should be treated with steroids and pain medications.



FIGURE 25.56 ■ Synovial Fluid—Gout. Crystals were identified supporting the diagnosis of gout. Note the turbid, dark appearance suggesting inflammatory cells. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 25.57 ■ Synovial Fluid—Septic Joint. Purulent aspirate from the right hip is obtained. Cell count suggests and culture confirms a septic joint. (Photo contributor: Lawrence B. Stack, MD.)

Osteoarthritis should be treated with nonsteroidal anti-inflammatory drugs.

Pearls

1. Do not aspirate through an area of cellulitis or psoriasis to avoid seeding a joint with bacteria.
2. Do not perform arthrocentesis if the international normalized ratio (INR) is greater than 3 due to risk of precipitating a hemarthrosis.
3. The normal “string sign” of synovial fluid is lost with inflammatory conditions.

CEREBRAL SPINAL FLUID

Clinical Summary

Cerebral spinal fluid (CSF) is normally colorless and clear. Analysis provides clues to infectious diseases (meningitis, encephalitis, abscess) and noninfectious diseases (subarachnoid hemorrhage, Guillain-Barré syndrome, transverse

myelitis, idiopathic intracranial hypertension, vasculitis, carcinomatosis, multiple sclerosis, paraneoplastic syndromes). The presence of xanthochromia (see Fig. 25.58) in the context of recent onset of severe headache suggests a spontaneous subarachnoid hemorrhage. Turbid fluid (see Fig. 25.59) obtained from a patient with headache and neck stiffness suggests meningitis.



FIGURE 25.58 ■ Cerebral Spinal Fluid—Xanthochromia. Observed in this image is a sample of CSF with xanthochromia in a patient with subarachnoid hemorrhage. (Photo contributor: Shawna D. Bellew, MD.)



FIGURE 25.59 ■ Cerebral Spinal Fluid—Meningitis. Cloudy CSF in a patient confirmed to have bacterial meningitis as a result of *Streptococcus pneumoniae*. (Photo contributor: Scott H. Witt, MD.)

Management and Disposition

A patient with suspected subarachnoid hemorrhage requires blood pressure control and neurosurgery consultation for ICU admission. If obtunded, the patient should be intubated. Patients with suspected meningitis should be started

on intravenous antibiotics tailored to the suspected pathogen and admitted to the hospital.

Pearls

1. Xanthochromia is present in 90% of patient with a subarachnoid hemorrhage within 12 hours after the onset of bleeding.
2. As few as 200 white blood cells/mm³ and 400 red blood cells/mm³ will give CSF a turbid appearance.
3. Normal CSF production rate is 20 mL/h.
4. Lumbar punctures should not be performed with a platelet count of less than 50,000/mm³ or an INR of greater than 1.4 due to risk of developing a spinal hematoma.
5. Newer oral anticoagulants should be held for 48 hours prior to lumbar puncture.
6. Serum bilirubin greater than 15 mg/dL can cause CSF xanthochromia.

PERITONEAL FLUID

Clinical Summary

Cirrhosis, cancer, heart failure, tuberculosis, and dialysis account for 97% of cases of ascites, the accumulation of fluid within the peritoneal cavity.

Paracentesis is performed when the ascites is new or the cause of the ascites is unclear (diagnostic tap) or when the patient becomes symptomatic (abdominal pain, shortness of breath, fever) (therapeutic tap). Gross appearance of peritoneal fluid provides a clue to the cause of the ascites. Uncomplicated ascitic fluid in cirrhosis is transparent and yellow. Ascitic fluid can be completely clear if the bilirubin is normal. Turbid or cloudy fluid suggests infection (peritonitis) (see [Figs. 25.60](#) and [25.61](#)). Milky fluid has a high triglyceride level and is considered chylous ascites. Pink or bloody fluid is due to a “traumatic tap,” which clears over time, or malignancy, which is heterogeneous.



FIGURE 25.60 ■ Bacterial Peritonitis. Cloudy ascetic fluid in a patient with a new diagnosis of cirrhosis. Cell count of 375 polymorphonuclear lymphocytes (PMNs)/mL suggests bacterial peritonitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 25.61 ■ Bacterial Peritonitis. Slightly cloudy peritoneal dialysis fluid in a pediatric patient with 150 PMNs/mL suggests bacterial peritonitis in a peritoneal dialysis patient. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Spontaneous bacterial peritonitis (SBP) requires intravenous antibiotics and hospital admission. Patients undergoing paracentesis of greater than 5 L should consider albumin replacement of 6 to 8 g per liter of fluid removed.

Pearls

1. Cirrhosis is the cause of 80% of ascites.
2. A neutrophil count of greater than 250/mm³ suggests SBP, and antibiotics should be started.
3. A neutrophil count of greater than 100/mm³ suggests SBP in continuous ambulatory peritoneal dialysis patients.
4. Albumin infusion should be considered for paracentesis of greater than 5 L.
5. Albumin dose is 6 to 8 g per liter of fluid removed
6. Most SBP is caused by *Escherichia coli* and *Klebsiella*.

BLOOD

Clinical Summary

The appearance of blood in a tube from a peripheral blood draw can give a clue to disease. A specimen may demonstrate lipemia (see [Fig. 25.62](#)), which is defined as visible turbidity in serum samples due to lipoprotein particles, specifically chylomicrons. The most common cause of lipemia is hypertriglyceridemia.

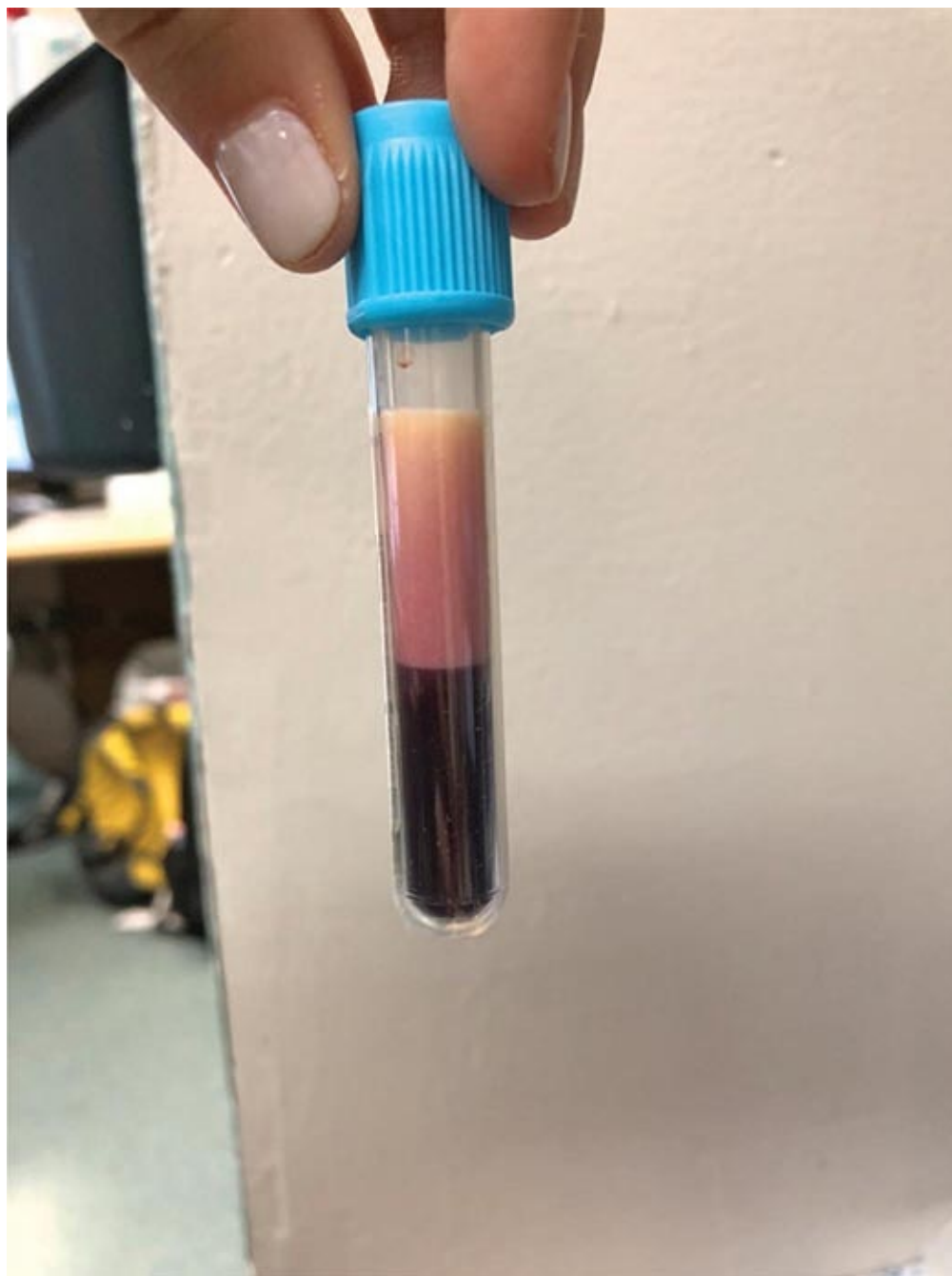


FIGURE 25.62 ■ Hypertriglyceridemia. Grossly lipemic serum sample seen in a patient with a triglyceride level of 9000 causing acute pancreatitis. (Photo contributor: Sarah Monks, MD.)

Methemoglobinemia results in a deep brown color of a peripheral blood draw due to the reduced oxygen-carrying capacity of methemoglobin. The most common cause is drug or toxin exposure. Medications notorious for causing methemoglobinemia include benzocaine, dapsone, amyl nitrite, sulfonamides, chloroquine, metoclopramide, and nitroprusside.

Management and Disposition

Patients with hypertriglyceridemia typically come to the emergency department because of consequences of the disease such as acute pancreatitis, myocardial infarction, or stroke.

Treatment of methemoglobinemia is intravenous methylene blue.

Pearls

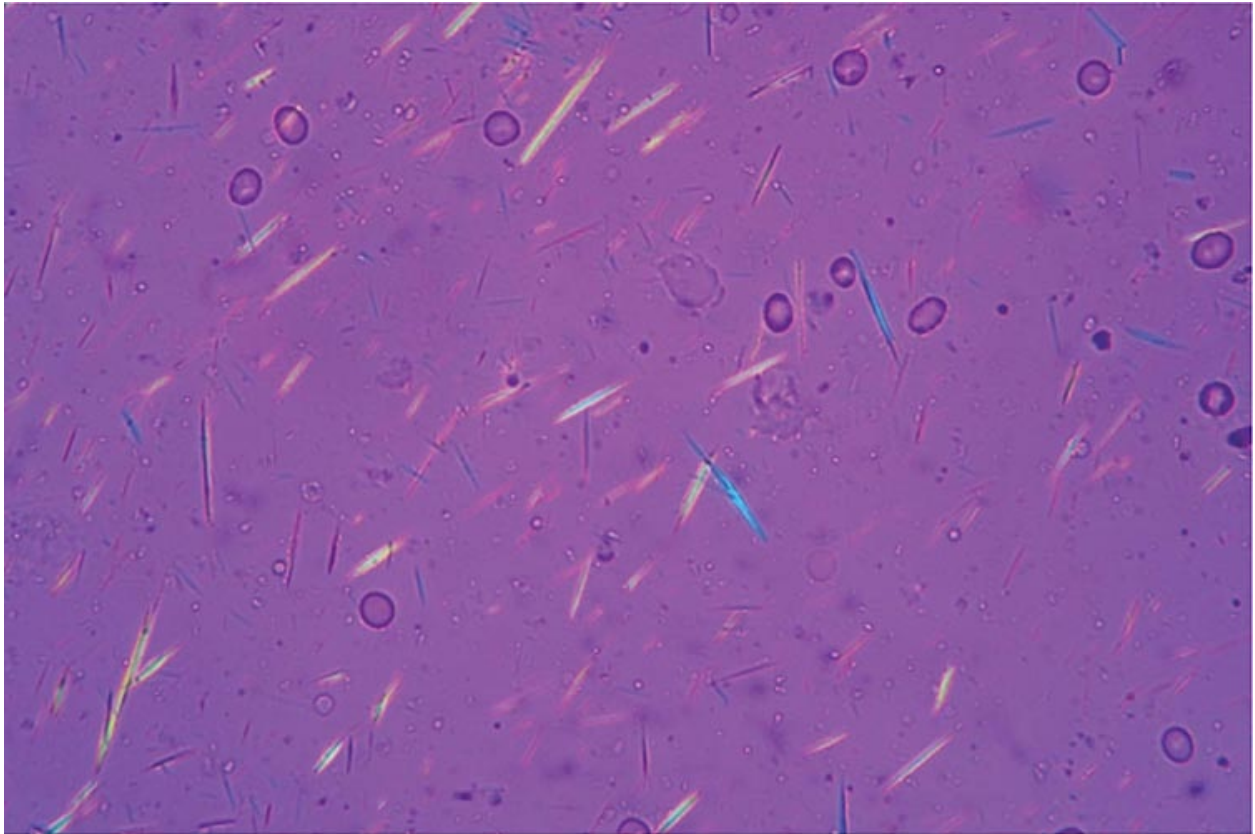
1. The most common cause of endogenous laboratory interference after hemolysis is lipemia.
2. Methemoglobin levels of 15% to 30% will cause cyanosis and brown blood.
3. Methylene blue, the antidote for methemoglobinemia, can cause methemoglobinemia.

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Chapter 26

RHEUMATOLOGIC CONDITIONS

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Uric Acid Crystals. (Photo contributor: Michael Bezzerides, MS4.)

GOUT

Clinical Summary

Gout is the most common form of inflammatory arthritis in the United States. Its incidence has been rising significantly over recent years. This has been at least partially attributed to an increasing prevalence of obesity. The disease is caused by deposition of monosodium urate in joints and periarticular structures. This process is typically asymptomatic but does set the patient up for acute flares of inflammatory arthritis.

An acute gout flare is characterized by rapid-onset pain, redness, and swelling in the affected joint area. The acute attack often starts at night or during the early morning hours. The signs of inflammation can extend well beyond the primarily involved site. This “pseudo-cellulitis” can cause diagnostic confusion because it may be difficult to distinguish from a soft-tissue infection.

While the 1st metatarsophalangeal (MTP) joint is the 1st joint affected in most patients, gout can involve any joint or periarticular structure. This is particularly true for recurrent gout. Polyarticular gout flares are more frequently seen in patients with long-standing and poorly controlled disease.

When a patient with acute inflammatory arthritis presents to the emergency department (ED), several factors should raise suspicion that the patient may be affected by a gout attack. These include a history of prior gout flares, renal failure, use of medications that impair uric acid excretion such as thiazide diuretics, and location in the 1st MTP joint. Importantly, gout can coexist with other types of inflammatory joint diseases such as septic arthritis. Therefore, even in the patient with an established history of gout, the possibility of an alternative process needs to be assessed.



FIGURE 26.1 ■ Gout—Acute Flare. Recurrent first metatarsophalangeal joint redness, swelling, pain, and tenderness consistent with an acute gout flare. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

The majority of patients with a suspected gout flare will require a joint aspiration. Testing of the synovial fluid should include a cell count with differential, Gram stain and culture, and crystal analysis (polarizing microscopy).

The synovial fluid or white blood cell count in patients with an acute gout flare is usually in the 10,000 to 100,000 cells/ μ L range. Importantly, this range overlaps with the cell counts typically seen in septic arthritis. Therefore, the white blood cell count in joint fluid does not allow separation of these two conditions.



FIGURE 26.2 ■ Gout—Tophi. Multiple tophi are seen (index finger proximal interphalangeal [PIP], distal interphalangeal [DIP], long finger PIP, and small finger PIP joints) in this patient with long-standing gout. (Photo contributor: Kevin J. Knoop, MD, MS.)

The demonstration of intracellular urate crystals does confirm a diagnosis of gout. However, it does not exclude a 2nd overlapping process such as septic arthritis. Therefore, select patients who are at high risk for infection should be admitted and empirically treated for septic arthritis even if crystals are present.



FIGURE 26.3 ■ Gout—Acute Flare. Patient with known gout and tophi with acute flare of the second toe DIP joint. The medial aspect of the joint is draining synovial fluid from which a smear for crystals was obtained. (Photo contributor: Michael Bezzerides, MS4.)



FIGURE 26.4 ■ Gout—Acute Flare: Knee with Joint Aspiration. Joint aspiration of the right knee in a patient with known gout who has not had gout in his knee. Synovial fluid is mildly cloudy and has crystals on the smear consistent with acute gout. (Photo contributor: Lawrence B. Stack, MD.)

Most patients with acute gout can be treated as an outpatient. Treatment modalities include oral steroids, colchicine, and nonsteroidal anti-inflammatory drugs. Drug selection will often depend on the patient's comorbidities, successful treatment of prior flares, and possible interactions with already established medications.

Pearls

1. In select patients, gout can be diagnosed based on established clinical or radiographic criteria without a synovial fluid aspiration. The Acute Gout Diagnosis Rule (mdcalc.com/acute-gout-diagnosis-rule) is an excellent tool to help determine whether a patient is likely to benefit from joint aspiration.
2. Dual-energy computed tomography (CT) scanning can visualize microtophi [CD1](urate crystals in the synovium) in joints and soft-tissue structures. It is particularly helpful in situations of periarticular, intratendinous, or enthesal gout where a joint aspiration is negative or not feasible

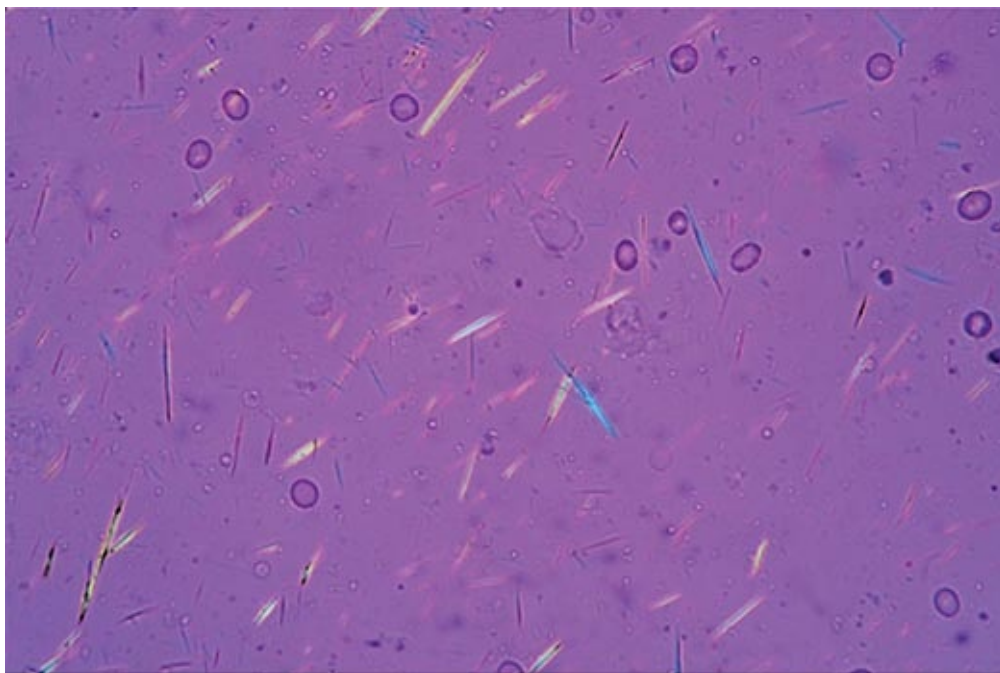


FIGURE 26.5 ■ Uric Acid Crystals. Numerous urate crystals are seen from the wound from the patient in Fig. 26.3. A high concentration of crystals is seen due to the tophi involving the joint. (Photo contributor: Michael Bezzerides, MS4.)



FIGURE 26.6 ■ Pseudogout. Markedly swollen left knee in a patient with an acute flare of pseudogout. (Photo contributor: Robert Tubbs, MD.)

TABLE 26.1 ■ SYNOVIAL FLUID ANALYSIS

	Normal	Septic Arthritis	Gout	Pseudogout
Appearance	Clear	Purulent, turbid	Turbid	Turbid
WBC	<200	Usually >50,000	2000-50,000	2000-50,000
PMN	<25%	>75%	>50%	>50%
Special Tests/ Crystals	Normal “string sign”	+ Gram stain, culture positive	Negative birefringence, needle crystals	Positive birefringence, rhomboid crystals

3. Low-dose colchicine therapy (1.2 mg orally, followed by 0.6 mg after 12 hours) is as effective as high-dose therapy but associated with less side effects.
4. Pseudogout has identical signs and symptoms of gout. Calcium pyrophosphate dihydrate (rod-shaped, rhomboid, weakly birefringent) crystals are deposited in the joint. Treatment of acute flares is similar to treatment of gout.
5. Fifty percent of acute gout flares are polyarticular.

SYSTEMIC LUPUS ERYTHEMATOSUS

Clinical Summary

Systemic lupus erythematosus (SLE) is an autoimmune disease that can affect

any organ. It is nine times more common among woman, typically occurring during the reproductive years. The most common manifestations include constitutional symptoms, skin rash, oral ulcers, polyarthralgias/arthritis, and renal and central nervous system involvement. In addition, there is a high prevalence of antiphospholipid antibodies among patients with SLE, resulting in hypercoagulability with an increased risk of deep vein thrombosis and pulmonary embolism. Patients with SLE are at significant risk for cardiovascular complications including ischemic stroke and myocardial infarction. In fact, the most common cause of premature death in patients with SLE is accelerated atherosclerosis.

Management and Disposition

The SLE patient with a possible lupus flare does require a comprehensive evaluation in order to identify life- or organ-threatening disease. Serious manifestations such as worsening lupus nephritis, autoimmune hemolytic anemia, or lupus cerebritis require rapid rheumatology consultation, aggressive treatment with high-dose corticosteroids, and hospital admission. While select patients with lupus flares that are limited to skin, joints, and pleuritis/pericarditis may be discharged with a steroid taper and close rheumatology follow-up, this should ideally be done after discussion with the treating rheumatologist.

Distinguishing between lupus flare and infection can be challenging. When in doubt, empiric antibiotic treatment with close observation and further workup may be required.



FIGURE 26.7 ■ Systemic Lupus Erythematosus—Butterfly Rash. Appearance of the malar “butterfly” rash in a dark-skinned patient. Note scales on the cheeks and frontal thinning hairline. (Photo contributor: Lawrence B. Stack, MD.)

Given the hypercoagulability and accelerated atherosclerosis in patients with SLE, chest pain in this patient population needs to be approached with a high level of suspicion for serious complications such as acute coronary syndrome and pulmonary embolism. Prediction tools such as the PERC score, Wells score, or HEART score have been found to underestimate the risk in these patients.



FIGURE 26.8 ■ Systemic Lupus Erythematosus—Butterfly Rash. Erythematous malar rash with slight edema and minimal scaling (“butterfly pattern”). (Reproduced with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick’s Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw Hill; 2005, p. 385.)

Pearls

1. New-onset or worsening psychosis and/or seizures in a patient with lupus should raise suspicion for lupus cerebritis. Of note, magnetic resonance imaging (MRI) can be normal in patients with neuropsychiatric lupus.

2. Hypersensitivity to sulfa is more common in patients with SLE, and sulfonamides can induce disease flares. Thus, sulfa-containing medications such as trimethoprim-sulfamethoxazole should be avoided.
3. Lupus patients are immunosuppressed, even when not on immunosuppressive medications, and have a higher incidence of infections such as shingles, candidiasis, and pneumonia.
4. The serum creatinine may be within normal limits early during a lupus nephritis flare. The presence of active urinary sediment (hematuria and proteinuria) is an alarm for renal involvement.



FIGURE 26.9 ■ Systemic Lupus Erythematosus—Palmar Patches. Hyperpigmented patches of the palm and fingers. Patches may be erythematous. (Image appears with permission from VisualDx [www.visualdx.com].)

INFLAMMATORY MYOPATHIES

Clinical Summary

Muscle weakness, not pain, is the hallmark feature of inflammatory myopathies. In polymyositis and dermatomyositis, the muscle weakness is typically proximal and symmetric. Inclusion body myositis can present with more distal and asymmetric muscle involvement.

Patients will typically present with various manifestations of muscle weakness such as difficulties walking, climbing stairs, getting up from a chair, or swallowing. The most concerning manifestation is respiratory muscle weakness with shortness of breath due to restrictive respiratory failure. Muscle pain is typically mild and present in about half of patients.

Patients with dermatomyositis may present with new or worsening skin findings, including Gottron papules, erythroderma, heliotrope erythema, shawl sign, or mechanic's hands.

The vast majority of patients with active inflammatory myositis will have a profoundly elevated creatinine kinase. In inclusion body myositis, the creatinine kinase elevation may be less prominent.

Just as in other systemic rheumatic diseases, various organ- or life-threatening manifestations can be subclinical in these patients and do require a careful diagnostic evaluation. The most feared complications in inflammatory myositis involve the lungs. Patients can have various types of interstitial lung disease and profound respiratory muscle weakness. While renal involvement in inflammatory myositis is rare, it can be associated with other connective tissue disorders such as systemic lupus or scleroderma. Therefore, diagnostic testing should always include a urinalysis to detect glomerulonephritis.



FIGURE 26.10 ■ Atrophic Dermal Papules of Dermatomyositis (ADPDM). Formerly Gottron papules, ADPDM are erythematous to purpuric, scaly, flat-topped papules on the dorsal metacarpophalangeal joints. Mechanic's hands features (hyperkeratosis and scaling of ulnar side of the thumb and radial side of the index finger) are also seen. (Image appears with permission from VisualDx [www.visualdx.com].)



FIGURE 26.11 ■ Dermatomyositis—Heliotrope Eruption. A violaceous, edematous periorbital erythema with mild scaling is considered pathognomonic for dermatomyositis. This patient with undiagnosed dermatomyositis presents with the periorbital swelling, myalgias, and elevated creatinine kinase. (Photo contributor: R. Jason Thurman, MD.)

Management and Disposition

Patients with respiratory failure either due to worsening interstitial lung disease or respiratory muscle weakness may require respiratory support such as bilevel positive airway pressure or intubation.

In cases of organ- or life-threatening manifestations such as severe pulmonary or renal involvement, immediate treatment with high-dose corticosteroids may be indicated. This should be done in close consultation with nephrology and rheumatology.



FIGURE 26.12 ■ Dermatomyositis—Shawl Sign. Sunlight-exacerbated poikilodermatous or macular erythema of the upper back, neck, and posterior shoulders (“shawl sign”) or anterior neck and upper chest (“V sign”). (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Even with a normal oxygen saturation, mild tachypnea in a patient with active inflammatory myositis can indicate looming respiratory failure. The inflamed muscle tires much easier than healthy musculature, and patients can

decompensate rapidly.

2. A negative inspiratory force can be helpful to determine the degree of respiratory muscle weakness in patients with inflammatory myositis
3. Patients with inflammatory myositis have a significantly increased risk of venous thromboembolism. When these patients present with cardiopulmonary symptoms, pulmonary embolism has to be considered as an important differential diagnosis.



FIGURE 26.13 ■ Dermatomyositis—Holster Sign. Violaceous poikilodermatous plaques found over the

right lateral hip are characteristic of the “holster sign” of dermatomyositis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 26.14 ■ Dermatomyositis—Calcinosis. Calcinosis is the abnormal deposition of calcium salts within the dermis, muscles, and tendons and is seen in 70% of patients with juvenile dermatomyositis. (Photo contributor: Jodi Dingle, MD.)

SMALL-VESSEL VASCULITIS AND PURPURA

Clinical Summary

Small-vessel vasculitis and its most common presentation, palpable purpura, often present a major diagnostic challenge to the emergency physician.

Inflammation of small (cutaneous) vessels is the pathomorphologic end-product of a very diverse group of diseases, including infectious, malignant, and autoimmune conditions.

Most commonly, patients will present to the ED because of systemic symptoms such as fever, malaise, and weight loss, together with skin manifestations. These include a wide range of erythematous, macular, and urticarial lesions. However, the most typical manifestation is palpable purpura. It is typically symmetrically distributed and most prominently affects pressure- and gravity-dependent areas such as the lower extremities and buttocks.

Management and Disposition

Management of patients presenting with small-vessel vasculitis focuses on the following two questions: (1) What is the underlying disease?, and (2) What is the extent of organ involvement? The answers to these questions will have significant impact on the treatment and disposition of the patient.

It is often impossible to achieve a high level of diagnostic certainty in the ED. Classification of small-vessel vasculitis often requires advanced testing such as skin biopsies and autoimmune and infectious serologies, which are beyond the scope of the ED, and patients typically require hospital admission.

Particular differential diagnoses, however, should be considered and identified as early as possible. This includes infectious endocarditis and sepsis, which can cause immune complex–mediated small-vessel vasculitis. As a result, every adult patient presenting with concern for small-vessel vasculitis requires a careful exposure history and physical exam.



FIGURE 26.15 ■ Small-Vessel Vasculitis. Palpable purpura confined to bilateral lower extremities that has been present for 1 month. The eruption started 1 week after a viral illness. Lack of other symptoms (fever, malaise, myalgias, lymphadenopathy, bloody stool, abdominal pain, hematuria) suggests leukocytoclastic vasculitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 26.16 ■ Leukocytoclastic Vasculitis. Acute necrotic leukocytoclastic vasculitis. Note necrotic centers with punched-out eruption. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 26.17 ■ Leukocytoclastic Vasculitis. Coalescing purpura that do not blanch. (Photo contributor: J. Matthew Hardin, MD.)

The importance of careful staging of a patient with small-vessel vasculitis cannot be overstated. The patient may only be presenting with palpable purpura but could be harboring *organ- or life-threatening disease* in other areas. More serious manifestations of small-vessel vasculitis include alveolar hemorrhage, mononeuritis multiplex, bowel ischemia, and glomerulonephritis.



FIGURE 26.18 ■ Small-Vessel Vasculitis. Palpable purpura confined to bilateral lower extremities that has been present for 1 month. Lack of other symptoms suggests an idiopathic cause. (Photo contributor: Lawrence B. Stack, MD.)

At a minimum, initial diagnostic testing should include a complete blood count, creatinine, chest x-ray, urinalysis, and rectal examination for blood. During physical examination, particular attention should be given to the presence of a heart murmur and subtle neurologic findings that could indicate vascular leg neuritis, such as a mild foot drop.

Pearls

1. Every adult patient with palpable purpura requires a careful diagnostic workup for possible (asymptomatic) organ or life-threatening disease.
2. An active urinary sediment in the patient with small-vessel vasculitis should raise concern for glomerulonephritis, requiring urgent nephrology consultation.



FIGURE 26.19 ■ Urticarial Vasculitis. Urticarial plaques present over the previous 24 hours. (Photo contributor: J. Matthew Hardin, MD.)

RHEUMATOID ARTHRITIS

Clinical Summary

Rheumatoid arthritis (RA) is a peripheral, symmetric, and inflammatory polyarthritis. Most manifestations of RA are subacute in nature, including gradual onset of morning stiffness, excessive fatigue, and synovitis in various joint areas, most commonly the wrists, metacarpophalangeal, interphalangeal, and shoulder joints, knees, and neck. There are, however, acute and a rapidly worsening manifestations of RA that can be life threatening. Several different extra-articular manifestations can be seen such as pericarditis, myocarditis, interstitial lung disease, or rheumatoid vasculitis.

In addition, there are a variety of important complications of medications used for the treatment of RA that may present as a true emergency. These include infusion reactions, serum sickness, and infectious complications due to the medications' immunosuppressive properties.

Importantly, an acute flare of mono- or oligoarthritis in the RA patient should not be automatically attributed to the underlying disease. Patients with RA are at a significantly increased risk for septic arthritis. Therefore, emergency physicians should have a very low threshold of performing an arthrocentesis with a cell count, Gram stain, and cultures in the patient with RA who presents with an acute arthritic flare in a single joint.

Patients with RA have an increased risk of premature death. This is largely due to an increased incidence of coronary artery disease. The likelihood for a patient with RA to die from coronary artery disease is more than 50% higher than in the general population. Therefore, the RA patient presenting with chest pain, shortness of breath, or other symptoms that can occur in the context of coronary artery disease needs to be very carefully evaluated for acute coronary syndrome. Risk of prediction tools such as the HEART score are likely to underestimate the risk of major coronary events in this patient group, since RA is an independent risk factor for arteriosclerosis.



FIGURE 26.20 ■ Rheumatoid Arthritis—Flare. Acute pain, swelling, redness, and morning stiffness of the MCP and PIP joints consistent with rheumatoid arthritis. (Image appears with permission from VisualDx)

[www.visualdx.com].)



FIGURE 26.21 ■ Rheumatoid Vasculitis. A rare complication of rheumatoid arthritis can manifest as deep ulcers on the legs as a result of medium-sized vessel vasculitis. (Image appears with permission from VisualDx [www.visualdx.com].)

Management and Disposition

Patients with RA disease flares without severe extra-articular manifestations such as a rheumatoid vasculitis, can typically be treated as outpatients with a steroid taper and close rheumatology follow-up. If worsening extra-articular disease is present, patients usually require admission and intravenous (IV) treatment with high-dose steroids.

Emergency providers need to maintain a high index of suspicion for (atypical) infections. Independent of treatment, patients with RA have a higher risk for various infectious diseases. This risk can be further amplified when the patient is on immunosuppressive medications. It can be very challenging to distinguish RA flare from infectious diseases such as sepsis. In these situations, providers may have to cover patients with broad-spectrum antibiotics and admit them for further workup.

Pearls

1. Patients with RA can develop subclinical atlantoaxial dislocation. This is particularly common among patients with long-standing and erosive disease. If a patient with RA does require crash intubation, spinal precautions should be maintained.
2. RA can cause pleural effusions. Just like in empyema, the pH of these effusions is low, and the white blood cell count is high. However, unlike in bacterial empyema, there is a lymphocytic predominance.



FIGURE 26.22 ■ Rheumatoid Vasculitis. Palpable purpura of the lower extremities as a complication of rheumatoid arthritis. (Image appears with permission from VisualDx [www.visualdx.com].)

3. Rheumatoid vasculitis typically occurs in patients with long-standing, burned out RA. It can present with pericarditis, leg ulcers, mononeuritis multiplex, and rheumatoid eye disease.
4. Beware of the “red eye” in patients with RA. Scleritis is an important manifestation of rheumatoid vasculitis and can result in corneal melt syndrome when affecting the keratoscleral junction.

SEPTIC ARTHRITIS

Clinical Summary

Joint pain with or without erythema and swelling is the most frequently encountered rheumatologic problem in the ED. A multitude of conditions can present as inflammatory arthritis, including septic arthritis, gout, pseudogout, and several autoimmune diseases such as RA, SLE, serum sickness, and sarcoidosis.

Among these, the one diagnosis that cannot be missed in the ED is septic arthritis. Failure to recognize infectious arthritis at initial presentation can result in significant morbidity and mortality. Therefore, the key question that the emergency provider needs to answer in the patient presenting with a nontraumatic painful joint is: “Does my patient have septic arthritis?”



FIGURE 26.23 ■ Septic Elbow. Left elbow swelling and redness in a patient with septic arthritis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 26.24 ■ Septic Elbow—Bony Erosion. Erosion of the proximal olecranon in a patient with septic arthritis. (Photo contributor: David Effron, MD.)

Unfortunately, very few clinical parameters are helpful when trying to differentiate infectious from other types of inflammatory arthritis. For example, fever, erythema, and limited range of motion are common in all types of inflammatory arthritis. Furthermore, serum parameters such as white blood cell count or C-reactive protein (CRP) are of limited value. A normal white blood cell count can be seen in 50% of patients with septic arthritis. CRP is elevated in

most types of inflammatory arthritis but does not allow separation between septic and nonseptic etiologies.

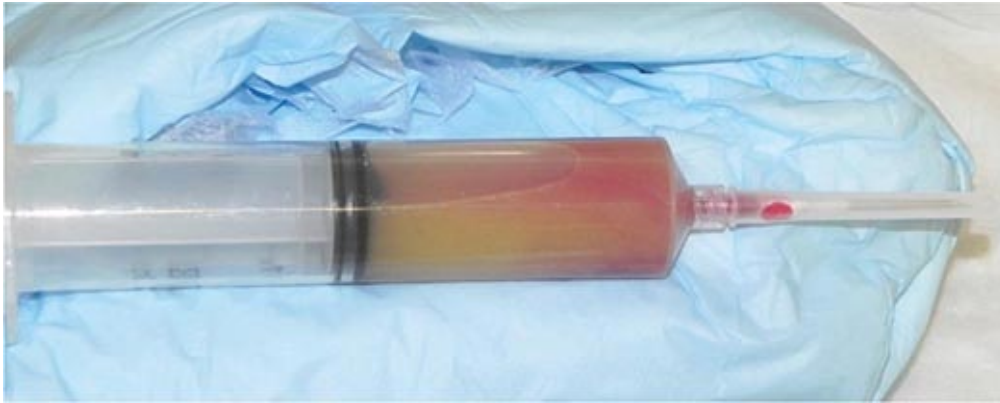


FIGURE 26.25 ■ Septic Synovial Fluid. Purulent synovial fluid from a patient with septic arthritis. (Photo contributor: David Effron, MD.)

The most important diagnostic step in evaluating the patient with inflammatory arthritis is synovial fluid aspiration and analysis including cell count, crystal analysis, and Gram stain and culture.



FIGURE 26.26 ■ Septic Hip Joint Aspiration. Purulent synovial fluid aspirated from the hip of a child with a septic hip. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

If a patient is thought to have septic arthritis, immediate IV antibiotic treatment and timely orthopedic consultation are warranted. Of note, it is often not possible to confirm a diagnosis of septic arthritis in the ED with certainty, since synovial fluid cultures require time to grow and the Gram stain will be negative in the majority of cases. In general, inflammatory mono- or oligoarthritis should be treated as septic arthritis if no alternative etiology is found.

The antibiotic of choice is vancomycin, as it provides good gram-positive coverage, including methicillin-resistant *Staphylococcus aureus*. In patients with IV drug use or immunosuppression, gram-negative coverage such as third-generation cephalosporin should be added.

Pearls

1. Fever is not useful in differentiating different types of inflammatory arthritis.
2. The often-cited threshold of 50,000 cells/ μ L for septic arthritis is a myth. A cell count less than 25,000/ μ L is only associated with a likelihood ratio of 0.3 for septic arthritis and should not be used as a sole criterion to exclude the diagnosis.
3. Uric acid and calcium pyrophosphate dihydrate (CPPD) crystals can be present in the synovial fluid of patients with septic arthritis.

JUVENILE IDIOPATHIC ARTHRITIS

Clinical Summary

The term *juvenile idiopathic arthritis* (JIA), formerly known as juvenile rheumatoid arthritis, describes several subtypes of chronic childhood arthritis further classified by number of joints involved and other clinical characteristics. Oligoarticular JIA (less than five joints involved) and polyarticular JIA (five or more joints involved) patients generally present with joint swelling with painful, limited range of motion that persists for more than 6 weeks. Joints may be warm but are not typically erythematous. The onset is usually insidious, but these patients may present to the ED with concern for mechanical injury or infection. Systemic JIA (known as Still disease in adults) is an important distinct category

of disease characterized by arthritis in addition to a classic daily fever (often spiking in the evening) with accompanying erythematous macular rash that tends to worsen with the fever. Patients with systemic JIA are at risk for developing macrophage activation syndrome, especially early in the disease course and with infections.

Management and Disposition

JIA should be considered in patients with joint effusion and limited range of motion, especially if symptoms have been chronic. Basic plain films can be performed to rule out many traumatic or mechanical causes, and if history of symptoms is chronic, patients with suspected JIA can be referred to follow-up with rheumatology as an outpatient. Acute onset of these symptoms should raise suspicion for infection or mechanical injury, and arthrocentesis or MRI should be considered. Patients with systemic JIA presenting to the ED with fever should have complete blood count (CBC), erythrocyte sedimentation rate (ESR), ferritin, aspartate aminotransferase (AST), and alanine aminotransferase (ALT) drawn to evaluate for macrophage activation syndrome (cytopenias, markedly elevated ferritin, elevated AST/ALT), which can be life threatening. Patients treated with immunosuppressing systemic therapy for JIA (often methotrexate or biologic medications) should have careful evaluation of fever as well, and medications should be held during febrile illnesses.

Pearls

1. Young patients with JIA may not complain of pain; often the presenting symptoms are swelling and functional changes, such as a limp.
2. All patients with systemic JIA who present with fever should have workup for CBC, ferritin, ESR, AST, and ALT to evaluate for macrophage activation syndrome.



FIGURE 26.27 ■ Juvenile Idiopathic Arthritis—Oligoarthritis. Right knee swelling and fever in this 2-year-old female with positive antinuclear antibodies and eventual confirmation of JIA. This was the only joint involved. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 26.28 ■ Juvenile Idiopathic Arthritis. Systemic JIA with characteristic salmon-colored eruption that is typically accompanied by fever. (Image appears with permission from VisualDx [www.visualdx.com].)



FIGURE 26.29 ■ Juvenile Idiopathic Arthritis—Rheumatoid Factor– Positive Polyarthritis. Symmetric polyarthritis of the small joints with positive rheumatoid factor serology. Usually affects adolescent females. (Image appears with permission from VisualDx [www.visualdx.com].)

ACUTE RHEUMATIC FEVER

Clinical Summary

Acute rheumatic fever is a postinfectious complication of group A *Streptococcus* tonsillopharyngitis. Clinical signs can include migratory polyarthritis, carditis, subcutaneous nodules, erythema marginatum, and Sydenham chorea (the five major diagnostic criteria). Arthritis is the most common manifestation, which can be distinguished from other rheumatologic or infectious conditions by its tendency to affect large joints. The migratory pattern is also characteristic and less commonly seen in other conditions. Carditis is the next most common manifestation, often causing endocarditis and possible valvular damage. Subcutaneous nodules (located on extensor surfaces) and erythema marginatum (macular erythematous rash with serpiginous border, as pictured) are less commonly seen. Sydenham chorea presents with jerky, involuntary movements of the face and extremities. While the other major manifestations are typically seen 2 to 3 weeks after streptococcal pharyngitis, chorea can develop several months later. Diagnosis of rheumatic fever also requires evidence of preceding

streptococcal infection, either by positive rapid strep test or evidence of elevated streptococcal antibody tiers (anti-streptolysin O [ASO] or anti-DNAse B). Diagnosis is made based on the presence of two major criteria (carditis, arthritis, chorea, erythema marginatum, subcutaneous nodules) or one major and two minor criteria (fever, arthralgia [in absence of arthritis], elevated acute phase reactants, prolonged PR interval).

Management and Disposition

Patients with migratory polyarthritis or other features concerning for rheumatic fever, as described earlier, require careful auscultation for murmur, echocardiogram, and electrocardiogram for evaluation for carditis. ASO and anti-DNAse B antibodies should be drawn to confirm recent streptococcal infection. Inflammatory markers are often elevated as well. Consult rheumatology and cardiology for assistance with diagnosis. Treatment includes treating the streptococcal infection, as well as long-term antibiotic prophylaxis to prevent recurrent episodes of streptococcal infection, which can lead to further cardiac damage.

Pearls

1. Streptococcal antibodies are helpful in determining preceding infection, as many patients were unaware of or do not recall symptoms of streptococcal pharyngitis.
2. Patients with migratory arthritis following streptococcal infection who do not otherwise meet criteria for rheumatic fever are diagnosed with poststreptococcal reactive arthritis. These patients are given prophylactic antibiotics as well, but for a shorter time course.
3. Obtaining family history of acute rheumatic fever or valvular heart disease is important, as there appears to be a genetic susceptibility.



FIGURE 26.30 ■ Acute Rheumatic Fever—Erythema Marginatum. An evanescent, erythematous, macular eruption with a pale center on the arm of a patient with acute rheumatic fever. (Image appears with permission from VisualDx [www.visualdx.com].)



FIGURE 26.31 ■ Acute Rheumatic Fever—Erythema Marginatum. An evanescent, erythematous, macular eruption with a pale center on the arm of a patient with acute rheumatic fever. (Image appears with permission from VisualDx [www.visualdx.com].)



FIGURE 26.32 ■ Acute Rheumatic Fever. Bilateral knee arthritis in a patient with acute rheumatic fever. (Image appears with permission from VisualDx [www.visualdx.com].)

GIANT CELL ARTERITIS

Clinical Summary

Giant cell arteritis (GCA) is a disease of the elderly, most commonly occurring in the 7th decade and almost never among patients less than 50 years of age. It is a form of vasculitis that involves the aorta and its branches. Constitutional symptoms such as fatigue and weight loss are common. About one-half of patients can have fevers.

The more defining symptoms of GCA are the result of organ and tissue ischemia, caused by inflammatory narrowing of the lumen of the extracranial vessels. The reduced blood flow to facial muscles, the scalp, eyes, tongue, inner ear, and brain can result in a wide variety of symptoms. Headache and scalp tenderness are common presentations; however, they are very non-specific. Importantly, there are no “typical” GCA headaches, as they can be frontal, temporal, occipital, unilateral, or generalized. The most specific symptom of GCA is jaw claudication as a result of reduced blood flow to the masticatory

muscles. The clinical presentation of jaw claudication is often not straightforward. Patients may report facial pressure with talking, difficulties chewing, and problems with mouth opening or speaking.

Vision loss is the most dramatic presentation of GCA and a common reason for patients to present to the ED. It is typically the result of arteritic anterior ischemic optic neuropathy, an inflammatory occlusion of the posterior ciliary arteries. Impairment of vision in patients with GCA is typically monocular and can initially present as transient visual loss (amaurosis fugax), visual field defects, or swift permanent vision loss. Although fundoscopy in these patients is typically abnormal (optic disc edema), fundoscopic findings are never pathognomonic for GCA. On rare occasions, fundoscopy can be completely normal in patients with acute vision loss and a GCA. This can occur in cases where the ocular symptoms are caused by posterior ischemic optic neuropathy or cortical ischemia.

Symptoms of polymyalgia rheumatica, such as proximal myalgias and polyarthralgias, can be associated with GCA in up to one-third of cases.

The diagnosis of GCA in the emergency setting will mostly rely on history and physical exam. Acute phase response parameters such as ESR or CRP are typically elevated but can be normal in rare cases. Although temporal artery biopsy, vascular ultrasound, magnetic resonance angiography, and positron emission tomography are important testing modalities to confirm a diagnosis of GCA, these are typically not available in the ED when a therapeutic decision needs to be made.

Management and Disposition

Patients with suspected GCA require prompt initiation of therapy with high-dose systemic glucocorticoids. This is particularly true for patients with ocular symptoms and threatened visual loss. Importantly, treatment should never be withheld while awaiting more definite tests such as a temporal artery biopsy if there is a strong clinical suspicion for GCA.

In patients without visual symptoms, treatment with 1 mg/kg of prednisone equivalent is typically sufficient. There is no evidence that IV therapy is superior to oral therapy in this group of patients.

In cases of (threatened) visual loss, IV pulse therapy with 500 to 1000 mg of methylprednisolone is recommended.

The vast majority of patients diagnosed with a GCA in the ED will require admission to the hospital for further testing to confirm the clinical diagnosis and

continuation/monitoring of high-dose corticosteroid therapy.

Pearls

1. Leukocytosis is not a laboratory abnormality of GCA. The white blood cell count is typically normal before prednisone is started. If a patient with GCA-like symptoms is found to have an elevated white blood cell count, and alternative diagnoses should be strongly considered.
2. GCA can cause ischemic stroke as a result of significant extracranial disease such as vertebral artery or internal carotid artery involvement.
3. Unexplained pain and/or organ dysfunction above the neck and signs of systemic inflammation in the elderly patient should always alert to a possible diagnosis of GCA.



FIGURE 26.33 ■ Giant Cell Arteritis. Scalp necrosis and alopecia 6 weeks after the diagnosis of GCA was made. (Photo contributor: Jessica Kozel, MD.)

Chapter 27

MENTAL HEALTH CONDITIONS

Brian D. Bales

Max Hensel



Nonsuicidal Self Harm—Superficial Cutting. Superficial cutting with inscribing the word “Die” in a patient with intermittent suicidal ideations. (Photo contributor: Kevin J. Knoop, MD, MS.)

TRICHOTILLOMANIA

Clinical Summary

Trichotillomania (“hair pulling disorder”) is an impulse control disorder resulting in compulsive hair pulling, which leads to noticeable hair loss. This disorder is often seen in patients who exhibit characteristics of obsessive-compulsive behaviors. Hair is repeatedly twisted around the finger and then pulled out or rubbed until it breaks off. Various body sites may be involved, but the scalp is most commonly affected. A hallmark of the hair loss is variable lengths of hair within the region of hair loss.

Other locations commonly affected include the beard line, face, arms, legs, eyebrows, and eyelashes. Young children, adolescents, and women are most commonly affected.

Management and Disposition

Psychiatric referral for habit reversal therapy is first-line management of trichotillomania in children. *N*-Acetylcysteine is more effective than placebo in treating trichotillomania. Tinea capitis, alopecia areata, malnutrition, monilethrix, and other medical causes of hair loss should be considered. Evaluation for behavior such as ingestion of hair, forming a trichobezoar (hairball), is important. A trichobezoar is a rare complication that can result in intestinal or gastric obstruction and often requires surgical removal. Computed tomography (CT) imaging is necessitated when symptoms of obstruction are present.



FIGURE 27.1 ■ Trichotillomania. Note complete hair loss (alopecia) is not present, but rather patchy incongruent areas of hair loss are noted with variable lengths of hair within the affected area. (Photo contributor: Timothy D. McGuirk, DO.)



FIGURE 27.2 ■ Trichotillomania. Lateral upper and lower lid eyelashes have been pulled with mild blepharitis as a complication of eyelash pulling. (Image appears with permission from VisualDx [www.visualdx.com].)



FIGURE 27.3 ■ Trichotillomania. Both eyebrows and eyelashes are missing in this patient with impulse control disorder. (Image appears with permission from VisualDx [www.visualdx.com].)

Pearls

1. Hallmarks of trichotillomania include marked areas of hair loss on the face and scalp with variable lengths of hair present in the affected area.
2. Trichotillomania is most often present with other obsessive-compulsive disorders, and the management of such associated conditions is paramount in correcting this behavior disorder.
3. Ingestion of hair may result in a trichobezoar, which can cause obstructive gastrointestinal symptoms.

SKIN PICKING DISORDER (NEUROTIC EXCORIATIONS)

Clinical Summary

Skin picking disorder (SPD) is a psychocutaneous syndrome characterized by recurrent and deliberate manipulation of the skin resulting in tissue damage. Many terms are used to characterize this disorder and include but are not limited to dermatillomania, neurodermatitis, psychogenic excoriation, lichen simplex chronicus, acne excoriée, and neurotic excoriation. In the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5), SPD is included as a separate diagnosis in the group of the obsessive-compulsive and related disorders, along with trichotillomania and body dysmorphic disorder.

Hallmarks of SPD clinical manifestations are polymorphic lesions at various stages of healing and severity. Distribution includes extensor surfaces of the upper arms and forearms, face, upper back, scalp, and buttocks. Acutely induced lesions are often angular in shape with serosanguinous crust.

Older lesions may be crusted or appear as hypertrophic nodules or atrophic scars at all stages of evolution with a background of postinflammatory hypo- or hyperpigmentation.



FIGURE 27.4 ■ Neurotic Excoriations. Middle-aged female with depression and facial neurotic excoriations. (Image appears with permission from VisualDx [www.visualdx.com].)



FIGURE 27.5 ■ Neurotic Excoriations-Legs. Excoriations of the lower leg at various stages of development and healing. (Image appears with permission from VisualDx [www.visualdx.com].)



FIGURE 27.6 ■ Neurotic Excoriations—Acute on Chronic Findings. Acute neurotic excoriations over extensor surfaces of the forearm. Lesion size ranges from a few millimeters to centimeters in size. (Image appears with permission from VisualDx [www.visualdx.com].)

Management and Disposition

Management of SPD is notoriously difficult, and evaluation must include a thorough medical and psychiatric history with particular focus on elicitation of underlying psychiatric illness while not excluding consideration of a primary underlying dermatologic etiology. Special attention should be placed on addressing secondary infections and/or skin disfigurement requiring surgical intervention. In some cases, semioclusive dressings may be helpful in preventing further skin damage and promoting healing. Topical glucocorticoids such as triamcino-lone acetonide may be helpful in relieving pruritis, although no clinical trials have been performed to date to determine efficacy. Cognitive-behavioral therapy has shown some promise in promoting discontinuation of skin picking behaviors. Psychiatric referral should be considered when an underlying organic cause has been excluded.

Pearls

1. Complications of SPD such as skin infection should be addressed prior to psychiatric evaluation.
2. Careful consideration of primary or chronic skin conditions should be carefully considered at the time of evaluation.
3. Successful management of SPD should include psychiatric referral, although many patients may be reticent to accept such a recommendation.

SELF-HARM BEHAVIOR (NONSUICIDAL AND SUICIDAL SELF-HARM)

Clinical Summary

Nonsuicidal self-injury is characterized by deliberate self bodily harm in the absence of intent to take one's own life. Such injuries may cause substantial bruising, bleeding and skin damage and are often associated with powerful negative emotions such as stress or anxiety. Most often, such self-injury is not preceded by substantial precontemplation and occurs impulsively. Methods of self-injury include cutting, stabbing, severe scratching, and burning of the skin, most often found on the nondominant limb. Other methods include inserting sharp objects under the skin, hair pulling, and carving words or symbols into the skin. Cutting tends to be the most common of self-injuries, estimated as being present in 70% to 90% of individuals engaging in nonsuicidal self-injury. Cuts are often deep enough to cause bleeding and are characterized by a series of parallel lines frequently located over the volar aspect of the upper extremities.



FIGURE 27.7 ■ Nonsuicidal Self-Harm—Superficial Cutting. Deliberate tissue injury without suicidal intent often manifests as superficial cutting, burning, and deep scratching in adolescents, as seen in this patient (Photo contributor: Christopher L. Stark, DO.)



FIGURE 27.8 ■ Nonsuicidal Self-Harm—Deep Cutting. Cutting to the degree that repair is required is likely to be nonsuicidal; however, some would argue this level of injury is a prelude to suicide attempt. (Photo contributor: Christopher L. Stark, DO.)



FIGURE 27.9 ■ Nonsuicidal Self-Harm—Soft-Tissue Foreign Body. Hairpin inserted under the skin in

an adolescent. Insertion of sharp objects under the skin, another form of nonsuicidal self-harm, suggests an increased level of distress compared to superficial cutting. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 27.10 ■ Nonsuicidal Self-Harm—Abdominal Foreign Body. Insertion of a foreign body into the abdominal cavity is common among institutionalized persons and is often done to avoid specific situations as opposed to true self-harm. (Photo contributor: R. Jason Thurman, MD.)

Patients who repeatedly exhibit nonsuicidal self-injury and use multiple methods over their lifetime are at greater risk for a suicide attempt.

Management and Disposition

The initial approach to the patient with nonsuicidal self-injury should address the patient's immediate safety as well as their associated injuries. Many superficial injuries may be easily repaired at the bedside, but the clinician should also consider the possibility of additional modes of self-harm such as ingestion. A thorough medical screening exam should be performed prior to psychiatric evaluation.

Pearls

1. Nonsuicidal self-injury is highly associated with risk for future suicide attempts, and psychiatric stabilization is paramount.
2. Physical injuries occurring from self-injury should be managed as any patient

presenting with trauma with the caveat that patient safety should be carefully considered, ensuring avoidance of any ability to cause further self-harm while the patient's evaluation and stabilization are occurring.



FIGURE 27.11 ■ Suicidal Self-Harm—Ligature Marks of the Neck. Suicide attempt by hanging as evident by the recent circumferential ligature marks around the neck. (Photo contributor: R. Jason Thurman, MD.)

SUBSTANCE USE DISORDERS

Clinical Summary

Substance use disorders (SUDs) are highly prevalent in the United States, with a 2017 national survey estimating that 7.2% of individuals over the age of 12 had a diagnosable SUD in the previous year.

Clinical presentation of SUD varies by substance. Skin manifestations of injectable illicit drug use include needle track marks and excoriations on the extremities, neck, and genitals. In patients with chronic injectable illicit drug use, lesions in various stages of healing overlying a background of chronic skin changes with hypo- and hyperpigmented scarring may be present. Patients presenting with long-term methamphetamine use may exhibit dental erosions associated with dry mouth and long periods of poor oral hygiene. Epistaxis in the

setting of acute or chronic cocaine may represent nasal septal perforation.



FIGURE 27.12 ■ Needle Track Marks. Track mark ecchymoses is seen in the patient's nondominant hand along the length of the visible dorsal veins. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 27.13 ■ Needle Track Marks. Needle track mark ecchymoses from intravenous heroin use on the patient's right thigh along the visible veins. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 27.14 ■ Skin Abscess from Intravenous Drug Use. Warmth, tenderness, swelling, and redness over the antecubital fossa at the site of intravenous drug use likely due to unclean needles, unclean technique, or drug extravasation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 27.15 ■ Skin Abscesses from Skin Popping. Acute complications of skin popping include abscess, cellulitis, folliculitis, and chronic erosions and are seen here. (Image appears with permission from VisualDx [www.visualdx.com].)

When a SUD is suspected, immediate medical complications of the presenting SUD must be excluded. The patient's comorbid psychiatric and medical conditions, their perception of their condition, and identification of barriers reducing their substance abuse is paramount. Specific clinical manifestations vary by substance but may include cardiovascular (endocarditis), pulmonary (septic pulmonary emboli, e-cigarette– or vaping-associated lung injury), infectious (necrotizing fasciitis), dermatologic, neurologic (stroke), psychiatric (mood disorders), hematologic, and pregnancy-related (neonatal abstinence syndrome) complications.

Management and Disposition

Specific interventions should address the presenting toxidrome and immediate complications in acutely ill patients: benzodiazepines for those presenting with agitation and aggression, naloxone for those presenting with respiratory depression, and further interventions to specifically address the bodily system affected by the substance abused. For stable patients deemed to be a danger to self or others or those seeking detoxification and rehabilitation for ongoing substance use and abuse, a medical screening examination is warranted prior to psychiatric evaluation.



FIGURE 27.16 ■ Skin Abscess and Scarring from Skin Popping. Skin abscess and chronic scarring are seen in this patient whose skin pops heroin. (Image appears with permission from VisualDx [www.visualdx.com].)

Pearls

1. Laboratory tests have limited utility in the diagnosis of SUD, although they may be helpful for medical screening prior to psychiatric referral and occasionally in those patients presenting with altered mentation secondary to substance use.
2. Treatment of SUD is often multimodal, requiring the clinician to consider acute symptom management, psychiatric care, and the ability to address social issues such as homelessness, domestic violence, and economic considerations.



FIGURE 27.17 ■ Methamphetamine Teeth. Tooth decay due to dry mouth from stimulant use is seen here. (Photo contributor: Christopher L. Stark, DO.)



FIGURE 27.18 ■ Cocaine Use—Nasal Septum Perforation. Perforation of the nasal septum due to chronic recurrent vasoconstriction. (Photo contributor: Lawrence B. Stack, MD.)

ALCOHOL USE DISORDER

Clinical Summary

It is estimated that between 4% and 40% of all medical and surgical patients experience problems related to alcohol. It well known that alcohol use contributes significant to morbidity and mortality, and it is estimated that roughly 1 in 10 deaths among working-age adults results from excessive alcohol use. DSM-5 characterizes alcohol use disorder as a problematic pattern of use leading to clinically significant impairment or distress, as manifested by multiple psychosocial, behavioral, or physiologic features. Clinical manifestations of alcohol abuse may present in all organ systems, although advanced liver disease is a hallmark of long-term alcohol abuse. Skin changes such as palmar erythema ([Fig. 27.19](#)) and spider angioma ([Video 27.1](#)) are thought to develop due to increased circulating levels of estrogen due to inadequate hepatic metabolism of steroid hormones. Asterixis, also coined “liver flap,” is a type of negative myoclonus seen in advanced liver disease ([Video 27.2](#)). Conjunctival icterus may be seen when circulating serum bilirubin levels rise above 3 mg/dL in the

setting of liver disease (Fig. 27.21). Alcohol is an indirect γ -aminobutyric acid (GABA) agonist, and when removed, symptoms of central nervous stimulation dominate, including agitation, tremulousness, hypertension, diaphoresis, and, when severe, seizures.



FIGURE 27.19 ■ Alcohol Use Disorder—Palmar Erythema. Patient with alcohol-related cirrhosis and palmar erythema. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 27.20 ■ Alcohol Use Disorder—Spider Angioma. Spider angioma, typically located on the upper chest, are seen in patients with cirrhosis due to impaired estrogen metabolism and increased serum vascular endothelial growth factor. (Photo contributor: R. Jason Thurman, MD.)

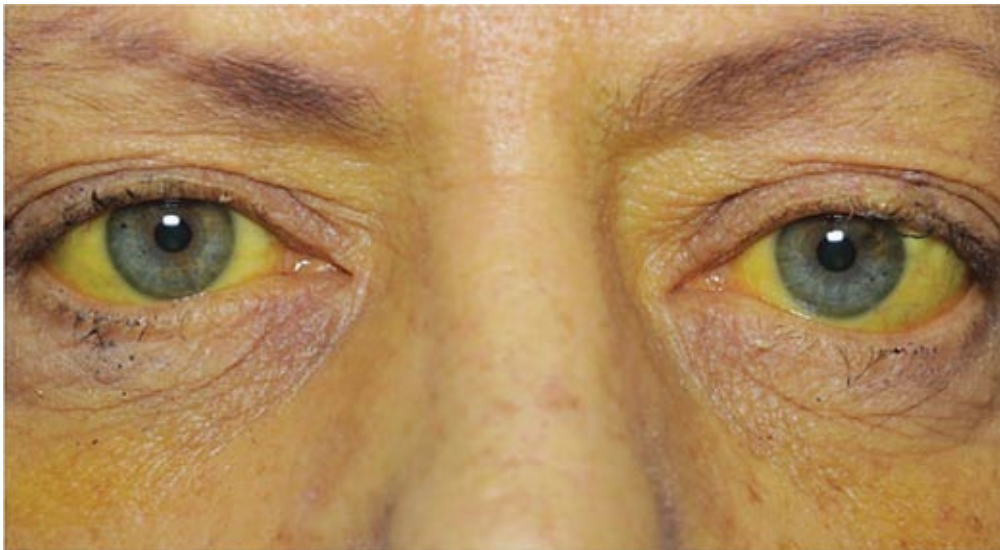


FIGURE 27.21 ■ Alcohol Use Disorder—Conjunctival Icterus. Icterus (jaundice) is seen in this patient's skin and conjunctiva due to advanced cirrhosis due to chronic alcohol use. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Treatment for acute alcohol intoxication is largely supportive. Benzodiazepines

are the mainstay of treatment for alcohol withdrawal, with diazepam as the preferred benzodiazepine due to its long half-life. Long-term alcohol abuse may affect all organ systems, and thus, therapy should be directed at the presenting symptoms and affected organ system. Long-term alcohol use is also associated with vitamin deficiencies. Folate and thiamine supplementation should be considered along with glucose supplementation in patients suspected of long-term alcohol abuse to prevent Wernicke encephalopathy.

Pearls

1. Treatment for acute intoxication is supportive, addressing airway, breathing, and circulation
2. Benzodiazepines are the mainstay of treatment for acute alcohol withdrawal.
3. Thiamine and folate supplementation should be provided along with glucose in patients with hepatic manifestations of long-term alcohol use disorder.

NICOTINE USE DISORDER

Clinical Summary

Nicotine is a naturally occurring alkaloid found primarily in tobacco. Nicotine delivery methods include cigarettes, pipe and cigar tobacco, smokeless tobacco, electronic cigarettes, nicotine gum, and lozenges. Long-term complications of nicotine use include coronary artery disease, stroke, peripheral vascular disease, thromboangiitis obliterans ([Fig. 27.22A](#)), chronic obstructive lung disease, and oropharyngeal and pulmonary malignancies. Lighting a cigarette while using supplemental oxygen may cause flash burns ([Fig. 27.23](#)). Cigarette smoking also causes staining of nails and teeth due to both direct staining from tar and nicotine as well as vascular vasoconstriction mediated by the nicotine and carbon monoxide present in tobacco smoke ([Fig. 27.22B](#)). More recently, the increased use of electronic cigarettes (vaping) has resulted in a rise in e-cigarette– and vaping-associated lung injury (EVALI) with typical manifestations of bilateral ground-glass opacities on chest CT ([Figs. 27.25 and 27.26](#)).



FIGURE 27.22A ■ Tobacco Use Disorder—Thromboangiitis Obliterans. Fingertips of a patient with chronic nicotine use with Buerger disease. Recurrent inflammation and thrombosis are associated with tobacco use. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 27.22B ■ Tobacco Use Disorder—Stained Fingers and Nails. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 27.23 ■ Tobacco Use Disorder—Flash Facial Burns. Facial burns due to rapid combustion of a cigarette while using oxygen. The burns are typically confined to the face and do not involve the supraglottic structures or trachea. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 27.24 ■ Tobacco Use Disorder—Electronic Cigarette Explosion Burns. Thermal burns to first web space and fingers when an electronic cigarette exploded in the user's hand. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 27.25 ■ Vaping-Associated Pulmonary Injury (VAPI)— Chest X-Ray. An anteroposterior chest radiograph that reveals bilateral airspace opacities. These findings in the context of vaping and excluding other causes are suspicious for VAPI. (Photo contributor: Kaitlyn R. Works, MD.)



FIGURE 27.26 ■ Vaping-Associated Pulmonary Injury—Axial Chest CT. The chest CT of the patient in Figure 27.25 reveals bibasilar dependent consolidations with diffuse patch ground-glass opacities. These findings in the context of vaping and excluding other causes are suspicious for VAPI. (Photo contributor: Kaitlyn R. Works, MD.)

Management and Disposition

Treatment of complications of nicotine use disorder is largely supportive and depends on the specific manifestations presenting. Flash cigarette burns to the face prompt evaluation for airway compromise. Optimal treatment of EVALI is not known, although empiric antibiotics are recommended in addition to close respiratory monitoring, with up to 30% of patients requiring ventilatory support.

Pearls

1. Tobacco use is the leading cause of preventable disease, and early intervention leads to improved long-term outcomes.
2. EVALI is a serious complication of e-cigarettes, presenting with bilateral pulmonary infiltrates and hypoxia, and in many states, it is a reportable disease.
3. Flash cigarette burns rarely cause airway injury, and intubation is rarely required.

EATING DISORDERS

Clinical Summary

Eating disorders are characterized by the persistent disturbance of eating impairing health or psychosocial functioning and include food restriction disorders as well as bingeing and purging disorders. Clinical features of restriction disorders such as anorexia nervosa include emaciation, bradycardia (Fig, 27.30), lanugo (Fig. 27.28), hair loss, and xerosis. Bingeing and purging disorders may present with clinical manifestations associated with frequent self-induced vomiting such as acid tooth erosion (Fig, 27.29) or Russell sign, characterized by callouses on the knuckles or back of the hand secondary to repetitive self-induced vomiting over long periods of time (Fig. 27.27).



FIGURE 27.27 ■ Eating Disorder—Russell Sign. Calluses over the dorsal metacarpophalangeal, proximal interphalangeal, and distal interphalangeal joints of the hand is Russell sign due to repeated self-induced vomiting over long periods of time. Callus formation occurs from the skin over the joints repeatedly rubbing against the maxillary incisors during self-induced vomiting. (Image appears with permission from VisualDx [www.visualdx.com].)



FIGURE 27.28 ■ Eating Disorder—Lanugo Hair. Fine, soft, unpigmented hair seen in an adolescent or adult is a clue to anorexia nervosa. Lanugo hair is normally found on a newborn baby. (Image appears with permission from Vivian Wong, MD and VisualDx [www.visualdx.com].)



FIGURE 27.29 ■ Eating Disorder—Acid Tooth Erosion (Bulimia). Erosive dentin exposure of the maxillary teeth due to acid from chronic vomiting. The acid erosion of lingual tooth surfaces is characteristic of bulimia. (Photo contributor: David P. Kretzschmar, DDS, MS.)

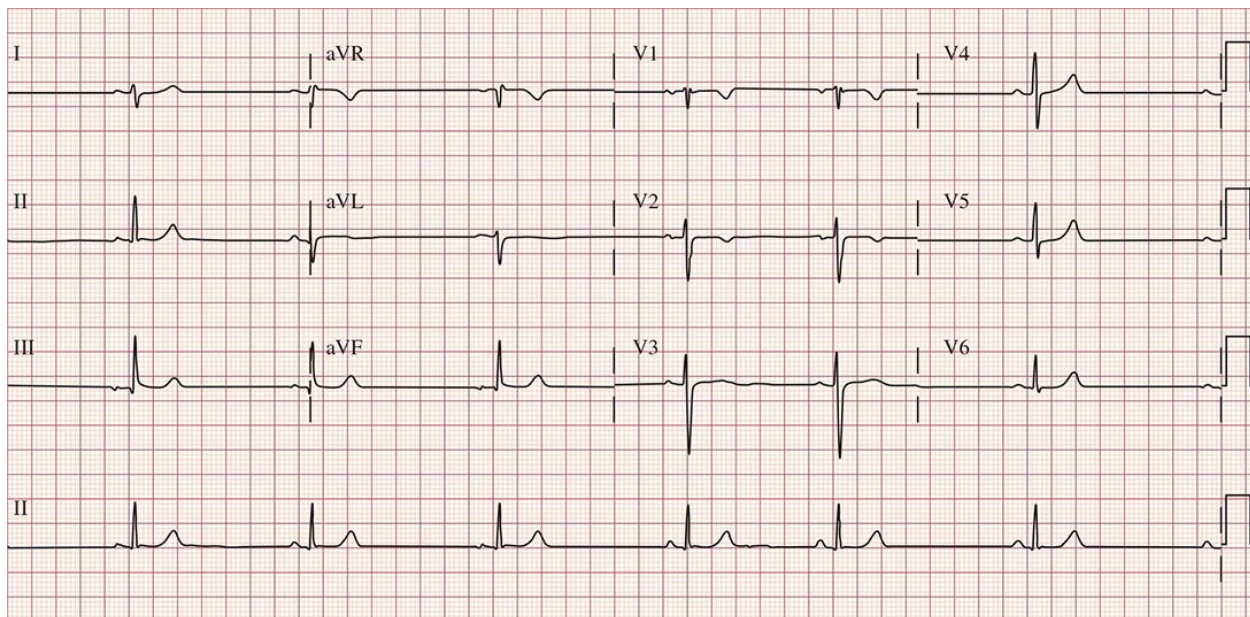


FIGURE 27.30 ■ Eating Disorder—ECG: Sinus Bradycardia. Bradycardia in anorexia nervosa is thought to be due to increased vagal tone due to decreased metabolism due to low caloric intake. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Treatment of electrolyte abnormalities such as hypokalemia and hypoglycemia should be addressed on presentation. Electrocardiogram (ECG) should be obtained to evaluate for cardiac dysrhythmias. Upon ensuring medical stability, psychiatric referral should be obtained.

Pearls

1. Electrolyte abnormalities and cardiac dysrhythmias should be screened for in patients presenting with an eating disorder.
2. Psychiatric evaluation is the mainstay of long-term therapy after medical stabilization has been achieved.
3. Unexplained sinus bradycardia should prompt suspicion of an eating disorder.

DELUSIONAL INFESTATION

Clinical Summary

Delusional infestation (delusional parasitosis) is a rare disorder in which individuals have the delusion that they are infected by a living organism such as worms, mites, bugs, and parasites. Rational reasoning does not impact the delusion. Delusional infestation is more common in patients over the age of 50 and in women. Three of four patients have a concurrent mental health illness, including depression, anxiety, and drug abuse. Such patients often present with excoriations ([Fig. 27.31](#)) and frequently complain of pruritis. It is common for patients suffering from this disorder to present with specimens to support their argument such as bags of stool ([Fig. 27.32](#)) or specimens including string or scabs that they have picked from their skin.



FIGURE 27.31 ■ Delusional Infestation—Multiple Excoriations. Numerous excoriations within reach of the patient's hands combined with a belief that the parasites are buried within the skin are characteristic of a delusional infestation disorder. Note the gloves this patient is wearing to prevent the spread of the parasites. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 27.32 ■ Delusional Infestation—Bags of Stool. Patients with delusional infestation will often collect the “parasites” in bags of stool or urine as evidence of their affliction. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

A nonconfrontational approach to the patient with delusional infestation is recommended, and reassurance can often be helpful in obtaining patient buy-in to a multimodal approach involving further evaluation for the suspected infestation in addition to psychiatric referral as antipsychotics have demonstrated benefit for such patients.

Pearls

1. Management of delusional infestation often requires a nonconfrontational approach requiring the avoidance of dismissing the patient’s concerns as well as not explicitly supporting such beliefs.
2. Antipsychotics are the mainstay of treatment for delusional infestation, although persuading patients to agree to pharmacotherapy is difficult.
3. Do not give the patient with a delusional infestation a chance to refuse seeing a psychiatrist. Simply ask the psychiatrist to see them.

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